McGill

Faculty of Medicine

60th Annual Refresher Course for Family Physicians

November 23-25, 2009

Hilton Montréal Bonaventure

Program Committee

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Zigman, Michael MDCM, FCFP

www.course-mcgill.ca

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Acknowledgements

Our thanks to the following companies for supporting the 60th Annual Refresher Course for Family Physicians with unrestricted educational grants.

BENEFACTORS

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Program • Monday, Nov. 23, 2009

A Question and Answer Period will follow all lectures on all days.

07:00	**Registration** **Continental Breakfast**		Lower Lobby
07:00	**BREAKFAST SYMPOSIUM** – Supported through un unrestricted educational grant from AstraZeneca		
	Moderator: Najmi Nazerali		
	Biomarkers and Cardiovascular Diseases: the Wheat and the Chaff	Jacques Genest Jr.	Le Portage
08:00	**PLENARY**		
	Morning Chairperson - David Dannenbaum		
08:00	Introduction	Ivan Rohan	Westmount
08:15	Official Opening	Richard Levin	Westmount
08:30	Hypertension Update, CHEP Guidelines	George N. Honos	Westmount
09:00	Lipid Update - 2009 Guidelines	Jacques Genest Jr.	Westmount
09:30	Cerebrovascular Accident: Recognition and Management	Theodore Wein	Westmount
10:00	Atrial Fibrilation and Other Arythmias	Magdi Hanna Sami	Westmount
10:30	**Refreshment Break**		Fontaine ABC
11:00	**WORKSHOP A**		
	A-01 ER: MI Acute Management	Eddie Lang	Lachine
	A-02 GER: Renal Failure in the Elderly	Sameena Iqbal	Lasalle
	A-03 PEDS: How to Help Children and Adolescents Deal with Divorce	Audrey Wise	Verdun
	A-04 Research in Your Office	Gillian Bartlett-Esquilant	St-Michel
	A-05 BP Assessment in the Office	Brian Gore	St-Pierre
	A-06 Is My Patient Fit to Fly?	Peter Rohan	Mont-Royal
	A-07 Heart Failure Management	Richard Sheppard	Hampstead
	A-08 ECG Interpretation	Marcel Fournier	Côte-St-Luc
12:00	**LUNCH SYMPOSIUM –** Supported through un unrestricted educational grant from Boehringer-Ingelheim		
	Moderator: Heather Abrahams		
	Diabetic Peripheral Neuropathic Pain (DPNP): A Pathways to Optimal Clinical Outcome	Angela Genge	Le Portage
12:00	**LUNCH**		Fontaine ABC

Program • Monday, Nov. 23, 2009

A Question and Answer Period will follow all lectures on all days.

13:30	**PLENARY**		
	Afternoon Chairperson - Michael Zigman		
13:30	Is There a Doctor in the Stand?	J. Scott Delaney	Westmount
14:00	Contraception	Cleve Ziegler	Westmount
14:30	**WORKSHOP B**		
	B-01 ER: ER Procedures	H. Mitchell Shulman	Lasalle
	B-02 GER: Andropause	Peter Chan	Lachine
	B-03 PEDS: Ortho in Newborn and Very Young	Thierry E. Benaroch	Verdun
	B-04 HANDS ON: Shoulder Exam	J. Scott Delaney	Hampstead
	B-05 Contraception - Practical Approach	Cleve Ziegler	Mont-Royal
	B-06 Exercise Prescription	Ivan Rohan	Côte-St-Luc
	B-07 Avoiding Amputation in the Diabetic Patient	Philip Weech	St-Michel
	B-08 Hot Topics in Adolescent Care	Michael Malus	St-Pierre
15:30	**Refreshment Break**		Fontaine ABC
16:00	**WORKSHOP C**		
	C-01 ER: ER Procedures (repeat of B-01)	H. Mitchell Shulman	St-Pierre
	C-02 GER: Delirium Evaluation	Robert Bailey	Lachine
	C-03 PEDS: Ortho Problems in Teenagers	Thierry E. Benaroch	Verdun
	C-04 PEDS: Pediatric Eye Exam	Rosanne Superstein	Lasalle
	C-05 Laboratory Investigations in Rheumatology and Immunology	Jan Schulz	Mont-Royal
	C-06 Electronic Health Record	Barry Fine	Hampstead
	C-07 Effective CME, E-learning	Michael D. Rosengarten Francesca Luconi	Côte-St-Luc
	C-08 Use of Diet & Excercise in Health Promotion in Teenagers	Alan Pavilanis	St-Michel
18:00	***Cocktails***		Verrière
18:30	60tha Anniversary Dinner and L'Ensemble du Carré St-Louis, followed by:		Le Portage
	The World of Magic - Now You See It Now You Don't	Joseph A. Schwarcz	

Program • Tuesday, Nov. 24, 2009

A Question and Answer Period will follow all lectures on all days.

07:00	**Registration** **Continental Breakfast**		Lower Lobby
07:00	**BREAKFAST SYMPOSIUM** – Supported through un unrestricted educational grant from Boehringer-Ingelheim		
	Moderator: Najmi Nazerali		
	Preventing Cardiovascular Disease in Patients with Diabetes	Sven Wassmann	Le Portage
08:00	**PLENARY**		
	Morning Chairpersons - Heather Abrahams / Lavanya Narasiah		
08:00	Pediatric Allergies	Reza Alizadehfar	Westmount
08:30	Back Pain	Mohan Radhakrishna	Westmount
09:00	CMPA	Ross Berringer	Westmount
09:30	**WORKSHOP D**		
	D-01 ER: Psychiatric Emergencies	Hani Iskandar	Verdun
	D-02 GER: Behavioral Problems in Elderly	Michel Élie	Lasalle
	D-03 PEDS: Wheezing Child	Reza Alizadehfar	Lachine
	D-04 HANDS ON: Back Exam	Mohan Radhakrishna	Mont-Royal
	D-05 CMPA - Obligation of Reporting, Suicide, Homicide	Ross Berringer	St-Pierre
	D-06 Addictions	John Sader	Hampstead
	D-07 Finding Answers to Your Clinical Questions in Two Minutes	Roland Grad	Côte-St-Luc
	D-08 Separation, Divorce and Family Mediation	Gerald Schoel	St-Michel
10:30	**Refreshment Break**		Fontaine ABCD
11:00	**WORKSHOP E**		
	E-01 ER: Acute Confusional State	Eric Tremblay	Hampstead
	E-02 Driving Assessment in the Geriatric Patient	Paul G. Lysy	Lasalle
	E-03 Knee Evaluation	Alan Vernec	Lachine
	E-04 HANDS ON: Back Exam	Mohan Radhakrishna	Verdun
	E-05 CMPA	Ross Berringer	St-Michel
	E-06 End of Life Care	Michael A. Dworkind	St-Pierre
	E-07 Treatment of Resistant Depression	Khalil Geagea	Mont-Royal
	E-08 Occupational Medicine - Returning Patient to Work	Avi Whiteman	Côte-St-Luc
12:00	**LUNCH SYMPOSIUM** – Supported through un unrestricted educational grant from Merck-Frosst		
	Moderator: David Dannebaum		
	Dislipidemia: Prevention of Cardiovascular Disease	Morris Schweitzer	Le Portage

Program • Tuesday, Nov. 24, 2009

A Question and Answer Period will follow all lectures on all days.

12:00	**LUNCH**		Fontaine ABCD
13:30	**PLENARY**		
	Afternoon Chairperson - Adrien Macek		
13:30	CPD, CME Requirements by the College des Medecins	Roger Ladouceur	Westmount
14:00	Antibiotic Prophylaxis	Michael D. Libman	Westmount
14:30	Managament of Ulcerative colitis	Gad Friedman	Westmount
15:00	Red Flags for Early Rheumatology Referral	Michael R. Starr	Westmount
15:30	**Refreshment Break**		Fontaine ABCD
16:00	**WORKSHOP F**		
	F-01 ER: Common Fractures	Robert Drummond	Lasalle
	F-02 GER: Osteoporosis in Elderly	Suzanne Morin	St-Michel
	F-03 PEDS: ADHD in Children	Lily Hechtman	Lachine
	F-04 HANDS ON: Joint Injections	Michael R. Starr	Verdun
	F-05 Anemia, Cases for Family Physician	Susan Solymoss	Mont-Royal
	F-06 Laboratory Medicine, Rational Use	Julie St-Cyr	Hampstead
	F-07 Approach to Pneumonias	Michael D. Libman	Côte-St-Luc
	F-08 IBS Diagnosis and Management	Gad Friedman	St-Pierre
17:00	**EVENING SYMPOSIUM** – Supported through un unrestricted educational grant from AstraZeneca		
	Moderator: Ivan Rohan		
	Pharmacotherapy of Mood and Anxiety Disorders: New Evidence for Improving Response and Remission Rates	Hani Iskandar	Le Portage

Program • Wednesday, Nov. 25, 2009

A Question and Answer Period will follow all lectures on all days.

07:00	**CONTINENTAL BREAKFAST**		Lower Lobby
07:00	**BREAKFAST SYMPOSIUM –** Supported through an unrestricted educational grant from Procter & Gamble		
	Moderator: Daniel E. Lalla		
	Management of Osteoporosis and Fracture Risk in the Elderly	Martin Cohen	Le Portage
08:00	**PLENARY**		
	Morning Chairperson - Najmi Nazerali		
08:00	EBM and Pharmacogenomics - Challenges and Opportunities	Martin Dawes	Westmount
08:30	What's New in Pain Management?	Mary-Ann Fitzcharles	Westmount
09:00	Travel Medicine	Dominique Tessier	Westmount
09:30	Metformin, beyond Type 2 Diabetes	Tina Kader	Westmount
10:00	**Refreshment Break**		Fontaine ABD
10:30	**WORKSHOP G**		
	G-01 ER "Zebras Run with Horses"	Joe Nemeth	Lasalle
	G-02 GER: Drugs in the Elderly	Louise Mallet	St-Pierre
	G-03 Peds - Heart Sounds and Murmurs in Children	Tiscar Cavalle-Garrido	Lachine
	G-04 HANDS ON: Steroid Injections	Michael Stein	Verdun
	G-05 Diabetes	Tina Kader	Mont-Royal
	G-06 Pain Management	Mary-Ann Fitzcharles	Hampstead
	G-07 Evidence Based Medicine in Real Clinics, No Ivory Towers	Martin Dawes	St-Michel
	G-08 Travel Medicine, Malaria and Other Diseases	Dominique Tessier	Côte-St-Luc
	G-09 Prehospital Management of Emergencies	John Boulay	Fontaine D
11:30	**LUNCH SYMPOSIUM** – Supported through un unrestricted educational grant from Pfizer		
	Moderator: Peter Rohan		
	New Horizons in Fibromyalgia: Bringing Hope Through Better Patient Care	Martin Cohen	Le Portage
11:30	**LUNCH AND TECHNOLOGICAL FAIR**		Fontaine ABC

Program • Wednesday, Nov. 25, 2009

A Question and Answer Period will follow all lectures on all days.

13:00	**PLENARY**		
	Afternoon Chairperson - Daniel E. Lalla		
13:00	David J.G. Tector Memorial Lecture	Susie Tector	Westmount
13:30	Pandemic Update	Brian J. Ward	Westmount
14:00	Breast Cancer Detection	John R. Keyserlingk	Westmount
14:30	**Refreshment Break**		Lower Lobby
14:45	Helpful and Harmful Herbs	Joseph A. Schwarcz	Westmount
15:15	Chocolate and Red Wine Anyone?	Joseph A. Schwarcz	Westmount
15:45	Dermatology Quiz	Wayne Carey	Westmount
16:15	Closing Comments	Ivan Rohan	Westmount

Course Faculty

Alizadehfar, Reza MD
Division of Allergy and Clinical Immunology,
The Montreal Children's Hospital – MUHC

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Weech, Phillippe Physiotherapist, Jewish Rehabilitation Hospital

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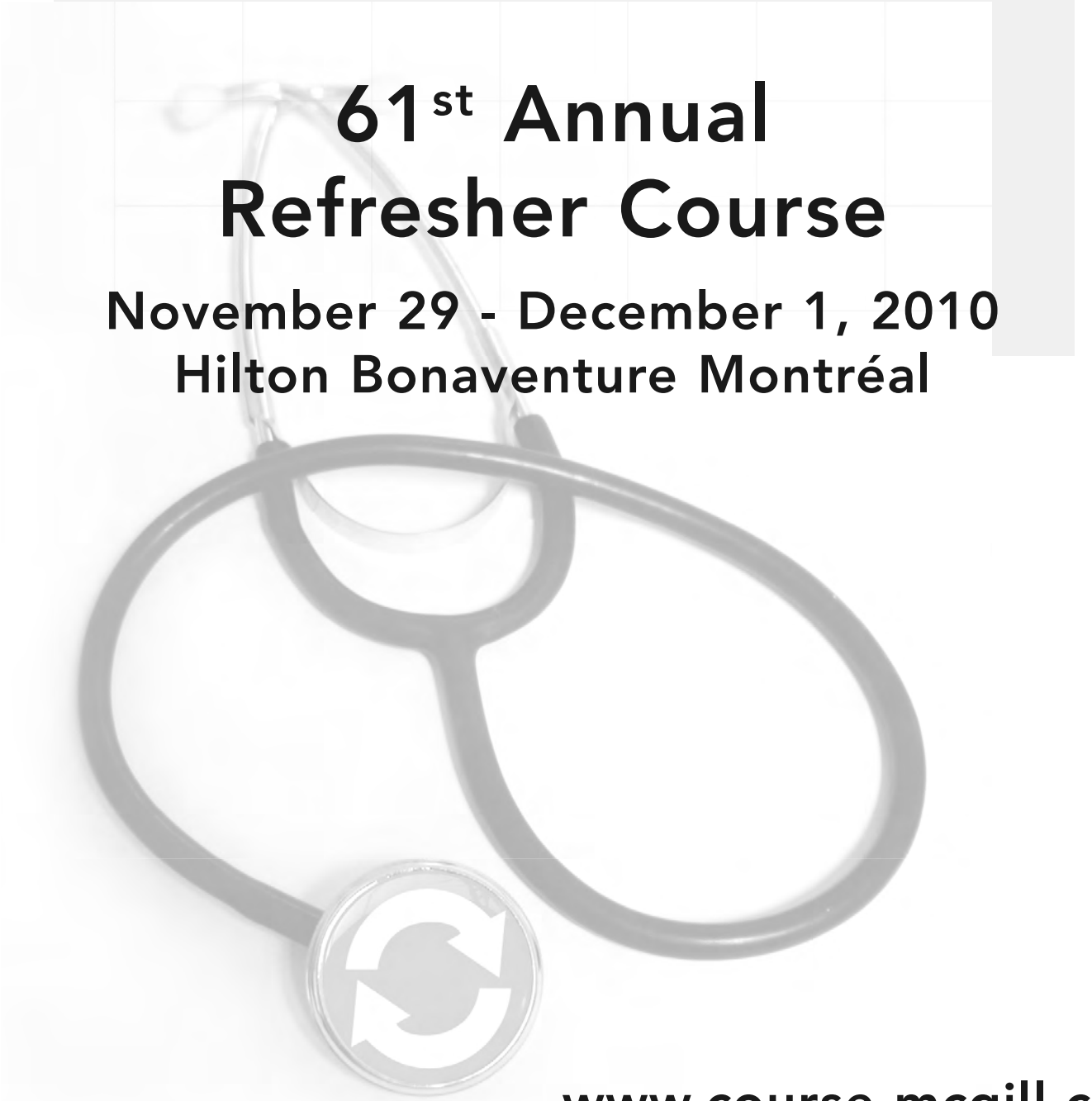
Looking Ahead to 2010

CIRCLE YOUR CALENDAR FOR NEXT YEAR!

61st Annual Refresher Course

November 29 - December 1, 2010
Hilton Bonaventure Montréal



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General Information

Study Credit Hours

Sign-in every morning at the registration desk will be required in order to receive attestation certificates.

This event is approved for up to **23.75** credits by the Centre for Continuing Health Professional Education (CCHPE). The Centre for CCHPE, Faculty of Medicine, McGill University is fully accredited by the Committee on Accreditation of Canadian Medical Schools (CACMS), and through the CACMS is accredited to award AMA PRA category 1 credits.

This program meets the accreditation criteria of the College of Family Physicians of Canada for MAINPRO-M1 credits. Members of the American Academy of Family Physicians are eligible to receive credit hours for attendance at this meeting due to a reciprocal agreement with the College of Family Physicians of Canada.

This event is an accredited group learning activity (Section 1) as defined by the Maintenance of Certification program of the Royal College of Physicians and Surgeons of Canada. Through a reciprocal agreement, The Centre for CCHPE, Faculty of Medicine, McGill University designates this activity for AMA Physicians Recognition Award, Category 1 credit up to the maximum number of credit hours noted above.

Each physician should claim only those hours of credit that he/she actually spent at the educational activity.

For more information:

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Lady Meredith House
1110 Pine Avenue, West, Room 301
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Web Site: http://cme.mcgill.ca

CME Reimbursement

Allowance Fund

This Course qualifies for CME reimbursement for those Québec physicians who meet the requirements for the fund from the Régie d'Assurance du Québec. To obtain an application form (see back of the binder) and for further information please visit the website:

http://www.ramq.gouv.qc.ca/fr/professionnels/form_pro/pdf/3814.pdf

Declaration of Potential Conflict of Interest

Speakers will be requested to disclose to the audience any real or apparent conflict(s) of interest that may have a direct bearing on the subject matter of this program.

Objectives

- The aim of this course is to provide an overall review of topics that would be of interest and relevance to family physicians in both rural and urban practice.
- A mixture of material will be presented and news breaking developments in Family Medicine reviewed. Exposure to basic topics across the entire spectrum of Family Medicine will be ensured and special emphasis given to psychosocial issues.
- A chance to brush up on "hands-on" skills will be offered. There will be ample opportunity for interaction with colleagues and faculty.

Methods

- Short and snappy didactic lectures will cover recent developments in Family Medicine.
- Numerous workshops will offer a choice from among a variety of basic medical topics.
- Streams in Emergency Medicine, Geriatrics and Pediatrics, as well as a Hands-On stream will be offered for those with particular needs. The workshops will allow for informal discussion and consultation with faculty. The David J.G. Tector Memorial Lecture will present an in depth examination of an academic topic in medicine.
- The faculty includes both family physicians and specialists affiliated to McGill University, as well as invited speakers.

60th Anniversary Dinner

Monday, November 23, 2009

Aperitif – 18:00 / Dinner – 18:30

The Course Dinner, preceded by an aperitif, will conclude with an after-dinner presentation by:

Dr. Joseph A. Schwarcz
The World of Magic - Now You See It Now You Don't

Monday, Nov. 23 – Breakfast Symposium

07:00 - 07:45 Breakfast Satellite Symposium

Chair • **Najmi Nazerali**

Biomarkers and Cardiovascular Diseases: The Wheat and the Chaff

Jacques Genest Jr. MD, FRCPC, FACC

Supported through an unrestricted educational grant from AstraZeneca.

Notes

Monday, Nov. 23 – Morning Plenary

08:30 - 09:00 Hypertension Update, CHEP Guidelines

George N. Honos MD, FRCPC, FACC
Director, Noninvasive Cardiology, SMBD-Jewish General Hospital;
Associate Professor, Faculty of Medicine, McGill University

Notes

Monday, Nov. 23 – Morning Plenary

09:00 - 09:30 Lipid Update - 2009 Guidelines

Jacques Genest Jr. MD, FRCPC, FACC
Professor, Faculty of Medicine, McGill University;
Director, Division of Cardiology, McGill University

Research Interests: Dr. Genest is currently Professor, Faculty of Medicine at McGill University and Director of the Division of Cardiology at McGill University Health Centre/Royal Victoria Hospital. Dr. Genest research interests are genetics and biogenesis of high-density lipoproteins (HDL). He is widely regarded as an authority on cardiovascular disease, specializing in the study of lipoproteins. He was recently credited with the discovery of the genetic defect that causes High-Density-Lipoprotein deficiency. Dr. Genest's clinical trial work covers a number of interesting areas including TNT study (Treat to New Targets), CAN-ada study (Canadian Atorvastatin in Diabetics with Atherosclerosis study) and most recently with Pfizer's Torcetrapib (CETP) trial which ended in December 2006.

Dr. Genest is a member of a number of associations including the Canadian Medical Association, American College of Physicians, Royal College of Physicians and Surgeons of Canada, American College of Cardiology and the American Heart Association. Additionally, he serves on the Board of Director of the Royal Victoria Hospital Foundation. Dr. Genest is on the Editorial Board and is a reviewer for the Canadian Journal of Cardiology and is a reviewer for a number of publications including The Lancet, Circulation, Arteriosclerosis Thrombosis and Vascular Biology, American Journal of Cardiology, Journal of the American Medical Association and Atherosclerosis, to name a few. He is the author of more than 160 peer reviewed journals as well as many reviews and book chapters. In 2003 Dr. Genest was awarded the Distinguished Physician Scientist Lecture, Canadian Lipoprotein Conference. Recently he was awarded the 2006 Heart and Stroke Foundation Club Lions de Buckingham / Robert Champagne award of excellence.

Child to Adolescent Basic Office Orthopaedic Principles

Thierry E. Benaroch, MD, FRCS(C)
MUHC

Adolescents

Psychologic Evolution

Physical Transformation

Chronic Lesions

- Training: Rule of Too 's
  - …Too often
  - …Too long
  - …Too hard

and also:
Too specialized, Too young !

Chronic Lesions

- Pb. training:
  - Coach : Voluntary
    - Pushy
  - Parents

…Achievement of dream through another person

Chronic Lesions

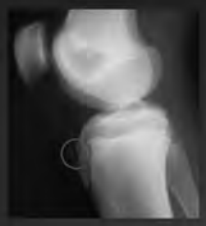
- Very tiny limit between juvenile sports and child abuse

Chronic Lesions

- Predisposing Factors:
  - Training errors
  - Too early specialization
  - Unbalanced ratio of Muscles/Skeleton
  - Anatomical anomalies
- Clinical Findings:
  - Pain
  - Physical Exam: N
  - Delay of growth and puberty

Chronic Lesions

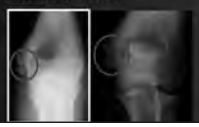
- Apophysitis:
  - Osgood-Schlatter
  - Sever
  - Tibial post.
  - Iselins



Chronic Lesions

« Kissing spine »

Pitcher's Elbow

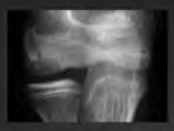


Chronic Lesions : Osteochondritis dissecans

- Treat the patient, not the X-Rays
- Check the other side

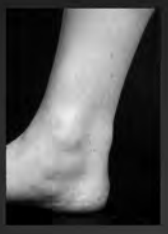
Other lesions

Osteonecrosis



Tendinitis

Very rare before puberty !



Physiological Differences 3

- Growth
  - Increase in length of bones
    - Muscle Stretching

ADOLESCENTS ARE NOT FLEXIBLE !!

Adolescent Knee Pain

Any flags?

Anterior Knee Pain

- Common site of complaint
- Acute trauma
- Repetitive minor trauma

Knee pain in skeletally immature patient = referred hip pain until proven otherwise

Anterior Knee Pain

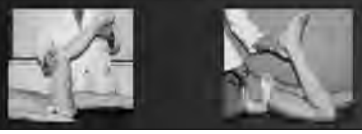
HISTORY:

- Poorly localised
- Usually bilateral
- Grab sign
- associated with prolonged sitting, stairs, + theater sign
- Pseudolocking

Anterior Knee Pain

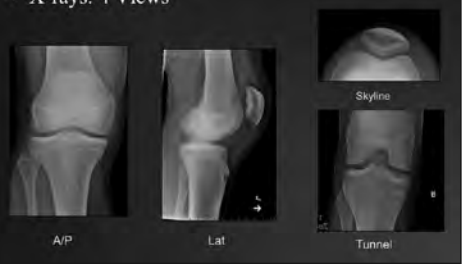
EX:

- Gait, ROM hips, knees, ankles, feet, knee stability, patellar tracking, meniscal tests, tenderness patella, patellar tendon, joint lines
- Atrophy, strength
- Quads/hamstrings flexibility



Anterior Knee Pain

- X-rays: 4 Views



Anterior Knee Pain

- Once other sources of anterior knee pain are R/O, 80% will respond to nonsurgical treatment

Physio: quad, hamstrings, flexibility/strength

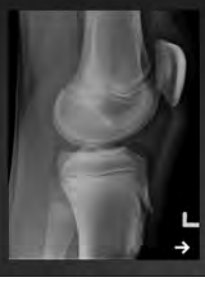
Knee brace

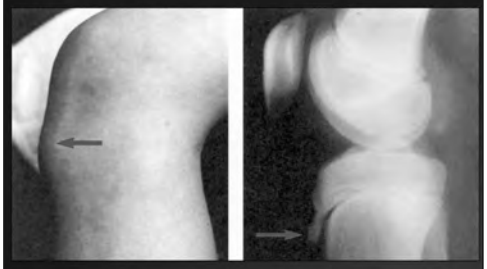
Adolescent anterior knee pain is a mythical disease,equivalent to a headache of the knee.

Surgical intervention has no more rationale than skull burr holes for headache tx.

Anterior Knee Pain

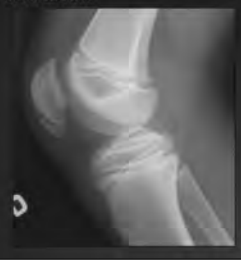
- Osgood-Schlatter





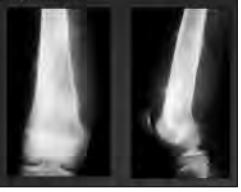
Anterior Knee Pain

- Sinding-Larsen-Johansson



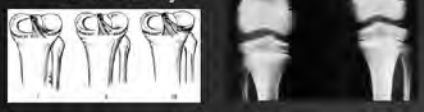
Red Flags

- Hx: Unilateral knee pain
  Swelling, locking, giving way
  No improvement post tx



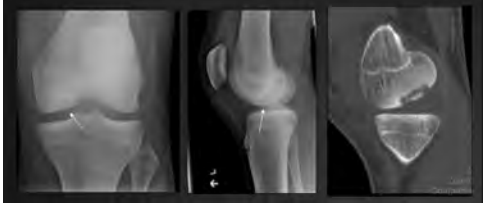
Red Flags

P/E: Swelling
Atrophy
Pain: fem.condyles , joint lines
Abnormal Lig.exam
+ Mc Murray



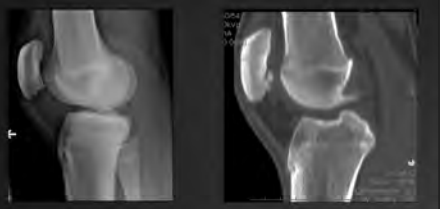
Red Flags

Osteochondritis Dissecans: Femoral Condyle



Red Flags

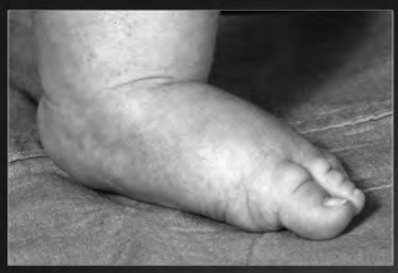
- Osteochondritis Dissecans:Patella



The Flat Foot: A Myth or a Problem?

Flat Feet

- Abnormally low longitudinal arch due to ligament laxity



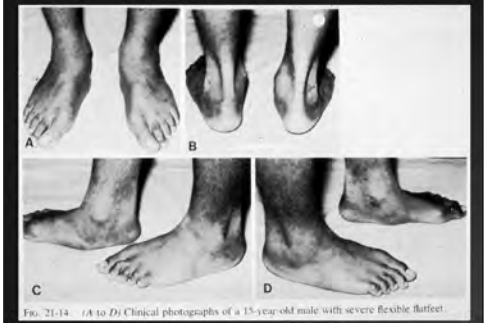
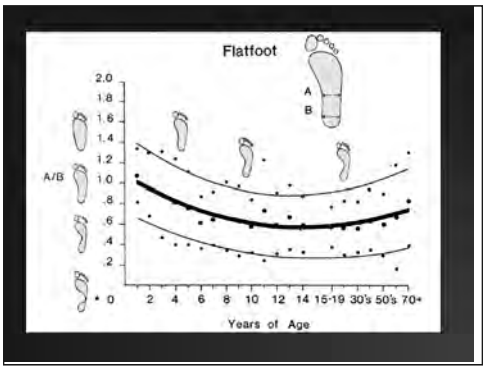
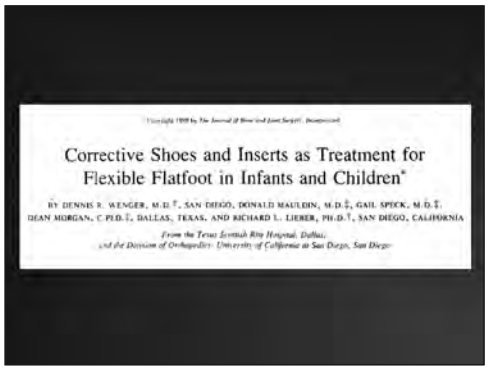


FIG. 21-14 *A* to *D*, Clinical photographs of a 15-year-old male with severe flexible flatfeet.

Flat Feet

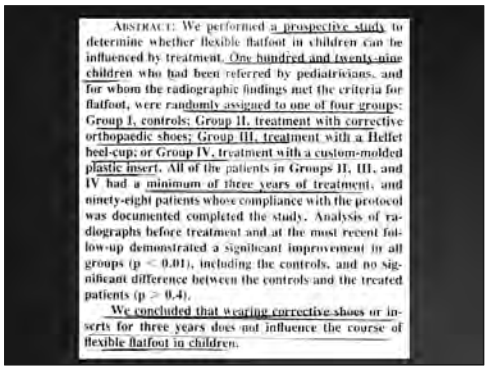
- Most always asymptomatic
- No correlation to back pain
- Major source of concern to parents




Corrective Shoes and Inserts as Treatment for Flexible Flatfoot in Infants and Children*

BY DENNIS R. WENGER, M.D.†, SAN DIEGO, DONALD MAULDIN, M.D.‡, GAIL SPECK, M.D.†, DEAN MORGAN, C.PED.‡, DALLAS, TEXAS, AND RICHARD L. LIEBER, PH.D.†, SAN DIEGO, CALIFORNIA

From the Texas Scottish Rite Hospital, Dallas, and the Division of Orthopaedics, University of California at San Diego, San Diego


ABSTRACT: We performed a prospective study to determine whether flexible flatfoot in children can be influenced by treatment. One hundred and twenty-nine children who had been referred by pediatricians, and for whom the radiographic findings met the criteria for flatfoot, were randomly assigned to one of four groups: Group I, controls; Group II, treatment with corrective orthopaedic shoes; Group III, treatment with a Helfet heel-cup; or Group IV, treatment with a custom-molded plastic insert. All of the patients in Groups II, III, and IV had a minimum of three years of treatment, and ninety-eight patients whose compliance with the protocol was documented completed the study. Analysis of radiographs before treatment and at the most recent follow-up demonstrated a significant improvement in all groups ($p < 0.01$), including the controls, and no significant difference between the controls and the treated patients ($p > 0.4$).

We concluded that wearing corrective shoes or inserts for three years does not influence the course of flexible flatfoot in children.

Treatment

- Only if symptomatic (rare) - almost never < 13 years
- Molded arch supports

Tarsal Coalition

- Congenital abnormality with a varying degree of union between 2 or more tarsal bones producing a rigid flat foot

Symptoms

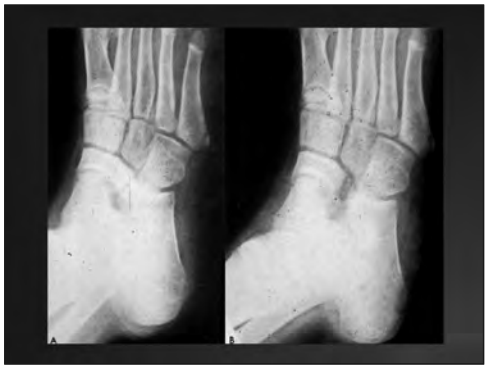
- Pain age 8 - 12 years
- Ossification of bar
- Decreased subtalar motion

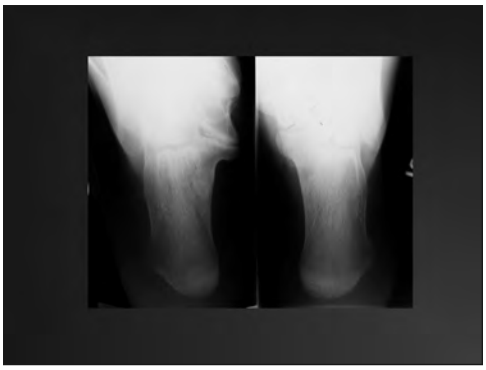
Most Common

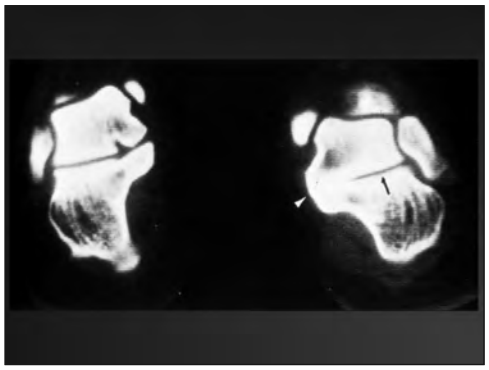
- Calcaneonavicular
- Talo calcaneal
- 50% bilateral

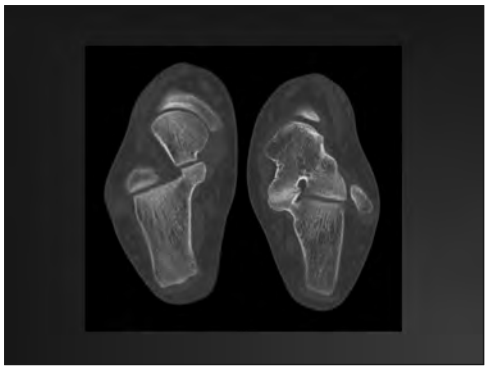
Radiographs

- 45 degree oblique
- Harris Axial Calcaneal View
- CT scan









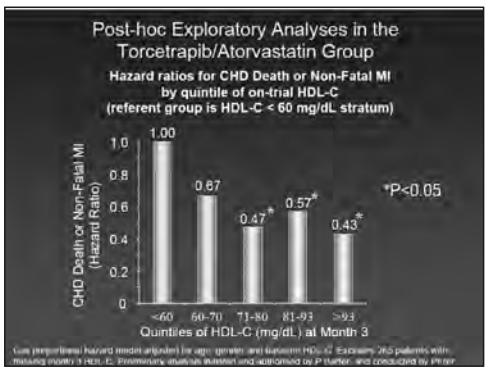
LIMPING

The challenge is to find where and what is the problem with the smallest number of tests, and to determine if further observation or immediate referral is necessary.

You do not want to miss serious conditions such as septic hip.

Look for Five Presentations

- Pain
- Limb length discrepancy
- Stiffness and contractures (sp. Rheumatoid arthritis)
- Neuromuscular conditions
- Hysteria



Pharmacotherapy (Combination therapy)

- Statin + niacin helps dyslipidemia with low HDL-C
- Niacin raises HDL-C better than fibrates
- Crystalline niacin side-effects
- Follow serum transaminase levels (hepatoxicity)
- Awaiting AIM-High and HPS2-THRIVE trial results
- Fibrates effectiveness/safety under study
- Omega 3 fatty acids + statins

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- Fibrates effectiveness/safety under study
- Omega 3 fatty acids + statins

Safety and laboratory monitoring

- Measure baseline lipoproteins, CK, ALT before pharmacological therapy
- Follow-up measurements semiannually or with therapy changes
- Statin side-effects: myalgias, myositis, rhabdomyolysis
- Niacin can elevate glucose and ALT
- Monitor parameters and adjust/withdraw doses
- Fibrates can raise plasma creatinine: avoid in renal insuffiency
- Re-evaluate renal functions and lipid parameters

Risk Assessment and Treatment Targets

Risk Assessment	Initiate/consider treatment if any of the following	Primary Target LDL-C	Primary Alternate ApoB
HIGH *FRS ≥ 20%* *RRS≥ 20%*	• CAD • PVD • Atherosclerosis • Most Diabetic Patients (consider treatment in all patients)	< 2 mmol/L or ↓ LDL-C 50%	ApoB < 0.80
Moderate *FRS 10-19%*	• LDL-C > 3.5 mmol/L • TC/HDL-C > 5.0 • hsCRP > 2 mg/L * • Family history		
LOW *FRS < 10%*	• LDL-C > 5.0mmol/L	↓ LDL-C 50%	

Harmonization of CVD Prevention Guidelines Across Canada

2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult – 2009 recommendations

Jacques Genest MD[1], Ruth McPherson MD PhD[2], Jiri Frohlich MD[3], Todd Anderson MD[4], Norm Campbell MD[4], André Carpentier MD[5], Patrick Couture MD[6], Robert Dufour MD[7], George Fodor MD[2], Gordon A Francis MD[3], Steven Grover MD[1], Milan Gupta MD[8], Robert A Hegele MD[9], David C Lau MD[10], Lawrence Leiter MD[11], Gary F Lewis MD[12], Eva Lonn MD[13], GB John Mancini MD[14], Dominic Ng MD PhD[11], Glen J Pearson PharmD[15], Allan Sniderman MD[16], James A Stone MD PhD[10], Ehud Ur MD[14]

J Genest, R McPherson, J Frohlich, et al. 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult 2009 recommendations. Can J Cardiol 2009;25(10): 567-579.

The present article represents the 2009 update of the Canadian Cardiovascular Society guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult.

Key Words: *Atherosclerosis; Cardiovascular risk factors; Cholesterol; Coronary artery disease; Dyslipidemia; Lipids; Secondary prevention*

Les lignes directrices canadiennes 2009 de la Société canadienne de cardiologie pour le diagnostic et le traitement de la dyslipidémie ainsi que pour la prévention des maladies cardiovasculaires chez l'adulte Des recommandations pour 2009

Le présent article contient la mise à jour 2009 des lignes directrices de la Société canadienne de cardiologie pour le diagnostic et le traitement de la dyslipidémie et pour la prévention des maladies cardiovasculaires chez l'adulte.

Cardiovascular disease (CVD) causes one-third of deaths in Canada more than any other illness (1,2). The prevalence of CVD is expected to increase in Canada in the next decade, predominantly because of increasingly sedentary lifestyles and an attendant increase in the prevalence of obesity and diabetes mellitus. The economic cost of CVD represents approximately $22 billion in direct and indirect health care costs and lost productivity annually. However, mortality from coronary artery disease (CAD) in Canada has decreased by nearly 40% in the past several decades (2). Intensive secondary prevention has resulted in a marked decrease in recurrent cardiovascular events in patients with established CAD, to a level approaching that of age- and sex-matched individuals without clinical CAD (at least in clinical trials). The decrease in cardiac mortality has been attributed to improvements in the control of CVD risk factors especially cholesterol levels, smoking and blood pressure and to improved medical management of patients with CVD. Despite these improvements, CVD still represents the major burden of disease in our society.

The incorporation of new data from clinical studies into clinical practice guidelines helps promote a standard of care that is current and uniform across Canada. Frequent updates are required to take this new information into account. The development of guidelines has undergone major changes to reduce bias by promoting a structured process that assesses and grades evidence, and highlights potential conflicts of interest among contributors. Duality of interest of participants of guideline development has been the focus of much attention and debate, recognizing that individuals have many potential sources of bias. In common with documents prepared in other therapeutic areas, the present guidelines were developed by volunteer experts in lipid disorders and CVD, with full and open disclosure of their relationships with the pharmaceutical industry. There was no direct financial support for this guideline development from industry, nor was there any involvement by them in the guideline writing process.

While the major principles of screening and risk stratification in the 2006 Canadian lipid guidelines (3) have been retained, the process by which this updated version was developed took into account comments and criticisms by many stakeholders. The process changes include working under the Canadian Cardiovascular Society (CCS) guidelines process, and the establishment of primary and secondary review panels. In addition, members of the Canadian Vascular Coalition have had input in the guideline process. A systematic electronic PubMed search of original research published in the medical literature between January 1, 2006, and February 1, 2009, was performed. The following key words were used: lipid-lowering therapy (including generic names of medications), statins, fibrates, niacin, ezetimibe, diet, cardiovascular disease, prevention and clinical trials. Only blinded randomized controlled trials with cardiovascular outcome data were retained for evaluation. Meta-analyses of studies of the efficacy and safety of lipid-lowering therapies

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TABLE 1
Patients whose plasma lipid profile should be screened

- Men ≥40 years of age, and women ≥50 years of age or postmenopausal
- All patients with the following conditions, regardless of age:
  - Diabetes
  - Hypertension
  - Current cigarette smoking
  - Obesity (Obesity Canada guidelines)
  - Family history of premature CAD (<60 years in first-degree relatives)
  - Inflammatory diseases* (systemic lupus erythematosis, rheumatoid arthritis, psoriasis)
  - Chronic renal diseases (eGFR <60 mL/min/1.73 m^2)
  - Evidence of atherosclerosis
  - HIV infection treated with highly active antiretroviral therapy
  - Clinical manifestations of hyperlipidemias (xanthomas, xanthelasmas, premature arcus cornealis)
  - Erectile dysfunction
- Children with a family history of hypercholesterolemia or chylomicronemia

**Data on inflammatory bowel diseases are lacking. CAD Coronary artery disease; eGFR Estimated glomerular filtration rate*

TABLE 2
International Diabetes Federation classification of the metabolic syndrome

Central obesity	
	Waist circumference
Europids	Men ≥94 cm; women ≥80 cm
South Asians	Men ≥90 cm; women ≥80 cm
Chinese	Men ≥90 cm; women ≥80 cm
Japanese	Men ≥90 cm; women ≥80 cm
Ethnic South and Central Americans	Use South Asian recommendations until more specific data are available
First Nations	Use South Asian recommendations until more specific data are available
Sub-Saharan Africans	Use European data until more specific data are available
Eastern Mediterranean and Middle East (Arabic) populations	Use European data until more specific data are available
Plus two of the following factors:	
Plasma triglycerides >1.7 mmol/L	
High-density lipoprotein cholesterol	
Men <1.03 mmol/L	
Women <1.3 mmol/L	
Blood pressure >130/85 mmHg (or treatment for hypertension)	
Fasting plasma glucose >5.6 mmol/L	

Data from reference 10

and on the predictive value of established and emerging risk factors were also reviewed. Strict criteria have been implemented for the incorporation of biomarkers of risk. **Novel biomarkers (4,5) must show improved risk prediction over the previously accepted markers and improved CVD risk stratification, and demonstrate that clinical decisions and outcomes are influenced by their measurement.**

The Canadian Vascular Coalition represents an informal group of stakeholders involved in CVD prevention under the banner of the Canadian Institutes of Health Research. Member organizations are listed in Supplementary Table 1. (Supplementary information begins on page 576.) The recommendations for the treatment of lipoprotein disorders are harmonized with those of the major Canadian stakeholders in CVD prevention. Areas of discordance between the various stakeholders and opinion leaders are highlighted and discussed. The CCS provided oversight and logistical support for the process. The recently released recommendations of the Canadian Heart Health Strategy and Action Plan (available at http://www.chhs-scsc.ca/web/) were also influential in writing these guidelines. The writing group used a widely accepted system to grade and assess the evidence behind the recommendations, based on consensus (Supplementary Table 2).

Since the previous publication of the recommendations for the management and treatment of dyslipidemia in 2006 (3), a number of new clinical studies have been published. When assessing interventions, the primary outcomes examined were cardiovascular death, nonfatal myocardial infarction (MI) and stroke as a combined end point, and total mortality as a secondary end point. Less emphasis was placed on the effects of biomarkers on cardiovascular risk or surrogate end points, such as invasive or noninvasive atherosclerosis assessment. The major changes in our recommendations since the 2006 guidelines are summarized in Supplementary Table 3. The high-risk population has been better defined, including patients with end-stage cardiac or renal disease (ie, severe heart failure or chronic kidney disease on hemodialysis, respectively). Improved, validated CVD event risk-stratification tools are provided. This is especially relevant in subjects at intermediate CVD risk for whom the justification of treatment, other than health behaviour interventions, is often extrapolated from studies of high-risk patients.

The screening strategy is defined in Table 1. The importance of genetic factors and family history of premature CVD is taken into account in the determination of risk (6,7). The importance of obesity (especially abdominal obesity) as a major modifiable CVD risk factor (8,9) is emphasized by including the International Diabetes Federation (IDF) classification of the metabolic syndrome (10) (Table 2) and including overweight and obesity in the screening strategy. We have included risk stratification for several inflammatory diseases, including rheumatoid arthritis, psoriasis and systemic lupus erythematosis (SLE) (11-13). Such patients require comprehensive assessment and treatment of the traditional cardiovascular risk factors. The association between inflammatory bowel diseases (which share many commonalities with other inflammatory diseases) and CVD is less well established (14,15). The use of biomarkers of inflammation is now included in the guidelines based, in large part, on the epidemiology of high-sensitivity C-reactive protein (hs-CRP) and clinical trials of patients with high hs-CRP levels (4,5). Similarly, recommendations for patients with chronic HIV infection who are on highly active antiretroviral therapies are included (16).

We also provide simplified target lipid levels. The emphasis is once again focused on atherogenic lipoproteins, as reflected by the serum (or plasma) levels of low-density lipoprotein cholesterol (LDL-C) or apolipoprotein (apo) B. The evidence favouring LDL-C reduction for the prevention and treatment of atherosclerosis is strong and compelling, and is based on multiple randomized clinical trials (17). Whereas a specific target level for LDL-C will remain a matter of debate, the data indicate that a lower level of LDL-C is associated with reduced CAD risk (18). LDL-C therefore continues to constitute the primary target of therapy; the alternate primary target is apoB. A summary is provided of optional secondary therapeutic targets of potential relevance once the LDL-C (or apoB) is at target, including (in alphabetical order) the apoB to apoAI ratio, the total cholesterol (TC) to high-density lipoprotein cholesterol (HDL-C) ratio, and the hs-CRP, non-HDL-C and serum (or plasma) triglyceride levels. Increased levels of all these parameters have been found to confer additional risk. However, clinical trial evidence is lacking on the importance of intervening on these variables to further reduce risk and thus, they are considered secondary and optional targets (19). We also provide further consideration for the noninvasive assessment of atherosclerosis in asymptomatic individuals, bearing in mind that data on cost effectiveness and outcomes are lacking.

While there is general agreement on the need for sustained, aggressive and multifactorial therapeutic interventions in the secondary prevention of CVD (18,20,21), controversy remains about the cost effectiveness and societal impact of primary prevention strategies. However, most heart attacks occur in subjects with relatively 'normal'

serum cholesterol levels (based on population distribution) but frequently suboptimal levels of cardiometabolic fitness in association with tobacco consumption. Many biomarkers, including levels of serum lipids, lipoproteins, apolipoproteins and various derived ratios, predict CVD risk (5). However, it is important to keep in mind that none of the traditional CVD risk factors or biomarkers reflect the actual presence or absence of atherosclerosis. They help to establish CVD event risk rather than the risk or presence of CVD itself. The inflammatory biomarker hs-CRP also predicts risk and identifies a population that responds particularly well to statin therapy. Importantly, however, our ability to predict CVD events does not always translate into our ability to prevent subsequent events. For instance, homocysteine level predicts CVD risk, but lowering an elevated homocysteine level with folic acid and other B vitamins to prevent recurrent cardiovascular events has proven to be unsuccessful (22). Therefore, we have focused on CVD risk factors whose measurement influences clinical decision making and for which there exists a proven effect on clinical outcomes.

CARDIOVASCULAR RISK FACTORS

Multiple epidemiological studies (23,24) have confirmed that the following risk factors account for the majority of CAD cases:

- Age (the major determinant of risk);
- Male sex;
- Cigarette smoking;
- Diabetes mellitus;
- Cholesterol (as assessed by TC, LDL-C or apoB);
- HDL-C;
- Blood pressure;
- Family history of premature CAD (younger than 60 years of age);
- Inflammatory biomarkers (especially hs-CRP); and
- Overweight and obesity.

Other variables conferring risk include poor nutrition, caloric excess resulting in overweight and obesity, physical inactivity and psychological stress. Because of the increase in prevalence of obesity in our society, the features of the metabolic syndrome (cardiometabolic risk) should be evaluated (Table 2), and should focus the physician's attention on anthropometric (ie, 'toxic waist') and metabolic abnormalities that can be improved or corrected by health behaviour interventions. Patients with chronic kidney disease (25,26), chronic autoimmune inflammatory diseases (rheumatoid arthritis, SLE and psoriasis) (11-13), as well as those with chronic HIV infection requiring highly active antiretroviral therapy (16), should be screened for the traditional CVD risk factors and treated according to their determined risk. Many novel and emerging risk factors have been demonstrated to improve risk prediction over and above the major risk factors considered in the Framingham risk score (FRS), albeit usually marginally, but these 'emerging' risk factors have not been shown to positively influence treatment outcomes. The measurement of hs-CRP, however, is being recommended in men older than 50 years and women older than 60 years of age who are at intermediate risk (10% to 19%) according to their FRS score and who do not otherwise qualify for lipid-lowering therapy (ie, if their LDL-C is less than 3.5 mmol/L).

The rationale for measuring hs-CRP specifically in these individuals is that we now have class I evidence (5) for the benefit of statin therapy in such individuals, if their hs-CRP is greater than 2.0 mg/L. Data from the Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin (JUPITER) (5) show that statin therapy reduces cardiovascular events (hazard ratio 0.56 [95% CI 0.46 to 0.69]; P<0.00001). Importantly, because hs-CRP can be elevated during acute illness, clinical judgment should be exercised in the interpretation of any single measurement of hs-CRP.

Screening (Table 1)

Screening of the plasma lipid profile is recommended in adult men who are at least 40 years of age, and in women who are at least 50 years of age or postmenopausal (class I, level C). In addition, all subjects with evidence of atherosclerosis in any vascular bed, irrespective of age, should be treated as being a high-risk patient (Table 3). Similarly, all adults with diabetes should have a complete lipid profile. Most adults with diabetes (men older than 45 years and women older than 50 years of age, as well as many younger patients who have diabetes with at least one additional traditional CVD risk factor) are considered to be at high risk for CVD events. Individuals with a family history of premature CVD (younger than 60 years of age) deserve earlier screening. Several medical conditions are associated with premature CVD. For instance, patients with arterial hypertension should be carefully assessed for concomitant metabolic disorders and dyslipidemias. Patients with abdominal obesity, as defined by an increased waist circumference or a body mass index (BMI) of greater than 27 kg/m^2 to 30 kg/m^2 (overweight), or greater than 30 kg/m^2 (obese) should also be screened. The metabolic syndrome classification recommended by the IDF classification is advocated because it most accurately reflects the diverse ethnic makeup of Canada (Table 2) (10). Autoimmune chronic inflammatory conditions such as rheumatoid arthritis, SLE and psoriasis are associated with increased CVD event risk. Patients with chronic kidney disease (estimated glomerular filtration rate of less than 60 mL/min/1.73 m^2) are also at increased risk for CVD events.

TABLE 3
Target lipid levels

Risk level	Initiate treatment if:	Primary targets	
		LDL-C	Alternate
High CAD, PVD, atherosclerosis* Most patients with diabetes FRS ≥20% RRS ≥20%	Consider treatment in all patients	<2 mmol/L or ≥50% ↓ LDL-C **Class I, level A**	apoB <0.80 g/L **Class I, level A**
Moderate FRS 10%–19%	LDL-C >3.5 mmol/L TC/HDL-C >5.0 hs-CRP >2 mg/L Men >50 years Women >60 years Family history and hs-CRP modulates risk (RRS)	<2 mmol/L or ≥50% ↓ LDL-C **Class IIa, level A**	apoB <0.80 g/L **Class IIa, level A**
Low FRS <10%	LDL-C ≥5.0 mmol/L	≥50% ↓ LDL-C **Class IIa, level A**	

*Grades and levels of evidence for each target are shown in bold. Clinicians should exercise judgement when implementing lipid-lowering therapy. Lifestyle modifications will have an important long-term impact on health and the long-term effects of pharmacotherapy must be weighed against potential side effects. Meta-analysis of statin trials show that for each 1.0 mmol/L decrease in low-density lipoprotein cholesterol (LDL-C), there is a corresponding RR reduction of 20% to 25%. Intensive LDL-C lowering therapy is associated with decreased cardiovascular risk. Those whose 10-year risk for cardiovascular disease (CVD) is estimated to be between 5% and 9% have been shown in randomized clinical trials to achieve the same RR reduction from statin therapy as those at a higher 10-year risk (25% to 50% reduction in events), but the absolute benefit of therapy is estimated to be smaller (in the order of 1% to 5% reduction in CVD), the numbers needed to treat to prevent one cardiac event are higher and the cost/benefit ratio of therapy is less favourable than for those at higher risk for CVD. For individuals in this category, the physician is advised to discuss these issues with the patient and, taking into account the patient's desire to initiate long-term preventive cholesterol-lowering therapy, to individualize the treatment decision. *Atherosclerosis in any vascular bed, including carotid arteries. apoB Apolipoprotein B level; CAD Coronary artery disease; FRS Framingham risk score; HDL-C High-density lipoprotein cholesterol; hs-CRP High-sensitivity C-reactive protein; PVD Peripheral vascular disease; RRS Reynolds Risk Score; TC Total cholesterol*

Clinical manifestations of genetic hyperlipidemias, including xanthomas, xanthelasmas and premature arcus cornealis, should be sought because they may signal the presence of a severe lipoprotein disorder, especially familial hypercholesterolemia the most frequent monogenic disorder associated with premature CVD. Survival of patients with chronic HIV infection has improved, due largely to highly active antiretroviral therapies, which may be associated with accelerated atherosclerosis (27). The consensus of opinion is that HIV patients should also be evaluated for CVD risk and should be treated accordingly.

The screening of children must be based on sound clinical judgment. Children of patients with severe dyslipidemia (familial hypercholesterolemia or chylomicronemia) should be evaluated and followed in specialized clinics if affected. Similarly, premature CVD in first-degree relatives should prompt the screening of family members for significant lipoprotein disorders.

Family history

The etiology of CVD can be explained by conventional risk factors (24), which can have both genetic and environmental determinants. Importantly, 10% to 15% of patients with CAD have no apparent major CAD risk factors. However, CVD and CVD-related events occur along a continuum of risk, and persons with no apparent exposure to the traditional CVD risk factors may be exceptionally susceptible to the presence of apparently physiological levels of those risk factors. Family and twin studies suggest a strong genetic influence on premature CAD in particular. Results from the Framingham Offspring Study (6) demonstrate that, after correction for known risk factors, parental CVD was associated with a 1.7- and 2.0-fold increased risk for women and men, respectively.

The metabolic syndrome

The metabolic syndrome is defined as the association of several metabolic abnormalities including visceral adipose tissue mass (ie, toxic waist), dyslipidemia (elevated triglycerides and low HDL-C), elevated blood pressure and elevated serum glucose. Several classifications of the metabolic syndrome share common elements that emphasize the increase of cardiometabolic risk factors (8). However, a uniform classification of the metabolic syndrome remains elusive. The IDF classification (10) has more stringent waist circumference criteria than the National Cholesterol Education Program Adult Treatment Panel-III (NCEP ATP-III) definition (3) and serves as the current diagnostic classification system recommended by the writing group (Table 2). Individuals with the metabolic syndrome are more likely to be at higher long-term CVD risk than estimated by the FRS alone. Currently, there is a paucity of data on the clinical usefulness of the new IDF definition of the metabolic syndrome to identify subjects with an intermediate FRS who may be at higher risk for cardiovascular events. A retrospective analysis of data from the Air Force/Texas Coronary Atherosclerosis Prevention Study (AFCAPS/TexCAPS) suggests that determining the presence of the metabolic syndrome using the NCEP ATP-III definition identifies subjects with an FRS of less than 20% who have a similar risk to those without the metabolic syndrome with an FRS of 20% or greater. Results from meta-analyses (28) suggest that there is a 1.5-fold increase in risk when adjusted for other cardiovascular risk factors and that the increase in risk was higher among women than among men. Therefore, some subjects in the higher range of intermediate FRS with the metabolic syndrome may require lipid-lowering therapy to reduce their cardiovascular risk (class IIb, level C). However, no study has thus far demonstrated an improvement in outcome when subjects at intermediate risk were selected for lipid-lowering treatment on the basis of the metabolic syndrome. The measurement of hs-CRP may provide further help in the risk stratification of subjects with the metabolic syndrome (29). As a practical rule, an adult with the metabolic syndrome is extremely unlikely to truly be at low risk for CVD; most are either at intermediate or high risk for CVD. The FRS is a good starting point for the global risk assessment of patients with the metabolic syndrome, as well as for those without the metabolic syndrome. We recommend that clinical judgement be used in some cases to move a patient up an FRS-determined risk score category based on his or her 'load' of metabolic risk factors or the 'severity' of the metabolic syndrome.

Other risk factors

Many other factors have been shown to be associated with increased CVD risk. These include specific lipoprotein subclasses, including lipoprotein(a) (30), inflammatory biomarkers such as lipoprotein-associated phospholipase A_2 (also called platelet-activating factor acetyl hydrolase) (31), cell adhesion molecules, homocysteine, uric acid, coagulation and a variety of thrombosis parameters, serum glycoproteins, and both anatomical and functional measures of vascular health available through an explosion of new imaging techniques, many of which are noninvasive (32). Despite an increasing number of new potential markers of risk, the traditional CVD risk factors remain the priorities for screening and treatment as appropriate. Unless a novel risk factor or marker has been proven to both influence clinical decision making and therapeutic approaches, and to change clinical outcomes, its use should remain within the specialized clinical and research setting (32).

RISK ASSESSMENT

Cardiovascular risk assessment remains imperfect. The FRS (Supplementary Tables 4A and 4B for men, and Supplementary Tables 5A and 5B for women) for total CVD is now recommended (33). The FRS has been shown to underestimate risk in specific categories of patients, especially in youth and women, and possibly in those with the metabolic syndrome (28). Arbitrarily, an FRS of 20% or greater at 10 years is considered to identify subjects at high risk for CVD events. The FRS has been validated in Canada with the Cardiovascular Life Expectancy Model (www.chiprehab.com) (34), and this model has been shown to increase adherence to therapeutic measures. The Reynolds Risk Score (RRS) constitutes an optional risk engine and includes the conventional CVD risk factors in addition to family history and hs-CRP (35,36) (http://www.reynoldsriskscore.org). It has been validated in men and women in an American population, but not yet in Canada. The Internet-based version of the RRS is now also available in mmol/L.

Short-term versus long-term risk

The FRS is applicable to a large percentage of the Canadian population and provides a reasonable estimate of the 10-year risk of a major CVD event. A family history of premature CAD is considered to increase the risk by 1.7-fold in women and 2.0-fold in men. An elevated hs-CRP level is also a modulator of risk, especially in the moderate-risk category (6). Many subjects at low or moderate short-term (10-year) risk are at a high risk over the long term due to the cumulative effects of single but significant elevated risk factors (eg, severe systemic hypertension), the exponentially interactive effects of multiple but only moderately elevated CVD risk factors and/or changes in risk factors over time (for example, the young person with diabetes). In the Framingham study, men in the lowest FRS tertile at 50 years of age experienced a 10-year cumulative risk of one in 25, but a lifetime risk of nearly one in two. Women in the lowest FRS tertile of risk at 50 years of age had a 10-year cumulative risk of one in 50, but a lifetime risk of one in four (37,38). CVD risk should be reassessed every three years (class IIB, level C). European guidelines use a risk score based on total mortality (39).

Risk levels

High risk: Subjects are considered to be at high CVD risk if they have any of the following:

- Evidence of atherosclerosis – vascular bruits, an ankle-brachial index of less than 0.9, documented CAD by invasive or noninvasive

testing, coronary angiography, nuclear imaging, stress echocardiography, previous MI, coronary revascularization (percutaneous coronary intervention, coronary artery bypass graft surgery) and other arterial revascularization procedures, cerebrovascular accident, including transient ischemic attack, evidence of carotid disease by carotid ultrasonography or angiography, or peripheral vascular disease;
- Men older than 45 years and women older than 50 years of age with diabetes, as well as some younger people with diabetes who have an additional risk as per Canadian Diabetes Association guidelines (40); or
- A calculated FRS or RRS of 20% or greater for 10-year risk of CVD. These subjects should receive intensive lifestyle modification advice and benefit from a pharmacological approach aimed at lowering serum LDL-C.

Moderate risk: Many middle-aged Canadians will be in the moderate-risk category. The increase in obesity in the adult population, coupled with an increase in the prevalence of the individual components of the metabolic syndrome, has created a major health concern. This was recently addressed at the federal level in the Canadian Heart Health Strategy and Action Plan (http://www.chhs-scsc.ca/web/). Subjects are considered to be at moderate risk when their FRS is 10% to 19% at 10 years (33). This risk is further modulated by a family history of premature CAD and high hs-CRP.

Alternatively, the RRS, which combines the Framingham risk factors, family history and hs-CRP, can be considered for use to stratify risk (35,36). The indications for pharmacological interventions are based on primary prevention studies including AFCAPS/TexCAPS (41), the West of Scotland Coronary Prevention Study (WOSCOP) (42), the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) (43), the Heart Protection Study (HPS) (44) and JUPITER (5). Following the initiation of health behaviour interventions, pharmacological therapy is indicated if:
- the LDL-C is greater than 3.5 mmol/L (apoB higher than 1.00 g/L) (class IIa, level A);
- the TC/HDL-C ratio is higher than 5.0 (class IIa, level C); or
- the hs-CRP is higher than 2 mg/L in men older than 50 years and in women older than 60 years of age, irrespective of LDL-C (class IIa, level B).

The measurement of hs-CRP should not be performed on everyone. Men older than 50 years and women older than 60 years of age who are at moderate risk for CVD (determined by FRS) and whose level of LDL-C is less than 3.5 mmol/L are candidates because such individuals have been shown to benefit from statin therapy (5) (class IIa, level B). Subjects should be free of acute illness and the lower of two values, taken at least two weeks apart, should constitute the baseline value.

Although widespread pharmacological therapy for those at low risk is not recommended, subjects whose 10-year risk for CVD is estimated to be between 5% and 9% have been shown in randomized controlled trials (5) to achieve the same RR reduction from statin therapy as those at a higher 10-year risk (25% to 50% reduction in events). However, the absolute benefit of therapy is estimated to be smaller (in the order of 1% to 5% reduction of CVD), the numbers needed to treat to prevent one cardiac event are higher and the cost/benefit ratio of therapy is less favourable than for those at a higher risk for CVD events. For individuals in this category, the physician is advised to discuss these issues with the patient and integrate the patient's beliefs regarding the benefits and risks of long-term preventive cholesterol-lowering therapy into the final individualized treatment decision.

Low risk: The low-risk category applies to individuals with an FRS of less than 10%. Pharmacological lipid-lowering treatment is advised for low-risk subjects with severe dyslipidemia (LDL-C of 5.0 mmol/L or greater), usually reflecting a genetic lipoprotein disorder, especially familial hypercholesterolemia (class 1, level C). Consideration for lipid-lowering therapy may also be indicated in subjects at low risk with a TC/HDL-C ratio of greater than 6.0 (class IIb, level C). This especially applies to patients with severe hypertriglyceridemia, in whom treatment may be indicated to reduce the risk of pancreatitis. The need for treatment of subjects with isolated HDL-C is a subject of debate because evidence that pharmacological treatment will reduce cardiovascular risk is lacking and currently available therapies may not increase HDL-C to a clinically significant extent. Clinical judgment should be used concerning the proper timing for the initiation of pharmacological therapy in these patients. A careful family history should be taken and the presence of additional CVD risk factors may indicate the need for intervention in selected individuals. The RRS has the potential to reclassify low-risk patients according to the FRS when there is a family history and elevated hs-CRP.

Ethnic differences in CAD risk

CAD rates vary among ethnic groups in Canada, with the highest incidence among individuals of South Asian ancestry and the lowest among individuals of Chinese ancestry (45). The higher risk among individuals of South Asian ancestry is partly explained by an increased prevalence of abdominal obesity, glucose intolerance, hypertriglyceridemia and low HDL-C. Individuals of First Nations ancestry are also at markedly increased risk for diabetes and CAD (46). For these reasons, the risk stratification approach provides an opportunity for greater focus on overweight and obese individuals, as well as patients with other related metabolic features, which should help ensure identification of modifiable CVD risks, even within those populations unique to the Canadian sociocultural milieu.

TREATMENT TARGETS

Cholesterol treatment target levels are derived from clinical trials. Nearly all studies have measured the serum (or plasma) level of LDL-C as an indicator of response to therapy (Table 3). The Cholesterol Treatment Trialists (CCT) meta-analysis (17) of 14 statin trials showed a dose-dependent relative reduction in CVD with LDL-C lowering. Every 1.0 mmol/L reduction in LDL-C is associated with a corresponding 20% to 25% reduction in CVD mortality and nonfatal MI. Data from the Pravastatin or Atorvastatin Evaluation and Infection Therapy (PROVE-IT) (47), Treating to New Targets (TNT) (48), Aggrastat to Zocor (A to Z) (49), Incremental Decrease in End Points Through Aggressive Lipid Lowering (IDEAL) (50) and the Study of the Effectiveness of Additional Reductions in Cholesterol and Homocysteine (SEARCH) (18) trials have confirmed that lowering LDL-C to a mean of 2.0 mmol/L or less is associated with the lowest risk of recurrent CVD events in secondary prevention patient populations (51). Extrapolating from the available data, a 2.0 mmol/L absolute reduction or a 50% relative reduction in LDL-C provides optimal benefit in terms of CVD reduction (52). Thus, for high-risk subjects, the target levels should be an LDL-C of less than 2.0 mmol/L, or a 50% or greater reduction from baseline LDL-C (class I, level A). In the majority of patients, this is achievable with statin monotherapy. Furthermore, because apoB levels have so frequently been measured in outcome studies in parallel with LDL-C, apoB can be substituted for LDL-C (53,54). The present version of the guidelines recommends apoB as the primary alternate target to LDL-C. Based on the available evidence, many experts have concluded that apoB is a better marker than LDL-C for the risk of vascular disease and a better index of the adequacy of LDL-lowering therapy than LDL-C (53). Also, there now appears to be less laboratory error in the determination of apoB than LDL-C, particularly in patients with hypertriglyceridemia, and all clinical laboratories could easily and inexpensively provide standardized measurements of apoB. However, not all experts are fully convinced that apoB should be measured routinely and, in any case, apoB is not presently being measured in most clinical laboratories. Consequently, a substantial educational effort for patients and physicians would be required for the most effective introduction of apoB into widespread clinical practice. Nevertheless, all would agree that physicians who wish to use apoB in their clinical care should be encouraged to do so. Furthermore, the present compromise approach represents a positive transitional phase in the assessment of lipid parameters to improve the prevention of CVD through the

clinical measurement of apoB. The apoB target for high-risk subjects is less than 0.80 g/L (class I, level A).

Targets other than LDL-C (or apoB)

Secondary targets have been determined in post hoc analyses or as part of prespecified analyses in a number of clinical trials. These secondary targets include a TC/HDL-C ratio of less than 4.0, a non-HDL-C level of less than 3.5 mmol/L, an apoB/apoAI ratio of less than 0.80, a triglyceride level of less than 1.7 mmol/L and an hs-CRP level of less than 2.0 mg/L. Adjusting lipid-lowering therapy to optimize one or more of these secondary targets may be considered in the high-risk patient after achieving a target LDL-C or apoB, but the clinical advantages of this approach, with respect to patient outcomes, remain to be proven.

The specific target for non-HDL-C should be less than 3.5 mmol/L (33). A TC/HDL-C ratio of less than 4.0 or an apoB/apoAI ratio of less than 0.8 is inferred from clinical trials and epidemiological data to convey reduced CVD event risk in high-risk subjects. To date, no specific targets for HDL-C or triglyceride levels have been determined in clinical trials, although increases in HDL-C predict atherosclerosis regression (55) and low HDL-C is associated with excess events and mortality in CAD patients, even when LDL-C is lower than 1.8 mmol/L (56). A specific target for hs-CRP in secondary prevention is based on the predetermined analysis (51) of the PROVE-IT and A to Z studies, which showed that patients with CAD who have reached both an LDL-C level of less than 2.0 mmol/L and an hs-CRP level of less than 2.0 mg/L had the lowest CVD event rate (class IIa, level B). Similarly, an analysis (57) of the JUPITER trial showed that the lowest cardiovascular event rate was achieved in subjects who attained both an LDL-C level of less than 2.0 mmol/L and an hs-CRP level of less than 2.0 mg/L. To date, no clinical trial has addressed the issue of treating the secondary targets of therapy more aggressively, including hs-CRP, once LDL-C (or apoB) is at target. Presently, hs-CRP as a secondary target of therapy is not recommended based on the lack of clinical trial evidence that targeting a particular hs-CRP level results in clinical benefit. Thus, clinicians must exercise expert judgment and caution when considering further treatment intensification in secondary prevention or in high-risk primary prevention. Although several clinical trials are ongoing, to date, no statin-based combination therapy has been shown to improve clinical outcomes.

The target level for subjects at moderate risk are extrapolated from high-risk clinical studies, especially ASCOT (43), HPS (44), AFCAPS/TexCAPS (41), WOSCOP (42) and JUPITER (5). The 2006 recommendations also focused on LDL-C as the primary target of therapy in these patients, with a treatment trigger LDL-C level of 3.5 mmol/L and a recommended 40% reduction (as was obtained in the ASCOT trial [43]), thus reaching a level close to 2.0 mmol/L. Based in large part on the JUPITER trial (5), in which a 50% reduction in LDL-C was achieved, we recommend the same targets of an LDL-C level of lower than 2.0 mmol/L (apoB lower than 0.80 g/L) or a 50% reduction from baseline LDL-C (class IIa, level A) when the baseline level is known. For the above reasons, secondary targets of therapy in the moderate-risk category are based on data extrapolation and therefore, clinical judgment is required before a final treatment plan is implemented (class IIb, level C). These revised recommendations are more stringent than the previous set (3). Clinicians should exercise judgement to avoid premature or unnecessary implementation of lipid-lowering therapy. Health behaviour interventions will have an important long-term impact on health and the long-term effects of pharmacotherapy must be weighed against potential side effects. A meta-analysis of statin trials (17) has demonstrated that for each 1.0 mmol/L decrease in LDL-C, there is a corresponding RR reduction of 20% to 25%. Intensive LDL-C lowering therapy is associated with a decreased risk of CVD events (18).

Congestive heart failure due to systolic dysfunction or end-stage renal disease

Recent studies (Controlled Rosuvastatin Multinational Trial in Heart Failure [CORONA] [58] and Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico Heart Failure [GISSI-HF] [59]) have addressed the issue of statin treatment in end-stage heart failure (left ventricular ejection fraction of less than 30%). These studies suggest that statin therapy does not reduce CVD morbidity or mortality in advanced heart failure of ischemic or nonischemic etiology. Similarly, the Deutsche Diabetes Dialyse Studie (4D) (60) and A study to evaluate the Use of Rosuvastatin in subjects On Regular haemodialysis: an Assessment of survival and cardiovascular events (AURORA) (61) trials examined statin treatment in hemodialysis subjects (who were not considered to be candidates for statin therapy by their physicians) and found no effect on CVD outcomes. Clinical judgement must be applied when considering the modest baseline elevation of LDL-C in these trials (approximately 3.5 mmol/L) and also the observation that patients on dialysis awaiting renal transplantation may still benefit from statins.

Surrogate markers of CVD risk testing for atherosclerosis

The ankle-brachial index is the ratio of systolic blood pressure in the dorsalis pedis or posterior tibial artery to the systolic blood pressure in the brachial artery. An ankle-brachial index value of less than 0.90 is a reliable index of peripheral arterial disease, with a sensitivity of 90% and a specificity of 98% for detecting greater than 50% stenosis. Such patients have a high likelihood of concomitant CVD (62).

Exercise stress testing in asymptomatic men older than 40 years of age can also be useful in risk stratification (63). A positive stress test is highly predictive of CAD and future cardiovascular events. However, the likelihood of detecting asymptomatic CAD remains low when the pretest probability is low. Furthermore, a negative stress test has a low negative predictive value, particularly in patient populations with a higher pretest probability of CVD.

Carotid B-mode ultrasonography is also useful in assessing preclinical atherosclerosis. In asymptomatic individuals 50 years of age or older, several studies have demonstrated up to a fivefold increase in future risk of CAD events when the carotid intima-media thickness (CIMT) is greater than 1 mm, although a better measurement would be a CIMT of greater than the 75th percentile for age, sex and ethnic background (64). A screening strategy, based on carotid ultrasonography, was recently proposed (64). Although CIMT quantification is not yet a standard measure, evidence of early carotid atherosclerosis (visible arterial wall plaques or IMT of 1.5 mm or greater) by routine carotid ultrasonography is probably an indication for statin therapy. Some believe that noninvasive imaging, especially in the moderate-risk category, may be useful to identify patients with undiagnosed, subclinical atherosclerosis. The presence of atherosclerosis places the individual in the high-risk category (class IIa, level C).

Cardiac computed tomography (electron-beam computed tomography) and multidetector computed tomography coronary angiography quantify the burden of coronary artery calcium and can be useful in risk prediction. Importantly, not all plaques are calcified and calcium cannot be used to reliably identify plaques at risk for rupture (65). Even so, the negative predictive value of a coronary artery calcium score of 0 remains very high (greater than 98%) for ruling out significant coronary atherosclerosis or the development of coronary events (65). Noninvasive imaging of the coronary arteries requires computerized gated images of the heart, frequently with pharmacologically induced bradycardia to improve image quality. While not as sensitive as coronary angiography (66), it may be useful for the differential diagnosis of chest pain in highly selected patients. It is not recommended for screening in asymptomatic subjects.

TREATMENT

Health behaviours

Health behaviour interventions remain the cornerstone of chronic disease prevention, including CVD prevention. They should be universally applied for the prevention of chronic diseases such as obesity, type 2 diabetes, atherosclerosis, cancer and neurodegenerative diseases. The major recommended health behaviour interventions are:

- Smoking cessation, including the use of pharmacological therapy as required;

- A diet low in sodium and simple sugars, with substitution of unsaturated fats for saturated and trans fats, as well as increased consumption of fruits and vegetables;
- Caloric restriction to achieve and maintain ideal body weight;
- Moderate to vigorous exercise for 30 min to 60 min most (preferably all) days of the week;
- Psychological stress management; and
- Alcohol consumption in moderation is not contraindicated if there are no metabolic or clinical contraindications (67).

Smoking cessation: Smoking cessation is probably the most important health behaviour intervention for the prevention of CVD. There is a linear and dose-dependent association between the number of cigarettes smoked per day and CVD risk (24). Pharmacological therapy is associated with an increased likelihood of smoking abstinence.

Diet: Recommendations regarding the type of diet favouring health maintenance have been fraught with controversy. Most authorities agree that reducing saturated fats and refined sugars in the diet, while increasing fruits, vegetables and fibres, is associated with increased health. For patients with hypertriglyceridemia, a reduction in the intake of alcohol and refined carbohydrates, in conjunction with increased consumption of omega-3 and omega-6 polyunsaturated fats, is indicated. Most important is the restriction of caloric intake to achieve and maintain a healthy body weight. In Caucasians, a BMI of less than 25 kg/m^2 is considered optimal, while in subjects of Asian, Chinese and Japanese descent, a lower BMI (less than 23 kg/m^2) may be indicated. The dietary content (percentage of protein, carbohydrate and fat) required to maintain a healthy weight does not appear to matter as long as caloric intake is reduced (68). A diet suited to the individual that provides adequate nutrition with a balance between caloric intake and energy expenditure, is best. Often, a professional dietician is of value to provide advice and follow-up. Moderate alcohol intake is acceptable (one drink per day for women and two drinks per day for men) if no metabolic or clinical contraindications are present (67).

Exercise: Physical activity is another important component of prevention. Many studies have shown the benefits of regular exercise in maintaining health and preventing CVD. Regular exercise also has beneficial effects on diabetes risk, hypertension and hypertriglyceridemia, and improves plasma levels of HDL-C. In several studies, a lower frequency of CVD was noted in physically active individuals independent of known CVD risk factors. A general recommendation for healthy individuals is at least 30 min to 60 min of moderate to vigorous physical activity on most, but preferably all, days of the week.

Psychological factors: The INTERHEART study (69) confirmed the importance of stress as a CVD risk factor. Following MI, patients with depression have a worse prognosis, but it remains unclear whether pharmacological treatment reduces this risk (70).

Pharmacotherapy (Table 4)

LDL-C: In high-risk individuals, treatment should be started immediately, concomitant with health behaviour interventions with respect to appropriate diet, physical activity, weight management and the cessation of tobacco consumption. The primary target of therapy is to achieve an LDL-C of less than 2.0 mmol/L, an apoB of less than 0.8 g/L or a 50% reduction in LDL-C from baseline values (class I, level A).

The majority of patients will be able to achieve target LDL-C levels on statin monotherapy. However, a significant minority of patients may require combination therapy with an agent that inhibits cholesterol absorption (ezetimibe) or bile acid reabsorption (cholestyramine, colestipol), or the concomitant use of niacin. These combinations are generally safe and can decrease LDL-C by an additional 10% to 15% for bile acid resins and up to 20% for ezetimibe and niacin. Clinical outcome data on the incremental benefit of combination therapy with statin plus ezetimibe, niacin or fibrate, versus statin monotherapy are lacking, although clinical trials are underway to examine this issue.

Triglycerides: A specific target for triglyceride levels in high-risk subjects or for the primary prevention of CAD has not been established.

TABLE 4
Lipid-lowering medications

Generic name	Trade name (manufacturer)	Recommended dose range (daily)
Statins		
Atorvastatin	Lipitor (Pfizer Canada Inc)	10 mg – 80 mg
Fluvastatin	Lescol (Novartis Pharmaceuticals Canada Inc)	20 mg – 80 mg
Lovastatin	Mevacor (Merck Frosst Canada Ltd)	20 mg – 80 mg
Pravastatin	Pravachol (Bristol-Myers Squibb Canada)	10 mg – 40 mg
Rosuvastatin	Crestor (AstraZeneca Canada)	5 mg – 40 mg
Simvastatin	Zocor (Merck Frosst Canada Ltd)	10 mg – 80 mg*
Bile acid and/or cholesterol absorption inhibitors		
Cholestyramine	Questran (Bristol-Myers Squibb, USA)	2 g – 24 g
Colestipol	Colestid (Pfizer Canada Inc)	5 g – 30 g
Ezetimibe	Ezetrol (Merck Frosst/Schering Pharmaceuticals Canada)	10 mg
Fibrates		
Bezafibrate	Bezalip (Actavis Group PTC EHF, Iceland)	400 mg
Fenofibrate†	Lipidil Micro/Supra/EZ (Fournier Pharma Inc, Canada)	48 mg – 200 mg
Gemfibrozil†‡	Lopid (Pfizer Canada Inc)	600 mg – 1200 mg
Niacin		
Nicotinic acid	Generic crystalline niacin	1 g – 3 g
	Niaspan (Oryx Pharmaceuticals Inc, Canada)	0.5 g – 2 g

**Increased myopathy on 80 mg; †Reduce dose or avoid in renal impairment; ‡Should not be used with a statin because of an increased risk of rhabdomyolysis*

Epidemiological studies show that lower triglyceride levels are associated with decreased CVD risk, and drugs that lower triglycerides have demonstrated a reduction of CVD events in the Helsinki Heart Study (71) and the Veterans Administration HDL Intervention Trial (VA-HIT) (72). In both cases, the drug used was the fibric acid derivative gemfibrozil. Gemfibrozil should not be used with a statin because of the increased risk of rhabdomyolysis. The Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study (73) in diabetic patients using fenofibrate failed to meet its primary end point in terms of CAD prevention. In patients with hypertriglyceridemia, dietary therapy, exercise and weight loss, with a focus on restriction of refined carbohydrates and reduced alcohol intake, in association with increased intake of omega-3 fatty acids, are first-line therapies. The use of fibrates as first-line agents is warranted in patients with extreme hypertriglyceridemia (triglyceride levels greater than 10 mmol/L) to prevent pancreatitis. For patients with moderate hypertriglyceridemia (triglyceride levels of 5 mmol/L to 10 mmol/L), fibrates may be useful, but the impact on CAD prevention is less clear. In high-risk patients already on a statin, elevated triglyceride levels (2 mmol/L to 5 mmol/L) may be further treated with a fibrate or niacin. However, it has not been established whether the addition of a fibrate or niacin to a statin further reduces CAD events once the LDL-C is at target (class IIb, level C).

HDL-C: Smoking cessation, weight loss, exercise and moderate alcohol intake all increase HDL-C. These favourable health behaviours stand on their own merit in terms of benefit over the long term and HDL-C may be a marker of cardiovascular health. There is considerable controversy regarding the treatment of a low HDL-C, in part because there are many genetic forms of HDL-C deficiency that do not increase (or increase only slightly) CVD risk (74). Furthermore, the treatment of a genetic HDL-C deficiency is often difficult with currently available medications (75). Statins have little effect on HDL-C and fibrates only modestly raise HDL-C (5% to 10%) in most cases. Niacin can increase HDL-C by 15% to 25%.

Novel approaches to raise HDL-C are being tested clinically. Despite early disappointing results (76), the data indicate that raising HDL-C may still prove to be a valuable therapeutic target (77).

Combination therapy: The combination of a statin with niacin is effective in improving the lipid profile of patients with combined dyslipidemia and low HDL-C. Niacin is more effective than fibrates in increasing HDL-C concentrations. Side effects are most manifest with crystalline niacin, and include flushing, dry skin, gastritis and worsened glycemic control in persons with diabetes mellitus. Crystalline niacin should be taken two to three times daily after meals and the dose should be increased slowly. Extended-release niacin (Niaspan; Oryx Pharmaceuticals Inc, Canada) is taken once daily and is better tolerated. The use of acetylsalicylic acid (325 mg) 30 min to 60 min before niacin attenuates the flushing in most patients. There is a small but significant risk of hepatotoxicity with niacin monotherapy or niacin plus statin combination treatment and therefore, serum transaminase levels should be followed. Until the results of the Atherothrombosis Intervention in Metabolic Syndrome with low HDL/High Triglyceride and Impact on Global Health Outcomes (AIM-HIGH) (78) and Heart Protection Study 2 Treatment of HDL to Reduce the Incidence of Vascular Events (HPS2-THRIVE) (79) trials using combined statin/niacin in high-risk patients are available, the data supporting the use of niacin are based on small studies not powered for major adverse CVD end points. Gradual titration of niacin and the use of acetylsalicylic acid to decrease flushing symptoms are recommended.

The combination of a statin with a fibrate may be used with close patient follow-up. Because fibrates may increase serum creatinine, the dose must be adjusted in patients with kidney impairment. Fibrates may also increase serum homocysteine levels. It should be noted that the recent FIELD study (73) demonstrated that fenofibrate monotherapy did not significantly reduce CVD events in patients with diabetes and mild hypertriglyceridemia. Available data suggest that fenofibrate is reasonably safe in combination with a statin. Studies are underway to determine whether the addition of a fenofibrate to a statin regimen alters CVD risk. Gemfibrozil is associated with a higher risk of myotoxicity and should not be used in combination therapy. For patients with moderate hypertriglyceridemia, the addition of omega-3 fatty acids (2 g to 4 g three times daily) to statin therapy is safe, and may lower triglycerides and help achieve the TC/HDL-C ratio target.

Safety and laboratory monitoring

Before initiation of pharmacological therapy for dyslipidemias, a baseline lipoprotein profile should be obtained after a 10 h to 12 h fast, preferably with the subject refraining from alcohol for 24 h to 48 h. The lipoprotein profile should include TC, HDL-C and triglycerides. The LDL-C is derived from the Friedewald formula and is considered accurate for triglyceride levels of less than 5 mmol/L. A fasting glucose level should also be obtained at baseline to identify the presence of impaired fasting glucose or diabetes. ApoB and apoAI measurements should be made at the discretion of the physician. Important issues for these newer biochemical analytes include standardization of laboratory measurement proficiency and reimbursement, both of which, at present, vary widely across Canada. ApoB measurement may also be useful for differentiation between familial hypertriglyceridemia and familial combined hyperlipidemia, and in subjects with a low HDL-C. A baseline thyroid-stimulating hormone level helps uncover the occasional hypothyroid-induced hyperlipidemia. Baseline transaminases (alanine aminotransferase [ALT] and aspartate aminotransferase), creatinine and creatine kinase (CK) are useful to monitor potential side effects associated with therapy. The frequency of follow-up measurements is debated but should probably be performed semiannually, or with any changes in lipid-lowering therapy.

Statins are well tolerated by most individuals. Myalgias represent the most common side effect of statins and may occur in approximately 5% of patients, although similar rates are often seen in the placebo groups in clinical trials. Statin-related myalgias are characterized by dull muscle aches and can be made worse by exercise, although they may occur in sedentary patients. Serum levels of CK may remain normal. The diagnosis should be based on drug cessation and re-challenge. Myositis is an inflammation of skeletal muscles and the diagnosis is based on muscle discomfort and elevation of CK to more than three times the upper limit of normal. This is a potentially serious condition and may be caused by strenuous exercise. Dose reduction and close monitoring of CK levels or discontinuation of the statin are often required. Of note, a genetic predisposition to myositis is thought to underlie a number of cases. Rhabdomyolysis is a potentially life-threatening condition with a prevalence of less than 1:100,000 statin-treated patients. It is characterized by severe muscle pains, myoglobinuria and possibly, acute renal failure and a CK level of greater than 10,000 U/L. The discontinuation of statins and prompt hospitalization for supportive treatment is required. Significant increases in hepatic transaminase levels, defined as an ALT level of greater than three times the upper limit of normal, occur in 0.3% to 2.0% of patients and are generally dose related.

Both crystalline niacin and extended-release niacin preparations can result in persistent significant elevations in ALT in approximately 1% of patients. A general recommendation is to measure ALT at baseline, and between one and three months after initiating niacin therapy. Fasting blood glucose and glycosylated hemoglobin should be monitored every six to 12 months in patients treated with niacin, in view of its tendency to raise blood glucose levels. If these parameters deteriorate significantly in patients treated with niacin, consideration should be given to dose reduction or withdrawal of niacin therapy. Uric acid levels should be monitored in patients taking niacin.

Reversible increases in plasma creatinine of 15% to 20% are common in fibrate-treated patients and more significant increases can occur in patients with underlying renal disease. In patients with renal insufficiency (estimated glomerular filtration rate of less than 60 mL/min/1.73 m^2), fibrates should be initiated at the lowest available dose and increased only after re-evaluation of renal function and lipid parameters.

Referral to a specialty clinic, advanced laboratory tests and genetic testing

Physicians are often confronted with issues of drug intolerance, complex diagnostic cases, lack of laboratory resources, seemingly unexplained atherosclerosis, extremes of lipoprotein disorders or a lack of response to conventional therapies. In such cases, referral to a specialized centre may be warranted. Most academic centres across Canada have specialized lipid clinics and the laboratory resources required for more extensive testing. In extreme cases, therapeutic modalities, such as extracorporeal LDL apheresis techniques, are available. We recommend that lipoprotein disorder specialists be available in each province to provide care for more difficult patients referred from primary care physicians.

Genetic testing for severe lipoprotein disorders is available in a few highly specialized centres. However, a molecular genetic diagnosis is not necessary for the majority of patients with severe dyslipidemia; the biochemical and clinical data usually suffice to make a diagnosis. As a research tool, however, the molecular study of extreme lipoprotein disorders has provided considerable scientific insight including the identification of potential future therapeutic targets.

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CANADIAN CHOLESTEROL GUIDELINES 2009: SUMMARY OF RECOMMENDATIONS

SCREENING FASTING LIPID PROFILE

- Screen men who are at least 40 years of age, and women who are at least 50 years of age or postmenopausal.
- Adults with the following risk factors should be screened at any age:
  - Diabetes;
  - Cigarette smoking;
  - Hypertension;
  - Obesity (body mass index greater than 27 kg/m^2);
  - Family history of premature coronary artery disease;
  - Clinical signs of hyperlipidemia;
  - Evidence of atherosclerosis;
  - Rheumatoid arthritis, systemic lupus erythematosis, psoriasis;
  - HIV infection on highly active antiretroviral therapy;
  - Estimated glomerular filtration rate of less than 60 mL/min/1.73 m^2; or
  - Erectile dysfunction.
- Screen children with a family history of hypercholesterolemia or chylomicronemia.

CARDIOVASCULAR RISK ASSESSMENT

Determine risk using the Framingham risk score modified for family history (double the cardiovascular disease risk percentage if any cardiovascular disease is present in a first-degree relative before 60 years of age). In men older than 50 years or women older than 60 years of age, of intermediate risk whose low-density lipoprotein cholesterol does not already suggest treatment, high-sensitivity C-reactive protein can be used for risk stratification.

TARGETS OF THERAPY

Risk level	Primary target: LDL-C	Class, level
High	<2 mmol/L	Class I, level A
CAD, PVD, atherosclerosis	or	
Most patients with diabetes	≥50% ↓ LDL-C	
FRS ≥20%	apoB <0.80 g/L	
RRS ≥20%		
Moderate	<2 mmol/L*	Class IIa, level A
FRS 10% to 19%	or	
LDL-C >3.5 mmol/L	≥50% ↓ LDL-C	
TC/HDL-C >5.0	apoB <0.80 g/L	
hs-CRP >2 mg/L in men >50 years and women >60 years of age		
Family history and hs-CRP modulate risk		
Low	≥50% ↓ LDL-C	Class IIa, level A
FRS <10%		

**Clinicians should exercise judgement when implementing statin therapy. Meta-analysis of statin trials show that for each 1.0 mmol/L decrease in low-density lipoprotein cholesterol (LDL-C), there is a corresponding 20% to 25% RR reduction. Those whose 10-year risk for cardiovascular disease is 5% to 9% have been shown in randomized clinical trials to achieve the same RR reduction from statin therapy as those at higher 10-year risk, but the absolute benefit of therapy is estimated to be smaller. apoB Apolipoprotein B; CAD Coronary artery disease; FRS Framingham risk score; HDL-C High-density lipoprotein cholesterol; hs-CRP High-sensitivity C-reactive protein; PVD Peripheral vascular disease; RRS Reynolds Risk Score; TC Total cholesterol*

Secondary (optional) targets (once low-density lipoprotein cholesterol is at goal)

- Total cholesterol to high-density lipoprotein cholesterol ratio of less than 4.0;
- Non-high-density lipoprotein cholesterol of less than 3.5 mmol/L;
- Triglycerides of less than 1.7 mmol/L;
- Apolipoprotein B to apolipoprotein AI ratio lower than 0.80; and
- high-sensitivity C-reactive protein of less than 2 mg/L.

Clinical trial evidence is lacking for secondary targets; clinical judgements are warranted.

TREATMENT

Health behaviours

- Smoking cessation;
- Diet (reduced saturated fats and refined sugars);
- Weight reduction and maintenance;
- Exercise (daily); and
- Stress management.

Medication

In high-risk patients, pharmacological therapy should be considered concomitantly with lifestyle changes. In moderate-risk patients, lifestyle changes should be implemented first, followed by medications if the targets are not reached.

Generic name	Trade name (manufacturer)	Dose range (daily)
Statins		
Atorvastatin	Lipitor (Pfizer Canada Inc)	10 mg – 80 mg
Fluvastatin	Lescol (Novartis Pharmaceuticals Canada Inc)	20 mg – 80 mg
Lovastatin	Mevacor (Merck Frosst Canada Ltd)	20 mg – 80 mg
Pravastatin	Pravachol (Bristol-Myers Squibb Canada)	10 mg – 40 mg
Rosuvastatin	Crestor (AstraZeneca Canada)	5 mg – 40 mg
Simvastatin	Zocor (Merck Frosst Canada Ltd)	10 mg – 80 mg*
Bile acid and/or cholesterol absorption inhibitors		
Cholestyramine	Questran (Bristol-Myers Squibb, USA)	2 g – 24 g
Colestipol	Colestid (Pfizer Canada Inc)	5 g – 30 g
Ezetimibe	Ezetrol (Merck Frosst/Schering Pharmaceuticals Canada)	10 mg
Fibrates		
Bezafibrate	Bezalip (Actavis Group PTC EHF, Iceland)	400 mg
Fenofibrate†	Lipidil Micro/Supra/EZ (Fournier Pharma Inc, Canada)	48 mg – 200 mg
Gemfibrozil†‡	Lopid (Pfizer Canada Inc)	600 mg – 1200 mg
Niacin		
Nicotinic acid	Generic niacin	1 g – 3 g
	Niaspan (Oryx Pharmaceuticals Inc, Canada)	0.5 g – 2 g

**Simvastatin 80 mg has a higher incidence of rhabdomyolysis; †Reduce dose or avoid in renal impairment; ‡Should not be used with a statin because of an increased risk of rhabdomyolysis*

Other risk factors/risk markers

The clinical usefulness of other risk factors or markers of risk has not been evaluated in large-scale clinical trials.

Noninvasive assessment of atherosclerosis

The determination of the ankle-brachial index, carotid plaque, coronary calcium score or multidetector computed tomography coronary angiography will detect asymptomatic atherosclerosis not always predicted by the cardiovascular risk assessment algorithms.

Follow-up

Most lipid-lowering medications are well tolerated. Serum transaminases and creatine kinase should be followed regularly (every six to 12 months) or when symptoms develop. Follow-up is not required if levels are consistently normal and the patient has no symptoms.

Referral to specialized clinics

Most Canadian universities have a specialized lipid clinic. Cases of unexplained atherosclerosis, severe dyslipidemias, genetic lipoprotein disorders and patients refractory to pharmacological treatment should be referred.

SUPPLEMENTARY INFORMATION

SUPPLEMENTARY TABLE 1
Stakeholders in the elaboration of the Canadian lipid guidelines

Canadian Cardiovascular Harmonization of National Guidelines Endeavor (C-Change). Putting Prevention into Practice
Canadian Association of Cardiac Rehabilitation
Canadian Cardiovascular Society
Canadian College of Family Physicians of Canada
Canadian Council for Tobacco Control
Canadian Council of Cardiovascular Nurses
Canadian Diabetes Association
Canadian Hypertension Society
Canadian Medical Association
Canadian Obesity Network
Canadian Pharmacists Association
Canadian Society for Exercise Physiology
Canadian Stroke Network
Canadian Working Group on Dyslipidemias
Obesity Canada
Public Health Agency of Canada
Royal College of Physicians and Surgeons of Canada
Canadian Institutes of Health Research

SUPPLEMENTARY TABLE 2
Criteria used for evaluation of evidence

Recommendation grade

Class I
Evidence and/or general agreement that a given diagnostic procedure/treatment is beneficial, useful and effective
Class II
Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the treatment
Class IIa Weight of evidence in favour
Class IIb Usefulness/efficacy less well established
Class III
Evidence that the treatment is not useful and in some cases may be harmful

Level of evidence

Level A
Data derived from multiple randomized clinical trials or meta-analysis
Level B
Data derived from a single randomized clinical trial or large nonrandomized studies
Level C
Consensus of opinion by experts and/or small studies, retrospective studies and registries

SUPPLEMENTARY TABLE 3
Major changes since the 2006 recommendations

Involvement of the Canadian Vascular Coalition and the Canadian Institutes of Health Research
Secondary and high-risk prevention
Strategy better defined
Clinical studies on end-stage disease (advanced heart failure and hemodialysis)
Primary prevention
Cardiovascular risk evaluation tools
Framingham risk score includes cardiovascular diseases
Intermediate risk defined as a Framingham risk score of 10% to 19% for 10-year risk
Family history part of risk stratification
High-sensitivity C-reactive protein part of risk stratification in intermediate-risk subjects whose low-density lipoprotein cholesterol level does not already suggest treatment (men older than 50 years and women older than 60 years of age)
Targets
Simplified target levels
Apolipoprotein B role defined
Secondary targets evaluated according to available evidence

SUPPLEMENTARY TABLE 4A
Estimation of 10-year risk of total cardiovascular disease in men (Framingham Heart Study)

POINTS	Age	HDL-C	Total Cholesterol	SBP Not Treated	SBP Treated	Smoker	Diabetic	
–2		>1.6		<120				
–1		1.3-1.6						
0	30-34	1.2-1.3	<4.1	120-129	<120	NO	NO	
1		0.9-1.2	4.1-5.2	130-139				
2	35-39	<0.9	5.2-6.2	140-159	120-129			
3			6.2-7.2	160+	130-139		YES	
4			>7.2		140-159	YES		
5	40-44				160+			
6								
7	45-49							
8	50-54							
9								
10	55-59							
11	60-64							
12								
13	65-69							
14	70-74							
15	75+							TOTAL POINTS
Points Allotted								

Adapted from reference 33. HDL-C High-density lipoprotein cholesterol; SBP Systolic blood pressure

SUPPLEMENTARY TABLE 4B
Cardiovascular disease risk for men

Points	Risk, %	Points	Risk, %	Points	Risk, %
–3 or less	<1	5	3.9	13	15.6
–2	1.1	6	4.7	14	18.4
–1	1.4	7	5.6	15	21.6
0	1.6	8	6.7	16	25.3
1	1.9	9	7.9	17	29.4
2	2.3	10	9.4	18+	>30
3	2.8	11	11.2		
4	3.3	12	13.3		

SUPPLEMENTARY TABLE 5A
Estimation of 10-year risk of total cardiovascular disease in women (Framingham Heart Study)

POINTS	Age	HDL-C mmol/L	Total Cholesterol	SBP Not Treated	SBP Treated	Smoker	Diabetic	
–3				<120				
–2		>1.6						
–1		1.3-1.6			<120			
0	30-34	1.2-1.3	<4.1	120-129		NO	NO	
1		0.9-1.2	4.1-5.2	130-139				
2	35-39	<0.9		140-149	120-129			
3			5.2-6.2		130-139	YES		
4	40-44		6.2-7.2	150-159			YES	
5	45-49		>7.2	>160	140-149			
6					150-159			
7	50-54				160+			
8	55-59							
9	60-64							
10	65-69							
11	70-74							
12	75+							TOTAL POINTS
Points Allotted								

Adapted from reference 33. HDL-C High-density lipoprotein cholesterol; SBP Systolic blood pressure

SUPPLEMENTARY INFORMATION – CONTINUED

SUPPLEMENTARY TABLE 5B
Cardiovascular disease risk for women

Points	Risk, %	Points	Risk, %	Points	Risk, %
–2 or less	<1	6	3.3	14	11.7
–1	1.0	7	3.9	15	13.7
0	1.2	8	4.5	16	15.9
1	1.5	9	5.3	17	18.51
2	1.7	10	6.3	18	21.5
3	2.0	11	7.3	19	24.8
4	2.4	12	8.6	20	27.5
5	2.8	13	10.0	21+	>30

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Notes

09:30 - 10:00 Cerebrovascular Accident: Recognition and Management

Theodore Wein MD, FRCPC

Department of Neurology & Neurosurgery, Montreal Neurological Hospital

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Research interests: Dr. Wein obtained his medical degree at the University of Vermont college of medicine and completed his training in neurology at McGill University. Subsequently he completed a fellowship in cerebrovascular disease at the unviersity of texas at houston and a neuromuscular fellowship at the University of Michigan. Currently, Dr. Wein works at the stroke prevention clinic at the montreal general hospital as well as at st mary's hospital. He is particularly interested in primary and secondary stroke prevention as well as stroke rehabilitation. He has numerous publications in the field of stroke and currently serves as a member of the American Heart Association stroke council.

Learning Objectives

1. To establish that stroke in not an "accident" but a preventable disease
2. Emphasize the need for patient and family education regarding recognition of stroke symptoms
3. To see the influence on Lifestyle as a predictor of stroke
4. Review current AHA/ASA guidelines on stroke prevention

"Anyone who sits around idle and takes no exercise will be subject to physical discomfort and failing strength."

Ivan Rohan M.D., CCFP
McGill University
Montréal

EXERCISE -Prescription
How can family doctor improve compliance

Educational goal:
To familiarize the participants with:
The benefits, risks, recommendations, guidelines and monitoring of exercise.

Look at the strategies to improve the compliance with exercise.

To review some specific conditions:
Cardiac, diabetic, elderly, women, children.

EXERCISE -PRO'S AND CON'S
How can family doctor improve compliance

- BENEFITS OF EXERCISE
- Risks of exercise
- Recommendation – Guidelines type, frequency, intensity
- Monitoring of exercise
- Specific conditions – Cardiac, Diabetes, Elderly, Women, Children
- Compliance issues

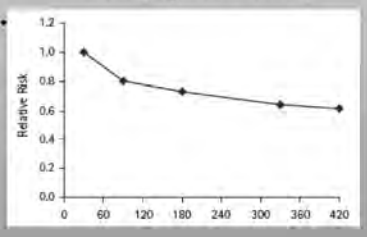
BENEFITS OF EXERCISE

- All-cause morbidity and mortality
- Cardiovascular health
- Obesity
- Diabetes mellitus
- Psychological
- Cognition
- Other - cancer prevention…

Health benefits

- 60 min/week - benefits start
- 150 min/week - most benefits
- 300 min/week - more benefits

All cause mortality declines with more physical activity



CARDIOVASCULAR BENEFITS

- All CV disease incidence and mortality
- Coronary heart disease
- Multiple metabolic risk factors
- Lipids
- Hypertension
- Stroke

Psychological benefits

- Depression
- Anxiety, stress
- Well-being, self image
- Adjunct in alcohol and substance abuse

Complications and risks of exercise

BENEFITS FAR OUTWEIGH THE RISKS

Complications and risks of exercise

- Injuries
- Overuse syndromes
- Exhaustion, heat stroke, dehydration
- Hypoglycemia in diabetics
- Myocardial infarction irregular vigorous exercise
- Sudden death - rare

Adverse Events

- **Moderate-intensity physical activity**, such as brisk walking, has a **low risk** of such **adverse events.**
- **The risk of musculoskeletal injury** increases with the total amount of physical activity. However, people who are physically active may have fewer injuries from other causes, such as motor vehicle collisions or work-related injuries.
- Participation in **contact or collision sports**, such as soccer or football, has a higher risk of injury than participation in non-contact physical activity, such as swimming or walking.
- **Cardiac events, such as a heart attack or sudden death** during physical activity, are **rare**. However, the risk of such cardiac events does increase when a person suddenly becomes much more active than usual. The greatest risk occurs when an adult who is usually inactive engages in **vigorous-intensity activity** (such as shovelling snow).

MI and exercise

MI risk during heavy exertion:

- 2.4x increase in regular exercisers
- 60 - 107x increase in irregular exercisers
- Higher risk in diabetics and the difference not fully accounted for the lack of regular exercise

Sudden death

- In young
- Middle age and older

1 death / 50,000 participants
1 death / 215,000 hours of competition
1 death / 396,000 exercise hours
1 cardiac arrest / 4,800,000 exercise hours

Prevention of cardiac events

- Screening is generally poor
- Teaching of cardiac symptoms typical and even less typical
- CAD patients should be encouraged to exercise
- But they should avoid vigorous exercise

Recommendations, Guidelines for Exercise

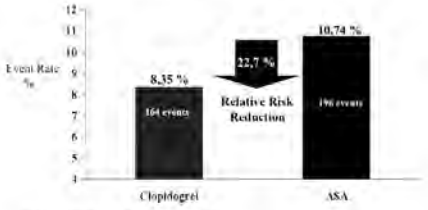
- TYPE
- FREQUENCY
- INTENSITY

3 Main kinds of exercise

- Aerobic
- Muscle strengthening
- Bone strengthening

- 2 other activity – Balance, Stretching

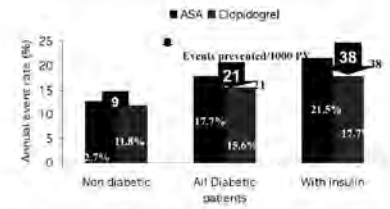
Benefit of Clopidogrel in Patients with polyvascular disease



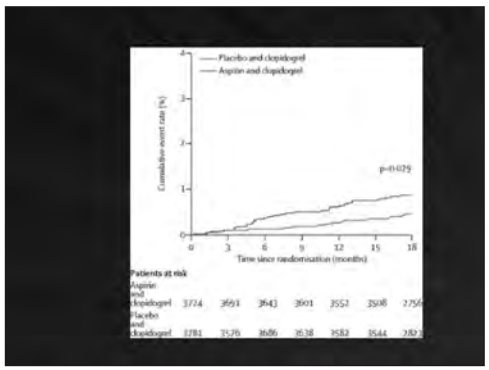
* Ischemic stroke, MI, vascular death

Lancet, 1996; 348(9038), p. 1329-39

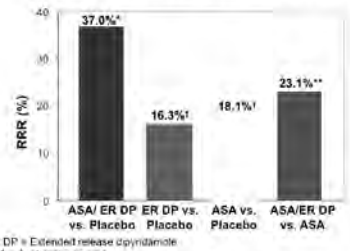
Additional CAPRIE Analyses: Risk Reduction in Patients with Diabetes



Events = ischemic stroke, MI, vascular death, hospitalization for ischemic events/bleeding.
Overall benefit p=0.032, multivariate analysis



ESPS-2: Effects on Stroke – RRR



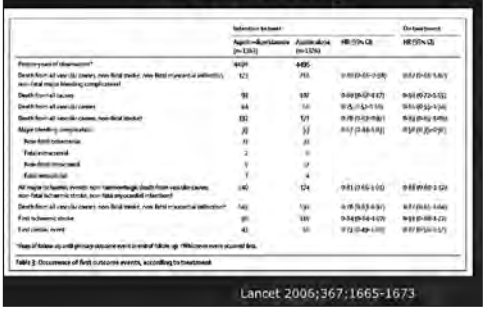
ER DP = Extended release dipyridamole
ASA = Acetylsalicylic acid
RRR = Relative Risk Reduction
* p<0.001, **p<0.006, †p<0.05

Diener et al. *J Neurol Sci* 1997;151:S1-S77
Diener et al., *J Neurol Sci* 1996;143:1-13

ESPS-2 Secondary Endpoint MI

Diener et al., Journal of Neurological Sciences, 1996;143:1-13.

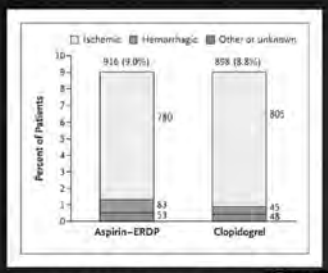
ESPRIT TRIAL



Lancet 2006;367:1665-1673

PROFESS

Frequency of Types of Recurrent Stroke among the Study Patients, According to Treatment Group



Bousser M et al. N Engl J Med 2008;10.1056/NEJMoa0805002
The NEW ENGLAND JOURNAL of MEDICINE

Treatment Algorithm

Not on treatment

Plavix>/=ASA/Dipyridamole > ASA 50-325 mg

ASA Allergic → Plavix 75 mg

Acute MI/Angina
PVD
DM

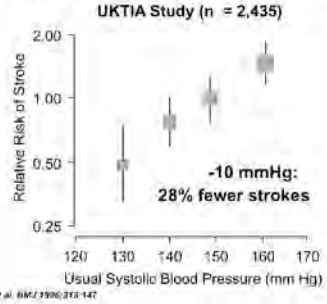
Table 3. AHA/ASA Recommendations for BP Management for Prevention of Recurrent Stroke[35]

1. Antihypertensive treatment is indicated to prevent recurrent stroke and other vascular events; for ischemic stroke, treatment is initiated beyond the hyperacute stroke period.
2. The absolute target BP level is uncertain and should be individualized by patient; benefit may be noted with an average BP drop of 10/5 mm Hg.
3. Lifestyle modifications are included as part of a comprehensive approach.
4. Choice of antihypertensive drug(s) remains uncertain; a diuretic or diuretic plus an angiotensin-converting enzyme inhibitor is a reasonable choice; however, compelling indications may exist for a specific class of antihypertensive and use of the JNC 7 report is recommended.

Aiyagari and Gorelick. Stroke 2009;40:2251-2256.

BP and *secondary* stroke

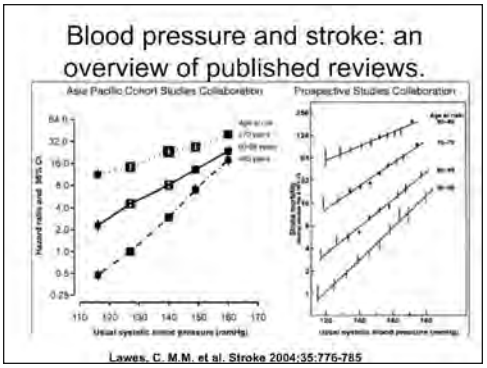
UKTIA Study (n = 2,435)

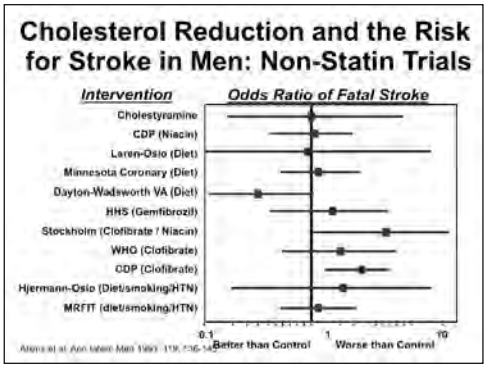


Hodgins et al. BMJ 1996;313:147

Benefit of BP lowering and stroke reduction

- A 10 mmHg reduction in SBP is associated with a reduction in risk of stroke of approximately one third. This association is continuous down to levels of at least 115/75 mmHg.
- A 10 mmHg reduction in SBP should be associated with a reduction in risk of stroke of 31% (R=0.71) in a few years. Similarly, there is a risk reduction in stroke of 35% for those ages 60-69, and 30% for those >70 years old.



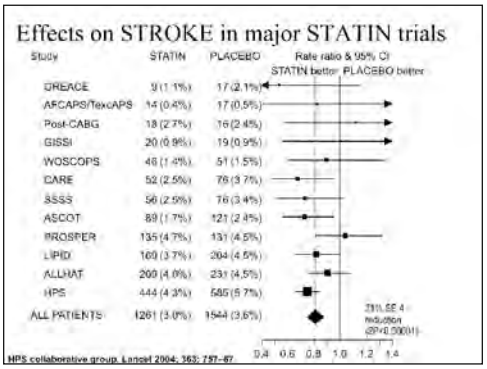


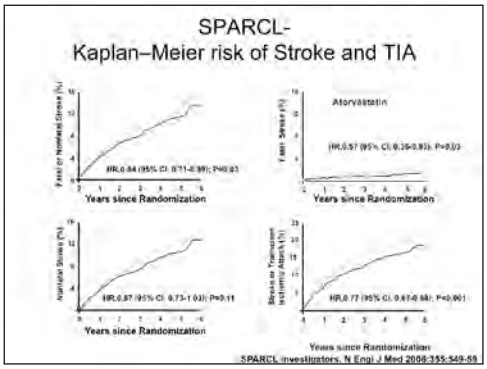
Inverse association of dietary fat with development of ischemic stroke in men.

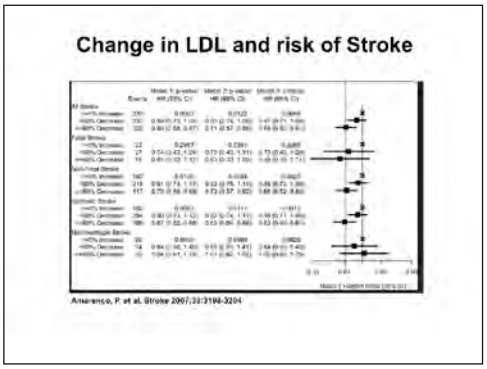
Gillman MW, Cupples LA, Millen BE, Ellison RC, Wolf PA. JAMA:1997;2146-50

Department of Ambulatory Care and Prevention, Harvard Medical School and Harvard Pilgrim Health Care, Boston, MA 02215, USA.

CONTEXT: A few ecological and cohort studies in Asian populations suggest an inverse association of the intake of both fat and saturated fat with risk of stroke. However, data among western populations are scant. OBJECTIVE: To examine the association of stroke incidence with intake of fat and type of fat among middle-aged US men during 20 years of follow-up. DESIGN AND SETTING: The Framingham Heart Study, a population-based cohort study. PARTICIPANTS: A total of 832 men, aged 45 through 65 years, who were free of cardiovascular disease at baseline (1966-1969). MEASUREMENTS AND DATA ANALYSIS: The diet of each subject was assessed at baseline by a single 24-hour dietary recall, from which intakes of energy and macronutrients were estimated. In Kaplan-Meier analyses, we calculated age-adjusted cumulative incidence rates of stroke. Using Cox regression, we estimated stroke incidence relative risks during 20 years of follow-up. MAIN OUTCOME MEASURE: Incidence of ischemic stroke, which occurred in 61 subjects during the follow-up period. RESULTS: Mean intakes were 10 975 kJ for energy, 114 g (39% of energy) for total fat, 44 g (15%) for saturated fat, 46 g (16%) for monounsaturated fat, and 16 g (5%) for polyunsaturated fat. Risk of ischemic stroke declined across the increasing quintile of total fat (log-rank trend P= 003), saturated fat (P= 002), and monounsaturated fat (P= 008) but not polyunsaturated fat (P= 33). The age- and energy-adjusted relative risk for each increment of 3% of energy from total fat was 0 85 (95% confidence interval [CI], 0 78-0 94), for an increment of 1% from saturated fat, 0 91 (95% CI, 0 85-0 98), and for 1% from monounsaturated fat, 0 85 (95% CI, 0 83-0 96). Adjustment for cigarette smoking, glucose intolerance, body mass index, blood pressure, blood cholesterol level, physical activity, and intake of vegetables and fruits and alcohol did not substantially change the results. Few cases of hemorrhagic stroke (n=14) occurred in these analyses. CONCLUSION: Intakes of fat, saturated fat, and monounsaturated fat were associated with reduced risk of ischemic stroke in men.







Stroke May 2006

Table 2 Recommendations for Lipid Management

Recommendations	Class, Level of Evidence
Class I Recommendations	
Ischemic stroke or TIA patients with elevated cholesterol, coronary artery disease, or evidence of atherosclerotic origin should be managed according to NCEP III guidelines, which include lifestyle modification, dietary guidelines, and medication recommendations.	Class I, Level A
Statin agents are recommended, and the target goal for cholesterol lowering for these who CHD or symptomatic atherosclerotic disease is an LDL-C level of <100 mg/dL. An LDL-C <70 mg/dL is recommended for very high-risk persons with multiple risk factors.	Class I, Level B
Best Recommendation	
On the basis of the SPARCL trial, administration of statin therapy with intensive lipid-lowering effects is recommended for patients with atherosclerotic ischemic stroke or TIA and elevated fasting LDL-C to reduce the risk of stroke and cardiovascular events.	Class I, Level B
Class II Recommendation	
Ischemic stroke or TIA patients with low HDL cholesterol may be considered for treatment with statin agents.	Class IIb, Level B

NCEP indicates National Cholesterol Education Panel; LDL-C, low-density lipoprotein cholesterol; and HDL, high-density lipoprotein.

Conclusion

1. CVA – Stroke is not an accident but a preventable disease
2. Patient and family education is needed
3. Lifestyle plays a pivotal role in stroke risk
4. Current AHA/ASA guidelines suggest the use of newer antiplatelets over ASA as well as aggressive lipid lowering
5. BP lowering of 10/5mm Hg is almost 2.5X more effective than ASA for stroke reduction.

10:00 - 10:30 Atrial Fibrilation and Other Arythmias

Magdi Hanna Sami MD

Division of Cardiology, Royal Victoria Hospital – MUHC

Research interests: Dr Magdi Sami, was born in Cairo, Egypt and graduated from Cairo University with honors in 1969. He completed his postgraduate training at McGill and the Montreal Heart Institute from 1971 to 1977. He was awarded a Canadian Heart Foundation fellowship for a 2 Years stint at Stanford University working both in Electrophysiology and post MI risk stratification.

He has been on staff at McGill since 1979, engaged in clinical cardiology, clinical research in arrhythmias, pacemakers and ICD research and follow up, and continuous medical education.

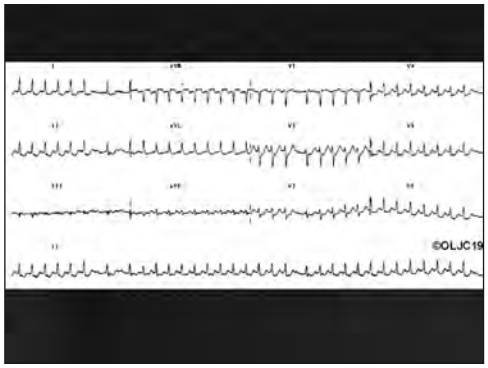
He is currently a professor of Medicine at McGill and a senior cardiologist at the MUHC. He has published more than one hundred articles in peer reviewed journals and a number of book chapters most of which related to arrhythmias and antiarrhythmic drugs.

What is new in the treatment of AF in 2009?

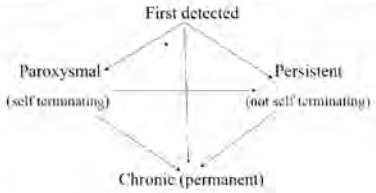
Magdi Sami. MD, FRCP(C), FACC
Prof. of Medicine, McGill univ.
Senior cardiologist MUHC

Disclosure

- I received Honoraria for lectures/ advisory boards from the following pharmaceuticals: BMS, Sanofi/ Aventis, Merck/Frost, Servier, Bohringer Ingelheim, Pfiser, Novartis, Solvay Pharma, Astra Zeneca and others…
- I am currently involved in Industry sponsored research involving BMS and Pfizer
- I participated in numerous advisory boards sponsored by all pharmaceuticals listed above.



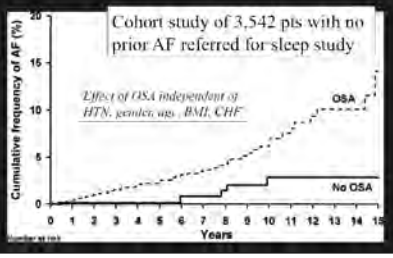
Classification of AF



What is new in etiology of AF?

Obstructive Sleep Apnea & Risk of AF

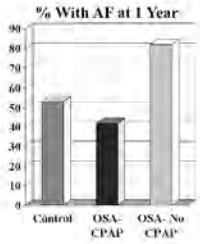
Gami, et al, JACC, 2/6/07



Obstructive Sleep Apnea & Risk of Recurrent Atrial Fibrillation After Cardioversion

Kanagala et al, Circulation, 6/03

- 39 pts with AF and OSA
  - 12 treated with CPAP
  - 27 untreated
- 79 randomly chosen control pts without OSA
- OSA pts heavier, but no other differences in clinical characteristics or drug rx



AF therapy

Anti thrombotic
and
Rate control or Rhythm control

AFFIRM STUDY

- In AF pts with high risk for stroke rythm-control was no better than rate-control in the long term composite outcome of stroke or death
- In the same study patients who were in SR did better than those in AF regardless of the strategy.

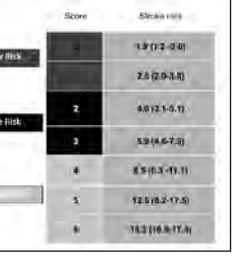
Treatment of new persistent AF should be individualized

Favors rhythm-control	*Favors rate-control*
Short duration of AF	Long duration of AF
Younger age	Older age
Symptomatic	Asymptomatic
Small LA	Large LA
Reversible cause	Irreversible cause

- WHAT IS NEW IN STROKE PREVENTION?

CHADS(2) Score

- 1 CHF
- 1 Hypertension
- 1 Age > 75 years
- 1 Diabetes Mellitus
- 2 Prior Stroke or TIA



Antithrombotic Therapy in Patients With AF: *Recommendations of ACC/AHA Practice Guidelines- JACC 8/06*

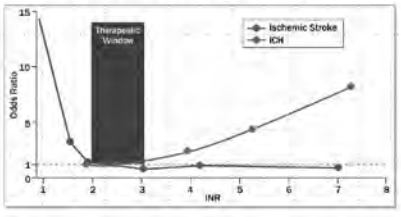
- $CHADS_2$ score 0 → ASA, 81-325 mg/day
- $CHADS_2$ score 1 → ASA or warfarin
- $CHADS_2$ score >1 → Warfarin (INR 2-3)
- Rheumatic Mitral Stenosis → Warfarin (INR 2-3)

1 point each
- *C= CHF*
- *H= Hypertension*
- *A= Age >75*
- *D= Diabetes*

S = Stroke, TIA, embolism :2 POINTS

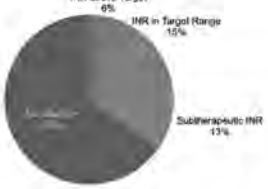
Warfarin Has a Narrow Therapeutic Window

Relationship between clinical events and INR intensity



Warfarin for Atrial Fibrillation Limitations Lead to Inadequate Treatment

Adequacy of Warfarin in Patients with atrial fibrillation in Primary Care Practice



So what are the alternatives?

ACTIVE A

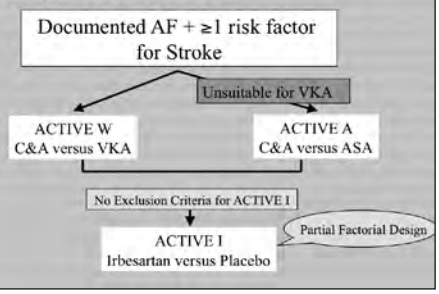
Effects of Addition of Clopidogrel to Aspirin in Patients with Atrial Fibrillation who are Unsuitable for Vitamin K Antagonists

NEJM S. Connolly et al

Hypothesis of ACTIVE A

In patients with AF, unsuitable for VKA therapy, addition of clopidogrel to aspirin will reduce the risk of major vascular events, at acceptable risk of major bleeding

Design of ACTIVE



Patient Eligibility

- Eligibility criteria for ACTIVE A and ACTIVE W were identical
  - Documented AF
  - One or more risk factors for stroke
  - Absence of major risk factor for bleeding
- Investigators selected patients for either study based on assessment of suitability for VKA

Reasons for Enrolment in ACTIVE A

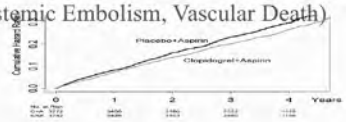
Relative risk factor for bleeding*	23%
Physician assessment that patient is inappropriate for VKA	50%
Patient Preference Only	26%

* Inability to comply with INR monitoring, predisposition to falling or head trauma, persistent BP >160/100, previous serious bleeding on VKA, severe alcohol abuse <2 years, peptic ulcer disease, thrombocytopenia, need for chronic NSAID

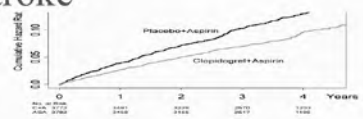
ACTIVE A Study Treatments

- All patients received aspirin at a recommended daily dose of 75-100 mgs
- Patients were randomized, double blind, to clopidogrel, 75 mg per day, or matching placebo

Primary Outcome (Stroke, MI, non-CNS Systemic Embolism, Vascular Death)

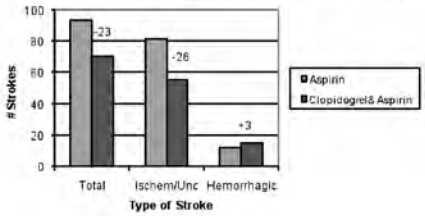


Stroke

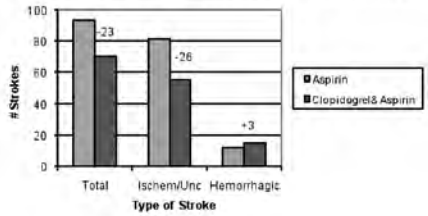


Myocardial Infarction

Numbers of Fatal Strokes Prevented



Numbers of Fatal Strokes Prevented



Benefits and Risks

1000 patients treated for 3 years

- Will prevent
  - 28 strokes (17 fatal or disabling)
  - 6 myocardial infarctions
- At a cost of 20 (non-stroke) major bleeds (3 fatal)

ACTIVE A and W: Stroke Rates and Risk Reductions

Treatment	VKA	C+A	Aspirin
ACTIVE W (Rate per year)	1.4	2.4	–
ACTIVE A (Rate per year)	–	2.4	3.3
RRR versus Aspirin	-58%	-28%	–
RRR versus C+A	-42%	–	–

Bleeding

Outcome	Clopidogrel + Aspirin		Aspirin		Clopidogrel + Aspirin versus Aspirin		
	#	rate/ year	#	rate/ year	RR	95% CI	P
Major	251	2.0	162	1.3	1.57	1.29-1.92	<0.001
Severe	190	1.5	122	1.0	1.57	1.25-1.98	<0.001
Fatal	42	0.3	27	0.2	1.56	0.96-2.53	0.07
Intra-cranial	54	0.4	29	0.2	1.87	1.19-1.94	0.006
Extra-cranial	200	1.6	134	1.1	1.51	1.21-1.88	<0.001

Case Hx

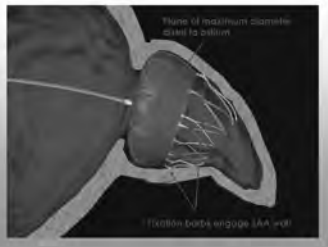
- 67 y/o man with chronic AF x 7 years
- Treated with rate-control drugs
- Hypertension, diabetes
- Probable TIA 6 years ago
- Anticoagulated with warfarin
- Hx prostate Ca, now with radiation proctitis
- Continuous rectal bleeding with INR 2-3

PROTECT AF:
US PIVOTAL Clinical Study

LAA Occlusion Device

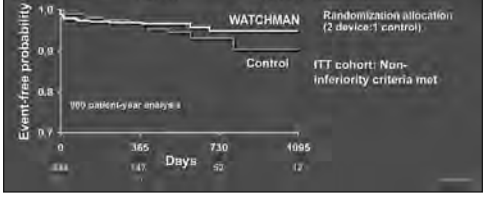
ATRITECH

WATCHMAN® Animation Still



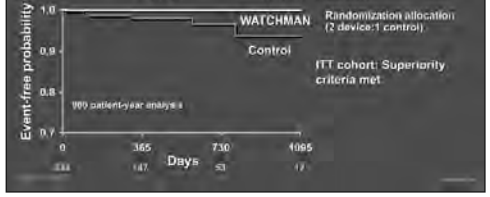
Intent-to-Treat
All Stroke

Cohort	Device Events	Device Total pt-yr	Device Rate (95% CI)	Control Events (no.)	Control Total pt-yr	Control Rate (95% CI)	RR (95% CI)	Posterior probabilities Non-inferiority	Superiority
600 pt-yr	14	409.3	3.4 (1.9, 5.5)	8	223.6	3.6 (1.5, 6.3)	0.96 (0.43, 2.57)	0.927	0.488
900 pt-yr	15	582.9	2.6 (1.5, 4.1)	11	318.1	3.5 (1.7, 5.7)	0.74 (0.36, 1.76)	0.998	0.731



Intent-to-Treat
Hemorrhagic Stroke

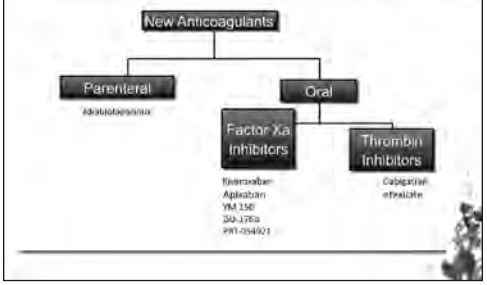
Cohort	Device Events (no.)	Device Total pt-yr	Device Rate (95% CI)	Control Events (no.)	Control Total pt-yr	Control Rate (95% CI)	RR (95% CI)	Posterior probabilities Non-inferiority	Superiority
600 pt-yr	1	416.7	0.2 (0.0, 0.9)	4	224.7	1.8 (0.5, 3.9)	0.13 (0.00, 0.80)	0.998	0.986
900 pt-yr	1	593.6	0.2 (0.0, 0.6)	6	319.4	1.9 (0.7, 3.7)	0.09 (0.00, 0.45)	>0.999	0.998



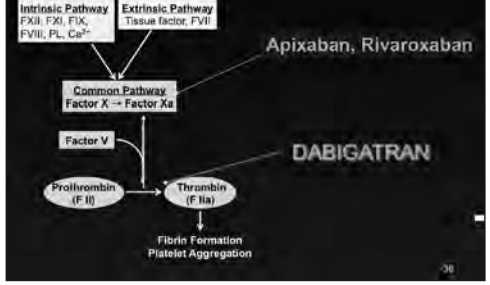
Intent-to-Treat
Primary Safety Results
Randomization allocation (2 device : 1 control)

Cohort	Device Events (no.)	Device Total pt-yr	Device Rate (95% CI)	Control Events (no.)	Control Total pt-yr	Control Rate (95% CI)	Rel. Risk (95% CI)
900 pt-yr	48	554.2	8.7 (6.4, 11.5)	13	312.0	4.2 (2.2, 6.7)	2.08 (1.18, 4.13)

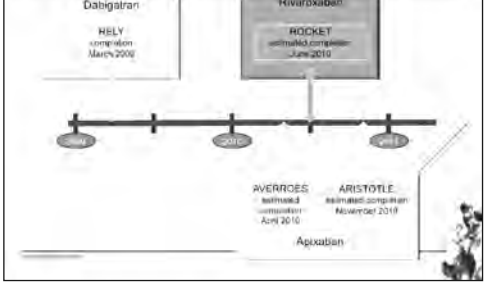
New Anticoagulant Therapy



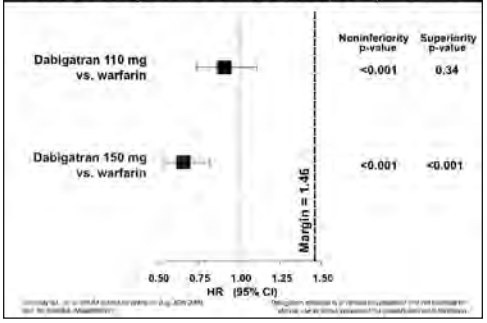
Coagulation Cascade



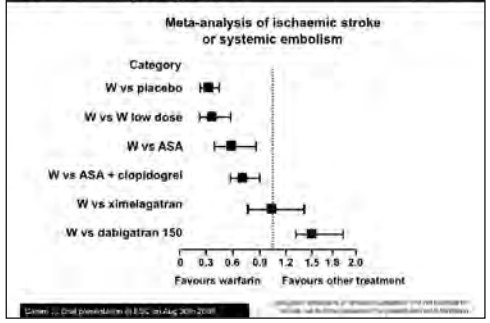
New Anticoagulants:
Atrial Fibrillation Phase III Study Timelines



Stroke or systemic embolism (SSE) RELY®



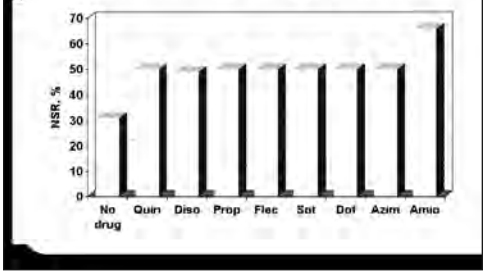
RE-LY® in perspective



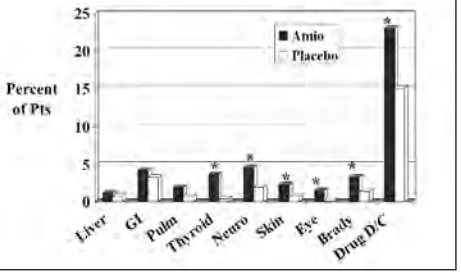
AF AA Treatment Goals

- AF is rarely life-threatening and typically recurrent
- Treatment goals
  - ↓ frequency of recurrences
  - ↓ duration of recurrences
  - ↓ severity of symptoms
  - Avoidance of adverse drug effects
- *Keep goals realistic!*

AF Efficacy:
Maintaining NSR ≥ 6 Months



Adverse Effects of Low-Dose Amiodarone:
Meta-Analysis of 1,465 Pts in Placebo-Controlled Trials
Cooperman et al. JACC 1997

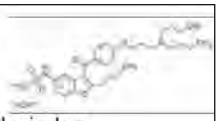


Amiodarone and Gonadal Function
Dubs, et al. JACC 1991

- 44 men with VT
  - Amiodarone- 18
  - Other AA's- 26
- Inverse correlation between total amiodarone dose and testosterone level
- Atrophic testes
  - Amiodarone: *60%*
  - Other drugs: *20%*

What's new in antiarrhythmics?

Dronedarone



- A new class III antiarrhythmic drug
- Noniodinated amiodarone analogue
- Blocks Na^+, K^+, Ca^{++}
- Also has beta-blocking activity
- Half-life 1-2 days (30-50 days for amiodarone)

ATHENA Trial: Study Design

4,628 patients ≥75 years with atrial fibrillation or 70-75 years with atrial fibrillation and at least one additional cardiovascular risk factor prior to randomization. Double blind, Randomized, Placebo controlled, International multicenter. Mean follow-up 21 months.

R

Multaq® (dronedarone) 400 mg BID

Placebo

12-30 mos. follow-up

- Primary Endpoint: composite of all-cause mortality combined with cardiovascular hospitalization
- Secondary Endpoint: death from any cause, cardiovascular death, hospitalization for cardiovascular reasons

ATHENA Trial: Primary Endpoint Results

Multaq® (dronedarone) decreased the risk of cardiovascular hospitalizations or death from any cause by 24% (p<0.001).

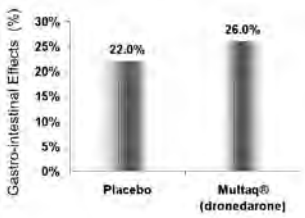
ATHENA Trial: Secondary Endpoint Results

- Compared to placebo, Multaq® (dronedarone) significantly decreased the risk of cardiovascular death by 30% (p=0.034).
- Multaq® (dronedarone) was associated with numerically fewer deaths from any cause (16%, p=0.17).
- First cardiovascular hospitalization was reduced by 25% (p=<0.001).

ATHENA Trial: Other Outcomes

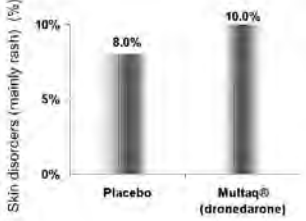
- Death from arrhythmias was reduced by 45% (p=0.01) when patients were treated with Multaq® (dronedarone).
- Multaq® (dronedarone) demonstrated a lower risk of pro-arrhythmia than placebo and no excess of hospitalizations for congestive heart failure.
- The rate of study drug discontinuation was similar between the two study arms.

ATHENA Trial: Adverse Events



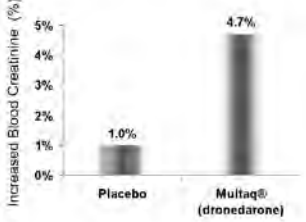
There was a higher frequency of reported gastro-intestinal complications in the Multaq® (dronedarone) group than in the placebo arm.

ATHENA Trial: Adverse Events



Multaq® (dronedarone) was associated with a more frequent occurrence of skin disorders as compared to placebo.

ATHENA Trial: Adverse Events



Patients treated with Multaq® (dronedarone) demonstrated increased serum creatinine more frequently than those given placebo.

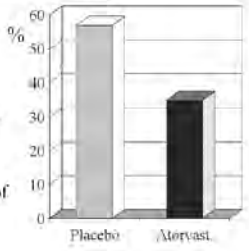
ATHENA Trial: Summary

- Multaq® (dronedarone) has been discovered as the first safe drug to benefit patients with atrial fibrillation.
- Findings include decreased rates of cardiovascular hospitalization and mortality.

Randomized Trial of Atorvastatin For Postoperative AF

Patti, et al, Circulation, 10/3/06

- Open heart surgery in 200 pts
- Randomized, double-blind:
  - Atorvastatin 40 mg/day
  - Placebo
- Started 1 week pre-op
- 61% reduction in risk of AF



Possible Mechanisms By Which Statins Reduce the Risk of Atrial Fibrillation

- Antioxidant effect
- Anti-inflammatory effect
- Effects on channel function by altering membrane lipids

Prevention of AF in 2009

- Weight loss
- CPAP for OSA
- ACE inhibitor or ARB for HBP
- Statins/Fish oil for CAD pts ar risk of AF

Notes

Monday, Nov. 23 – Workshop A-01

11:00 - 12:00 ER: MI Acute Management

Eddie Lang MDCM, CCFP (EM), CSPQ
Assistant Professor, Department of Family Medicine, McGill University;
Consulting Staff, Department of Emergency Medicine,
The Sir Mortimer B Davis-Jewish General Hospital

Research interests: Dr. Eddy Lang is an attending physician in the JGH Emergency Department. His area of expertise is evidence-based medicine, in which healthcare providers and their patients share in a decision-making process that is firmly based on the published results of medical research. Dr. Lang also leads a course on evidence-based medicine for McGill University medical students, and he teaches this subject to practicing physicians, as well lecturing internationally on the topic. In addition, he is Associate Editor of three journals related to emergency medicine and primary care. After receiving his M.D. from McGill University, Dr. Lang completed his specialty training at the Jewish General Hospital and has served since 1993 in the JGH Emergency Department and on the McGill faculty.

Notes

11:00 - 12:00 GER: Renal Failure in the Elderly

Sameena Iqbal MD

Director, Division of Nephrology, Department of Medicine,

McGill University Health Centre

Research Interests: Dr. Sameena Iqbal completed her MD degree and Internal Medicine Residency at Queens University, Kingston, Ontario. She went on to obtain her Nephrology Clinical Fellowship from McGill University in 1999. She joined the Division of Nephrology, Department of Medicine at McGill University Health Center on July 1st, 2002. In 2003, she completed a Master's in Community Health and Epidemiology.

Her research interests include: treatment of chronic kidney disease with early identification and management. She also has interest in primary care issues among chronic dialysis patients including physical activity promotion in this chronic disease population. She has ongoing projects in areas of acute kidney injury and infections in end-stage kidney disease.

Renal Failure in the Elderly

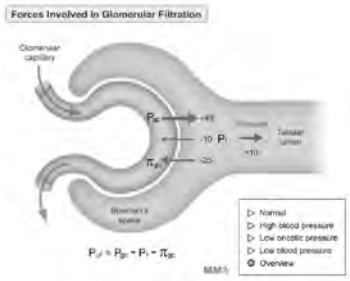
S. Iqbal
November 2009

Objectives

1. discuss the different measurements of renal function in the elderly
2. list the expected effects of aging on renal function
3. report the prevalence and significance of chronic kidney disease on the elderly population

GFR

- Glomerular filtration is the flow of fluid from the glomerular capillaries into the Bowman's capsule. The volume filtrate formed per unit time is called **glomerular filtration rate**
- The rate of glomerular filtration averages 120 ml/min in a normal adult



Methods for measuring GFR

- Serum Creatinine
- Serum Cystatin C
- Creatinine clearance
- Average of creatinine and urea clearances
- Equations for estimating GFR
- Nuclear renal scans

Renal function

- Vascular effects of aging: progressive intimal thickening or small renal arteries
- Glomerular sclerosis and progressive loss of glomeruli
- glomerular hypertrophy of the remaining glomeruli
- More prone to naturesis due to relatively low aldosterone levels and decrease in distal tubular sodium reabsorption

Physiological

- Dissociation of renal plasma flow (RPF) and GFR, more loss of cortical glomeruli, juxtamedullary glomeruli have a higher FF (GFR/RPF)
- Lower response to vasodilation induced by drugs, indicating that afferent arterioles are more vasodilated in resting conditions than the efferent arterioles

Rate of decline in GFR

- Longitudinal studies have been carried out to try to estimate 'normal' decline of GFR due to aging
- Baltimore Longitudinal Study of Aging followed 446 patients (22-97 years old) from 1958-1981
- Normal group had a decline of 0.75 ml/min/yr
- About 35% had stable renal function in the same group

Longitudinal studies

- Imai et al did a similar study in Japan, showed among 120,727 individuals over age 40, after 10 year follow up had a rate of decline of 0.36 ml/min/year
- If proteinuric, the rate of decline increased by two fold in all age groups and both genders.

Controversies

- Normal aging may mean now no decline in GFR
- The decline previously believed due to aging is in fact due to hypertension, insulin resistance, congestive failure etc.
- The elderly now are healthier than those 20 years ago

Equations to estimate GFR

Cockcroft-Gault

In men: Creatinine clearance =
(140-age) x weight in kg/(50 x serum creatinine)
In women:
[same equation]x 0.85

Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron 1976;16(1):31-41.

MDRD simplified-four variable

GFR=186 X ($Scr^{-1.154}$) x ($age^{-0.203}$) x 1.212 (if black)
x 0.742 (if female)

Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. Ann Intern Med. Mar 16;130(6):461-70, 1999.

CKD

NKF 2002 clinical practice guidelines (Kidney Disease Outcomes Quality Initiatives)

- Chronic Kidney disease definition: Kidney damage for 3 months or more defined by structural or functional abnormalities of the kidney, with or without GFR decrease
- Manifested by

a) pathological abnormalities
b) markers of kidney damage

Or

Stages of CKD

Five stages of chronic kidney disease

- proteinuria
- creatinine clearance < 90 ml/min
- creatinine clearance 60-90ml/min
- *creatinine clearance 30-60 ml/min*
- *creatinine clearance 15-30 ml/min*
- *ESRD <15ml/min*

Diagnosis of CKD

- Need a series of measurements to make a diagnosis of chronic kidney disease
- A single value may be abnormal for an acute illness
- CSN recommends for those who have an eGFR value between 30-60ml/min/1.73m^2, to repeat the test in 2-4 weeks and then repeat 3-6 months.

Prevalence of CKD in the elderly

- UK study general population 75 + from 1994-1999, n=13177

– < 30 ml/min/1.73m^2:	3.3%
– 30-44 ml/min/1.73m^2:	9.7%
– 45-59 ml/min/1.73m^2:	32%
– >60 ml/min/1.73m^2:	56.6%

NHANES 1988-1994

	<60 ml/min/1.73m²
Overall 70+	25.8%
HTN and 70+	30%
HTN and DM 70+	36%
Neither and 70+	16%

NHANES 1999-2004

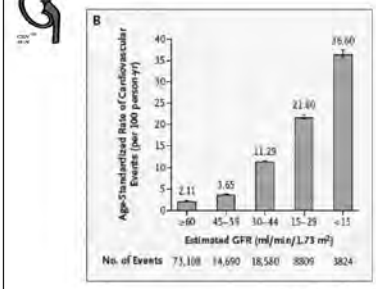
General population age 70 + n=2583

- 30-60 ml/min/1.73m²: 37.8%
- 15-29 ml/min/1.73m²: 2%

Why is CKD important to identify?

CKD is associated with

1. Cardiovascular events and mortality
2. Cancer risk
3. Cognitive function
4. Frailty



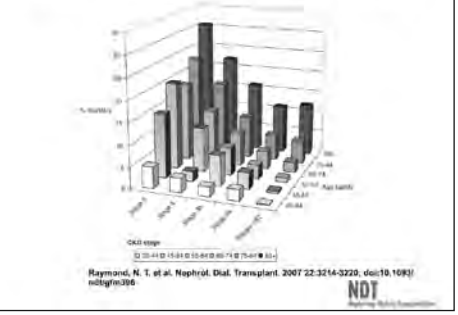
Go A et al NEJM 2004;351:1291-1305

CKD and mortality

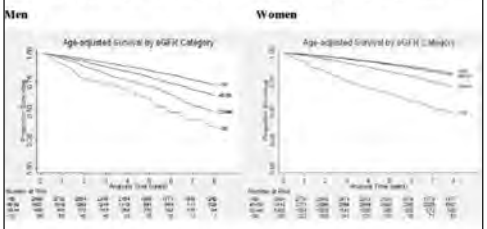
CKD	Crude 5-yr MR
Stage 2 (with prot)	19.5%
Stage 3	24.3%
Stage 4	45.7%

Keith et al

CKD in older adults and mortality



CKD and Survival in older adults



P. Roderick et al, *American Journal of Kidney Diseases*, Volume 53, Issue 6, Pages 950-960

Cancer risk

- Vajdic et al 2006, JAMA
  - Standardized incidence ratio: 1.16; 95% CI, 1.08-1.25 in ESRD patients not on dialysis
- Jorgensen et al 2008 JASN
  - Highest quintile of ACR (>1.1) had a 8.3 and 2.4 fold increase of risk for bladder and lung cancer than lowest quintile (<0.34) in general population

Cognitive impairment

Kurella et al 2005 JASN

- Cognitive impairment:

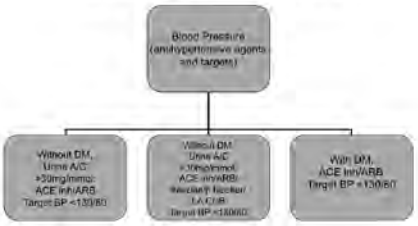
adjusted Odds ratio (OR) 1.32 (95% confidence interval [CI] 1.03 to 1.69) for eGFR 45 to 59 ml/min per 1.73 m² and

OR 2.43 (95% CI, 1.38 to 4.29) for eGFR <45 ml/min per 1.73 m²

CV Risk factor management

- Hypertension
- Glycemic control
- Dyslipidemia
- Anemia

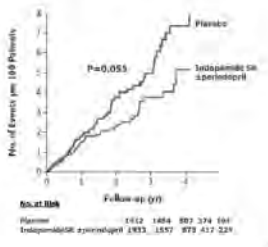
Levin et al: Guidelines for the Management of CKD. CMAJ. 2008 November 18; 179(11): 1154–1162



HTN in the very elderly (80+)

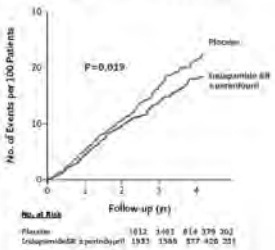
- Diuretics
- ACE inhibitors
- Long acting CCBs (if with CKD, 2nd or 3rd line)
- Target individualized
- Able to go down to 140/80 by HYVET group

All stroke (30% reduction)



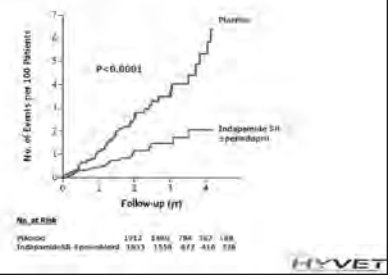
HYVET

Total Mortality (21% reduction)



HYVET

Heart Failure
(64% reduction)



Glycemic Control in type 2 diabetes

Target Hemoglobin A1c 7.0% by CKD and CDA guidelines but

2008 CDA guidelines added

"A target A1C of <6.5% may be considered in some patients with type 2 diabetes to further lower the risk of nephropathy *[Grade A Level 1A (4)]*, but this must be balanced against the risk of hypoglycemia *[Grade A Level 1A (4,5)]* and increased mortality in patients who are at significantly elevated risk of cardiovascular disease *[Grade A Level 1A (4)]*

Glycemic Control in elderly

- Individually based depend on the comorbidities/ frailty
- Tight glucose control is not as important as bp control
- No metformin eGFR < 30ml/min/1.73m^2
- Short acting agents
- Watch out for hypoglycemia

Hyperlipidemia

Pravastatin Pooling Project (sub-analyses)

Tonelli et al Circulation 2004 and 2005

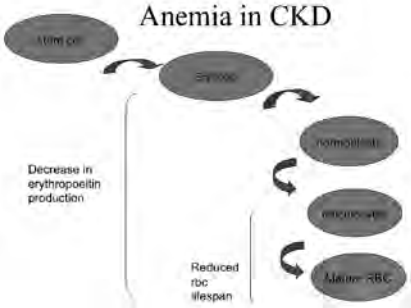
Stage 3 CKD – pravastatin lowered the risk of time to cardiac event (HR 0.77: 0.68-0.86)

Reduced loss of kidney function in the stage 3 CKD group

Recommended target

- LDL less than 2.0mmol/L as per high risk CVD population
- Use statins/ fibrates with caution

Anemia in CKD



Anemia in CKD

- Decrease in erythropoeitin production
- Other causes iron, folate, Vitamin B12 deficiencies
- Remember to rule out hypothyroidism/GI bleed
- Target Hb 110-120g/L

Lower risk for AKI

- Avoid nephrotoxins
- Radiocontrast dye, drugs to be avoided (NSAIDS) and those need to be adjusted based on eGFR
- Good perioperative care, hydration/removal of ACE inhibitor or ARB

CKD definition in elderly

- Trend towards changing CKD stages to reflect risk of progressive renal disease
- Incorporate proteinuria/combine stage 1 and 2
- Use standardized tables of eGFR values for age and gender based on their populations

11:00 - 12:00 PEDS: How to Help Children and Adolescents Deal with Divorce

Audrey Wise EdD
Counsellor, Sex and Couple Therapy Service, Royal Victoria Hospital - MUHC

Research interests: Dr. Audrey Wise obtained her Doctor of Education in Counselling Psychology from Boston University. She completed her pre-doctoral internships in clinical child psychology at the Massachusetts General Hospital of Harvard Medical School and in Behaviour Therapy at the Behaviour Therapy Unit of Boston University Medical Center.

She is a counsellor and an accredited mediator in the Province of Quebec. She was Founder and Director of the Training Programs in Family and Divorce Mediation at the Royal Victoria Hospital. She served on the Advisory Council of Family Mediation Canada. Dr. Wise was a founding member and the first Vice-President of L'Association de Médiation Familiale de Québec. At the Sex and Couple Therapy Service of the Royal Victoria Hospital her clinical interests include relationship difficulties with couples (conflict and separation) and chronic illness in relation to sexuality.

How to Help Children and Adolescents Deal with Divorce

Audrey Wise Ed.D. Counsellor
Sex and Couple Therapy Service
Royal Victoria Hospital

Introduction

- Stress/Taking Care of Yourself
- Changes in a Child's Life
- Dealing with Losses
- Families in Transition
- Restructuring of the Family
- New Beginnings

Telling the Children

- Dealing with Stress – First Take Care of Yourself
- How When What parents should tell their child/ adolescent about the divorce

Emery, Robert E. **The Truth About Children and Divorce Dealing with the Emotions So You and Your Children Can Thrive** Viking New York 2004

Neuman, M. Gary with Romanski, Patricia **Helping Your Kids Cope with Divorce The Sandcastles Way** Random House New York 1998

Talking to Children of Different Ages

- Infants and Toddlers
- Preschoolers Age Children Ages 3 to 5
- Early School Age Children Ages 6 to 8
- Late School Age Children Ages 9 to 12
- Adolescents Ages 13 to 18

Emery Robert E. **The Truth About Children and Divorce Dealing with the Emotions So You and Your Children Can Thrive** Viking New York 2004

M. Gary Neuman with Patricia Romanski **Helping Your Kids Cope with Divorce The Sandcastles Way** Random House New York 1998

How to Parent Children After the Separation

- Strategies and new ways of co-parenting
- Redefine your relationship with your ex-spouse
- The child's perspective
  - What is it like to live in 2 homes?
  - How does it feel to have a single parent?

Lowery, Vicki **Divorce Book for Parents – Helping Your Children Cope with Divorce and Its Aftermath** Book Peddlers Minnetonka Minnesota 1999
Rofes, Eric **Mom's House Dad's House Making Two Homes For Your Child: A Complete Guide for Parents who Are Separated, Divorce or Remarried** Fireside New York 1986
Rofes, Eric **Mom's House, Dad's House for Kids** Fireside New York 2000

Wallerstein's 6 Psychological Tasks for Adjustment to Parental Divorce 1983

1. Acknowledge the Marital Rupture
2. Disengage from Parental Conflict and Distress and Resuming Customary Pursuits
3. Resolution from Loss
4. Resolution of Anger and Self-Blame
5. Accepting the Permanence of the Divorce
6. Achieving Realistic Hope Regarding Relationships

Wallerstein, Judith S. Children of Divorce: The Psychological Tasks of the Child **American Journal of Orthopsychiatry** 1983, 53 230-243

Children's Adjustment to Divorce

"Divorce is a life-transforming experience…Whether the final outcome is good or bad, the whole trajectory of an individual's life is profoundly altered by the divorce experience… After the divorce, children were faced with loneliness caused by the loss of an intact family…the most powerful impact from divorce occurs in the early 20s, when man and woman relationships come center stage. That's when all the ghosts of their parents' divorce become very powerful and exercise a major influence on the young, it is here that the effects of divorce crescendo."

Wallerstein, Judith S., Lewis, Julia M., Blakeslee, Sandra **The Unexpected Legacy of Divorce The Landmark Study** Hyperion New York 2000

The Loss Inventory

Behensee,Barbara & Pequette, Jane Rich **Loss: A Manual for Educators** in **The Kids' Guide to Divorce** by John P. Borgan and Ula Maiden Fawcett Crest New York 1986

The Loss Inventory

Loss	Impact Factor
Death of a Parent	10
Death of a brother/sister	10
Divorce of Parents	10
Extended Separation of Parents (no divorce)	10

Table adapted from Brogan & Maiden 1986

The Loss Inventory

Loss	Impact Factor
Diagnosed Terminal Illness Self/Parent/Sibling	10
Death of Close Relative Moving to New City	9
Major Personal Injury or Illness (loss of limbs etc)	9
Abortion	9

Table adapted from Brogan & Maiden 1986

The Loss Inventory

Loss	Impact Factor
Rape	9
Marriage/Remarriage of a Parent	8
Love Relationship Breakup	7
Change in Physical Appearance (pimples, glasses)	4

Table adapted from Brogan & Maiden 1986

Helping the Child/Adolescent Deal with a Wide Range of Emotions and Reactions

- Confusion
- Anger
- Fear
- Depression
- Guilt
- Reconciliation Fantasies

Helping the Child Deal with Issues of Abandonment

- Fear of Losing the Other Parent – If one parent left – the other might also leave
- "My mom divorced my father and me."
- Feelings of Rejection
- How Truthful to Be with the Child
- Dependence on the Custodial Parent

Gardner Richard A. **The Boys and Girls Book About Divorce** Bantam Books New York 1992
Gardner Richard A **Psychotherapy with Children of Divorce** Jason Aronson Inc. Northvale New Jersey
Neuman, M. Gary with Romanowski, Patricia **Helping Your Kids Cope with Divorce The Sandcastles Way** Random House New York 1998

How to Decide on Counselling for Your Child

- For What Reasons?
- When is a Referral Appropriate?
- Dealing with Persistent Problems

Gardner R.A. **The Boys and Girls Book About Divorce** Bantam Books New York 1992

Neuman, M.G. with Romanowski P. **Helping Your Kids Cope with Divorce The Sandcastles Way** Random House New York 1998

The Different Paths

- Mediation vs. Litigation
- The Child/Adolescent in High Conflict Families
- The Concept of Parental Alienation Syndrome

Fisher, Roger, Ury, William Patton, Bruce **Getting to Yes: Negotiating Agreement Without Giving In** Penguin Books New York 1991
Gardner Richard A., **Child Custody Litigation: A Guide for Parents and Mental Health Professionals** Creative Therapeutics Cresskill New Jersey 1986
Hodges William F. **Interventions for Children of Divorce Custody Access and Psychotherapy** John Wiley & Sons New York 1991
Irving, Howard H., Benjamin, Michael **Family Mediation: Contemporary Issues** Sage Publications Thousand Oaks California 1995

How to Be a Long Distance Parent

- Issues of Relocation
- How to Remain Involved in a Child's Life
- Challenges in Long Distance Interaction
  - Unable to Share Thoughts As They Occur
  - Fewer Opportunities to Share Rituals
- Telephone Calls
- Use of Technology, E-mails
- Mail, Stamps, Games, Gifts
- Reading Books Together, Going to a Movie/Watching TV Together – (In a Different City)
- Sending Work and School Samples

Newman, George **101 Ways to Be a Long-Distance Super-Dad** Blossom Valley Press Tucson Arizona 2000

Rice, Joshua **Mom's House Dad's House Making Two Homes For Your Child: A Complete Guide for Parents who Are Separated, Divorce or Remarried** Fireside New York 2006

Rice, Joshua **Mom's House, Dad's House for Kids** Fireside New York 2006

Yarosh, Svetlana Supporting long-distance parent-child interaction in divorced families **Conference on Human Factors in Computing Systems** CHI Proceedings 2008 Florence Italy

Understanding the Role of Grandparents

- Role as "Firefighters"
- Friction Between Parent and Grandparent
- Grandparents Dealing with Former In-Law
- Advantages of Grandparents as a Support System
- Difficulties of Grandparents as Primary Caretakers

Clingempeel, W.G., Colyar, J.J., Brand, E., and Hetherington, E.M. Children's Relationships with Maternal Grandparents: A longitudinal Study of Family Structure and Pubertal Status Effects **Child Development** 63: 1404-1422

Hanson, T.L., McLanahan, S.S., Thomson, E., Windows on Divorce: Before and After **Social Science Research 27: 329-349**

Wallerstein, Judith S., Blakeslee Sandra B., **What About the Kids? Raising Your Children Before, During and After the Divorce** Hyperion New York 2003

How Parents Can Help Their Children Deal with Others

Maintain Relationships with:

- Siblings
- Teachers
- Neighbours
- Coaches

Kaplan, L., Hennon, C.B. & Ade-Ridder, L. Splitting Custody of Children Between Parents: Impact on the Sibling System. **Families in Society: The Journal Contemporary Human Services** 1993 131-144

Wallerstein, Judith S., Blakeslee Sandra B. **What About the Kids? Raising Your Children Before, During and After the Divorce** Hyperion New York 2003

Conclusions

- Change Your Expections
- Take Care of Yourself
- Focus on the Emotional Needs of Your Children and Adolescents
- Divorce is a Process
- Take Each Day in Stride –The Many Surprises
- The High Cost of Divorce For Children and Adolescents
- Moving on

References-Articles

Afifi, T.D. Huber, F.N. (8b), F. Parents' and Adolescents' Communication with Each Other About Divorce-Related Stressors and Its Impact on Their Ability to Cope Positively with the Divorce **Journal of Divorce & Remarriage** 45, 1&2, 1-30 May 2006

Amato, P.R. Children of Divorce in the 1990's: An Update of the Amato and Keith (Meta-Analysis) **Journal of Family Psychology** 15 3, 355-370 1991

Amato, P. Children's Adjustment to Divorce: Theories, Hypotheses and Empirical Support **Journal of Marriage and the Family** 55, 23-28. 1993

Amato, P.R. and Keith, B. Parental Divorce and the Well-Being of Children: A Meta-Analysis **Psychological Bulletin** 110, 1,26-46, 1991

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Drane, J., Hedrick, M. The Parental Alienation Syndrome: An Analysis of Sixteen Selected Cases **Journal of Divorce and Remarriage** 21 ½ 21-23 1994

Kaplan, L. Hennon, C.B. Ade-Ridder, L. Splitting Custody of Children Between Parents: Impact on the Sibling System **Families in Society: The Journal of Contemporary** Human Services 131-144 1993

Kyle, J.H. Current Research on Children's Preferences Adjustment No Simple Answers **Family and Conciliation Courts Review** 31 1 29-49 January 1993

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Snyder, J. Loss: Divorce, Separation and Bereavement in Klykylo, W.M. and Kay, J. (Editors) **Clinical Child Psychiatry** 307-320 2nd Edition John Wiley & Sons New York 2005

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Folberg, J. Milne, A.L. Salem, P. **Divorce and Family Mediation: Models, Techniques and Applications** Guilford Press New York 2004

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Kaslow, F.W. Schwartz, L.L. **The Dynamics of Divorce: A Life Cycle Perspective** Brunner/Mazel New York 1987

O'Rourke, K. Worthyl, J.C. **Support Groups For Children** Accelerated Development Philadelphia 1990

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Wolfinger, J.S. Blakeslee, S. **Second Chances Men, Women and Children A Decade After Divorce** Houghton Mifflin Company Boston 1996

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Wallerstein, J.S. Lewis, J.M. Blakeslee, S. **The Unexpected Legacy of Divorce: A 25 Year Landmark Study** Hyperion New York 2000

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Ackerman, M.J. **Does Wednesday's Mean Mom's House or Dad's? Parenting Together While Living Apart** John Wiley & Sons New York 1996

Abrams, C. **The Good Divorce: Keeping Your Family Together When Your Marriage Comes Apart** Harper Collins New York 1988

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Kornhaber, A. **The Grandparent Solution How Parents Can Build A Family Team for Practical Emotional and Financial Success** Jossey-Bass San Francisco California 1992

Kline, L. Rev. S. **For the Sake of the Children How to Share Your Children How to Share Your Children with Your Ex-Spouse in Spite of Your Anger** Prima Publishing Rocklin California 1992

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Mayle, P. **Why Are We Getting A Divorce?** Harmony Books, New York 1988

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Wolf, A.E. **Why Did You Have To Get A Divorce? And When Can I Get A Hamster? A Guide to Parenting Through Divorce** The Seven Day Press New York 1998

Resource-Divorce

Children's Rights Council

http://www.crckids.org/

Questions?

Notes

Monday, Nov. 23 – Workshop A-04

11:00 - 12:00 Research in Your Office

Gillian Bartlett-Esquilant PhD
Associate Professor, Department of Family Medicine, McGill University

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Research Interests: Dr. Gillian Bartlett is an Associate Professor in the Department of Family Medicine. She received her Ph.D. in epidemiology from McGill University in 2001 and her M.Sc. in 1996. Dr. Bartlett specializes in patient safety in primary care. Her research involves health informatics, pharmacoepidemiology, population health and evaluation methodologies for complex data sets in primary care. From her research interests, she has also developed a research program that deals with privacy issues related to health informatics. Through her PhD work and on-going research, she has extensive experience with the formation and analyses of large administrative database records in pharmacoepidemiology. Her current focus is on patient safety and pharmacogenomics in family medicine. Dr. Bartlett is supported by a FRSQ Chercheur-Boursier career award.

"If you want more evidence-based practice you need more practice-based evidence" -Lawrence W. Green, 2008

Learning Objectives:

1. To understand basic research methods.
2. To be able to identify the steps necessary to complete a research project proposal.
3. To understand how clinical research relates to critical appraisal and the practice of evidence based medicine.

Suggested Reading/References:

Dawes M et al. Evidence-based practice: A primer for health care professionals. Second Ed. Elsevier 2005.

Kramer MS. Clinical Epidemiology and Biostatistics: A primer for clinical investigators and decision makers. Springer-Verlag New York, 1991.

Last JM. A Dictionary of Epidemiology. Fourth Edition, Oxford University Press 2000.

Helpful Websites:

Basic Stats: http://www.sjsu.edu/faculty/gerstman/EpiInfo/basics.htm

Basic Sample Size: http://ravenanalytics.com/Articles/Sample_Size_Calculations.htm

McGill University
Department of Family Medicine

Research in Your Office

Gillian Bartlett, PhD

Monday, November 23, 2009

Lecture Outline

- Why research?
- Choosing your study population
- Clinically meaningful change
- Effect size and it's importance
- Choosing your methods: help for quantitative analysis
- Types of statistical tests – simple to complicated and what that means
- Implications for your ideas/practice
- Linking to critical appraisal and evidence based medicine or why research will help you be a better clinician

Why Research in Family Medicine?

"Self-image of family physicians has not included research as a normative descriptor" -Herbert, 2004

"If you want more evidence-based practice you need more practice-based evidence" -Lawrence W. Green, 2008

Research 101

Overview of the Basics

Starting a Project
COMMON QUESTIONS

1. How many participants do I need?
2. What do I put in the data analysis section?
3. How do I analyze my data?

MORE QUESTIONS TO GET ANSWERS

1. What is your question?
2. What is your outcome?
3. How is it measured? In what group?
4. How big an effect do you want to see?
5. Is the effect meaningful?

What is your question?

Question

↓

Background

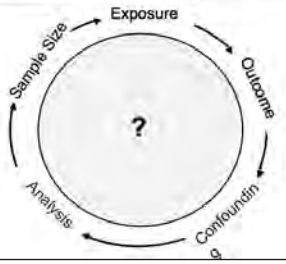
↓

Population

↓

Reasonable Question

In what group? Study Population



How big an effect do you want to see?
How BIG is BIG?

Effect size: ratio of change to variability

0.2 - 0.3 – **small**

0.5 – **moderate**

0.8 - **large**

Effect greater than "noise"

signal is difficult to detect against excessive background noise

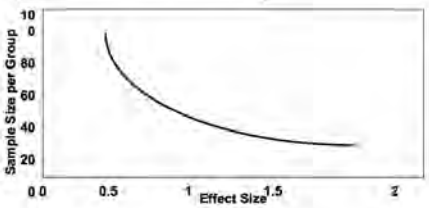
SAMPLE SIZE DEMYSTIFIED

Sample Size Formula = SD / delta

Effect size = delta / SD

Delta = clinically meaningful difference

Relationship between Effect Size and Sample Size



(Two group design)

How meaningful is it?
Clinically Meaningful Change

Meaningful to whom?

- Clinician - usually impairments
- Patient - disability, handicap, quality of life
- Society - quality of life, health services utilization, cost
- Payer – disability, prescription medication

What statistics should I use?
Review of 5 Essential Questions

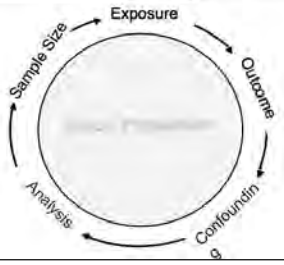
1. What question are you trying to answer?
2. What very specific question are you most interested in?
3. What form will your answer take?
   - Yes or No? Number ? Rank? Rate?
4. Do you need to worry about other things that might influence your answer?
5. Will your answer change what you do or how a clinician will practice? How much convincing do you need?

What statistics should I use?
5 Essential Answers

1. question
2. factor of interest (exposure) & outcome
3. type & complexity of statistics
4. source of bias - confounders
5. amount of power – number of people

↓

FEASIBLE?



Statistical Tests

1. Univariate (descriptives):
   - percents, counts
   - means, proportions
   - distribution (standard errors, ranges, etc)
2. Bi-variate (unadjusted inference):
   - T-tests
   - Chi-square
   - Simple regression (Crude odds ratios, relative risks, etc)
3. Multivariate (adjusted inference):
   - Regression adjusting for potential bias
   - GEE (repeated measures)
   - Survival analysis adjusting for potential bias

Regression 101 (prediction and control bias)

Linear (number)
- How much of an increase do you get in the outcome for every unit increase in the factor of interest?

Logistic (yes/no)
- How much more likely are you to have the outcome if you have the factor of interest?

Poisson (rates)
- How much will the proportion of the outcome change for every unit increase in the factor of interest?

Cox (time to event)
- How much sooner will the outcome happen if you have the factor of interest or increase the factor of interest?

Meaning for You

- Answering these questions will answer:
  - Is this appropriate in scope, feasibility and time line?
  - Is there an area of training you currently lack?
  - Details for objective, hypothesis and methods – imposes structure

Helpful Websites

- Basic Stats:
  [illegible]
- Basic Sample Size:
  [illegible]

Steps for Research Project

- Use the material presented to form a working "protocol" – start with one page then expand!
- Get feedback from peers, experts, participants…
- Obtain funding if necessary
- If funded – obtain ethics approval
- Follow your protocol!
- Fill in the blanks for your article – publish!
- Attend a fun conference to commiserate with your peers.

Steps for Research Project

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Why Evidence Based Medicine?

- An average year in practice for a family doctor :
  - 2,500 diagnoses
  - covering 450 conditions
  - prescribing 833 different drugs as part of a total of 20,000 prescriptions
- 70–80% of all scripts in Canada are written by primary care physicians

What is Evidence Based Medicine?

"the conscientious, explicit and judicious use of best current evidence in making decisions about the care of individual patients" -David Sackett,1996

"Evidence based medicine requires the integration of the best research evidence with our clinical expertise and our patient's unique values and circumstances." - Sharon E. Straus: Evidence Based Medicine 3rd Edition

Practicing EBM and Critical Appraisal

- **Ask** – identify your problem
- **Acquire** – define a structured question
- **Appraise** – select the best of the relevant studies and apply rules of evidence to determine their validity
- **Apply** – how should you apply the results to patient care?
- **Assess** – determine if the action was helpful
- **Adjust** – use the outcomes of intervention to modify the treatment

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Notes

11:00 - 12:00 BP Assessment in the Office

Brian Gore MD
Maimonides Geriatric Centre, McGill University

Research Interests: Dr. Gore is a family physician with special interests in geriatric medicine, geriatric research and hypertension.

He received his medical training at in France, with postgraduate training at Memorial and McGill Universities. He also completed postgraduate training in Epidemiology at McGill.

He currently holds positions in the Dept of Family Medicine at McGill, the MUHC in Primary Care and Maimonides Geriatric Centre where he is also Director of Professional Services.

He has been an investigator for over thirty national or international clinical trials, served on 13 national scientific committees including CHEP, published numerous geriatric related research and health care related articles and presented over 40 conferences to GPs and specialists both in Canada and abroad on hypertension and ambulatory blood pressure monitoring technologies.

60th Annual FP Refresher Course

BP Assessment in Your Office

Monday, November 23rd, 2009

Workshop A Series

11:00

Brian Gore, MD*

* Private Practice: Family Medicine Group, Westmount
Maimonides Geriatric Centre: Director of Professional Services
MUHC Affiliate Staff
McGill University Dept of Family Medicine

brian.gore@videotron.ca

Disclosures

- **Professional**
  - Former member CHS, CHEP
  - Member QGS
- **Consultant:**
  - on Advisory Boards for various clinical trials (honorariums)
  - medical device companies
- **Speaker:**
  - on expert panels for pharma companies (honorariums)
  - McGill CME lectures/workshops
- **Research:**
  - Clinical trials in hypertension
- **Educational:**
  - Honorariums for courses and lectures in BP assessment and monitoring technologies since 1995 for Aventis, Merck, Novartis, BMS, Pfizer, Servier, Boehringer, PG, etc.
- **Commercial:**
  - President of Cardiotronics Inc
  - Canadian agent and consultant for dabl of Ireland

Learning Objectives:

- **Using CHEP guidelines in your practice: (self-learning program)**
- **Evaluating your office BP routine**
- **Implementing changes in your practice:**
  - **BP measurement accuracy = better CVD management**
- **Recommending devices: office, home, ABPM**
- **Understanding ABPM patterns and clinical cases**
- **the future for ABPM evaluations: web-based dabl services**

The Canadian Hypertension Education Program (CHEP) is sponsored by:

Canadian Hypertension Society
Blood Pressure Canada
Public Health Agency of Canada
Heart and Stroke Foundation of Canada
College of Family Physicians of Canada.

CHEP: 2009 Recommendations

A slide kit for medical education can be downloaded from the Canadian Hypertension Society website at:

http://www.hypertension.ca

CHEP highlights for 2009

- **All agents are effective as 1st line in uncomplicated HTN. Keep B-Blockers < 60 years.**
- **Re-evaluate at least q 2/12 until controlled.**
- **Monotherapy controls <30%; 66% need 2 agents and half of these need 3 or more (average = 3.2).**
- **Lower doses of combined treatments are often more effective with fewer adverse effects than higher doses of fewer agents.**

CHEP highlights for 2009

- **Combining ACE and ARB +/- B-B: poor efficacy evidence in HTN without concomitant disease.**
- **Be aware of white coat (20%), masked hypertension (10%) and poor adherence (30-50%).**
- **Salt is the villain (<2300 mg daily)**
- **High normal BP should be followed at least annually: 40%→2 yrs, 60% →4 years**

Benefits of Treatment

NNT over 10 years

- **to prevent 1 patient from developing any complication from HTN: 6**
- **to prevent 1 diabetes-related death: 15**
- [illegible]

Your office BP routine: do you:

- know and use CHEP guidelines
- rely on OBP primarily for clinical decision
- use multiple readings in office
- recommend SBPM
- know which SBPM models to recommend
- recommend ABPM
- how to interpret ABPM

Clinic, Home/Self, Ambulatory (ABP) Blood Pressure Measurement equivalence numbers

A clinic blood pressure of 140/90 mmHg has the equivalent risk of a:

Description	Blood Pressure mmHg
Home/Self pressure average	135 / 85
Daytime average ABP	135 / 85
24-hour average ABP	130 / 80

Your office BP routine

- Rely on OBP primarily for clinical diagnostic and treatment decision

Office BP procedures

- Mercury ♡
- Electronic:single ♡
- Electronic: multiple ♡

Professional Digital OBP

BpTru

Approved Digital OBP Devices

Professional Digital OBP

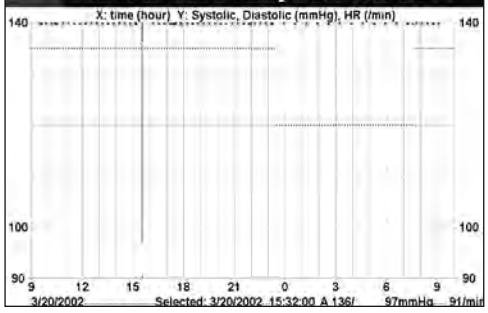
Omron HEM-907XL

Your office BP routine

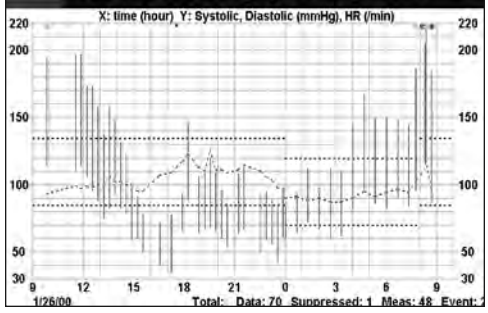
- Use multiple readings in office:
  - where, position, arms
  - How many readings
  - How frequently

Blood Pressure Assessment:
Patient position

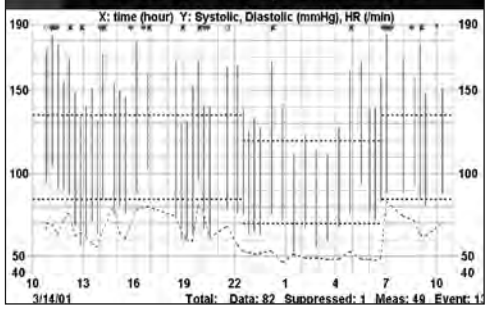
Casual OBP: representative?



Which BP is representative?



Which BP is representative?



Inherent Variability of BP

Implications

Inaccurate Diagnosis

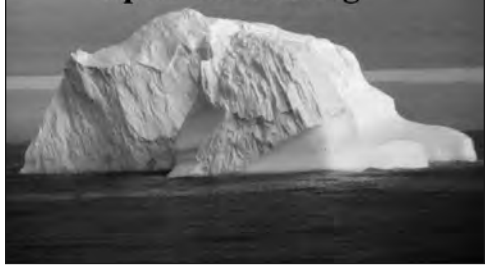
Inappropriate Treatment

Reflective moment .....

- Daily average no. of heartbeats: 103,680
- No of beats per OBPM: <15
- Sampling ratio is 1:6912 or .0145%
- Clinical decision in 3 to 5 office visits
- Include the BP variability
- Include the other common errors in BP measurement
- Include the concept of WCE/WCH

What is your level of confidence that you have representative BP measurements upon which you can make a clinical and therapeutic decision??

Office BP values = tip of the iceberg



Home (Self) Measurement of BP:
Patient Education
How to?

Use devices:
- appropriate for the individual (cuff size)
- have met the standards of the AAMI
- and or the BHS and or IP

Adequate patient training in:
- measuring their BP
- interpreting these readings

Regular verifications
- accuracy of the device
- measuring techniques

Values over **135 / 85 mm Hg** should be considered elevated

Self measurement can help to improve patient adherence

AAMI=Association for the Advancement of Medical Instrumentation
WHO-ISH=Hypertension Society, International Protocol

Home Blood Pressure Readings

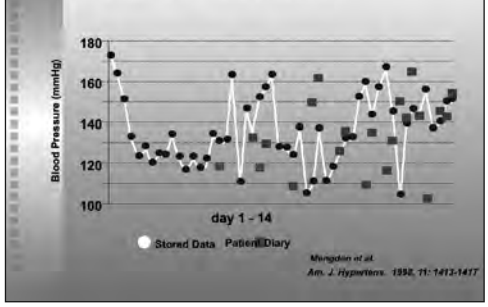
SELF BP "REPORTING"

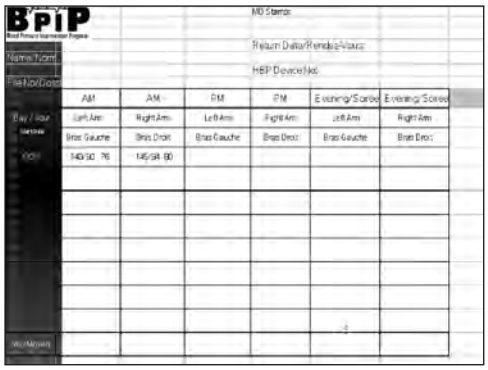
Suggested Protocol for Home (Self) Measurement of Blood Pressure

Home blood pressure values should be based on:
- duplicate measures,
- morning and evening,
- for an initial 7-day period.

Singular and first day home BP values should not be considered.

SELF-BP "REPORTING"





Recommended Self Monitoring Devices

Recommended SBPM

LifeSource 787EJ

Omron HEM-711DLX

A & D Model

Omron HEM-790IT w Cuff

Recommended SBPM

LifeSource-787W

Omron HEM-775 Model

BP 3BTO-A

Omron HEM-773 W ComFit Cuff

Clinical Indications for ABPM

Clinical Indications for ABPM

T Pickering, Am J Hypertens, 1996, O'Brien, Prague ISH, June 2002

- Suspected WCH or WCE w/o target organ damage
- Evaluation of treatment resistant HTN
- Hypotension symptoms on antihypertensive medication

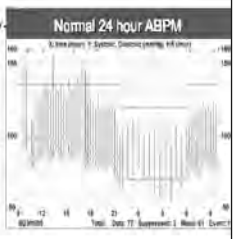
Clinical Indications (cont)

T Pickering, Am.J Hypertens. 1996, O'Brien, Prague ISH, June 2002

- Intermittent symptoms possibly related to blood pressure (postural, postprandial)
- Nocturnal hypertension (sleep apnea, diabetics)
- Autonomic failure: diabetics

What to assess in an ABPM

- ABPM readings: quality, #, pattern.
- Periods: total 24 hour, awake, asleep.
- Dipper status: Y,N, Excessive, Reverse
- 24-hour pulse pressure.
- White coat HTN or effect.
- Heart rate and rate-pressure product.



Summary Guide to Interpret ABPM

Analyzing the data:

Minimum number acceptable:

14 readings day-time

7 readings night-time

What are normal ABPM limits

Are office BP readings comparable to ABPM values ?

Recommended standards for normal and abnormal pressures during ABPM.

These pressures are only a guide, and lower pressures may be abnormal in patients whose total risk factor profile is high and in whom there is concomitant disease.

	Normal	Abnormal
Day	135/85	>140/90
Night	120/70	>125/75
24 hour	130/80	>135/85

Summary Guide to Interpret ABPM Results

Analyzing the data 1:

ABPM profiles:

- normal day and night periods
- white coat syndrome (includes WCH + WCE)
- borderline hypertension
- nocturnal hypertension

Summary Guide to Interpret ABPM Results

Analyzing the data 2:

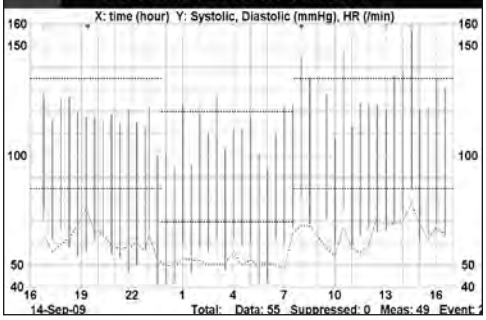
ABPM profiles:

- systolic and diastolic hypertension + dipper
- systolic and diastolic hypertension + non-dipper
- isolated systolic hypertension
- isolated diastolic hypertension
- excessive BP variability

Clinical ABPM Cases and ABPM patterns

ABPM recordings and cases of Brian Gore, MD

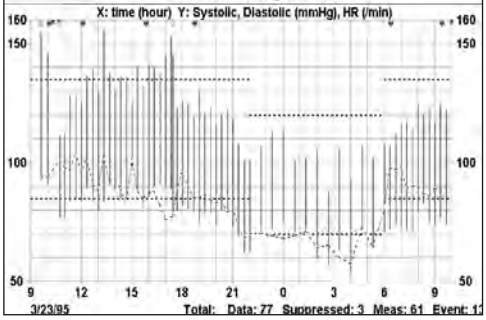
Normal 24 hour ABPM



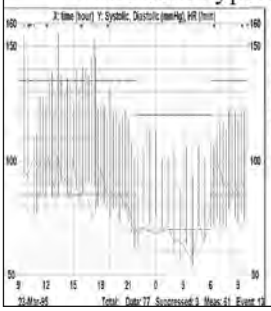
Case: Suzanne B is a 79 year-old female referred for elevated OBP and variable PBP

Past History: OA, OP Elevated OBP: 165/100 PBP: 130/85 to 150/95	**Family hx**: OP, HTA, AD **CV risks:** Age > 60, PM. **Physical exam:** Kyphosis, OA Wt: 105, Ht: 59"
Investigations: Routine lab normal. **Meds**: Risedronate, Vit D, Ca, Acetaminophen, ibuprofen prn **Referred to** confirm diagnosis.	**Pre-ABPM electronic BP:** SeRBP: 156/94 Sel.BP: 164/94

Your diagnosis



White coat hypertension



- 24 hour average: 119/76
- Awake period: 127/81
- Asleep period: 103/67
- 24 hour PP: 43 mmHg
- Dipper status: SBP: 19% DBP:18%

Prevalence of White Coat Hypertension

Ranges from 10-30% of hypertensive population based on review of clinical trials

Implications of WCE

- **Overestimation of OBP**
- **Potential for overtreatment**
- **Nonresponse to Rx**
- **Potential Rx adverse effects**

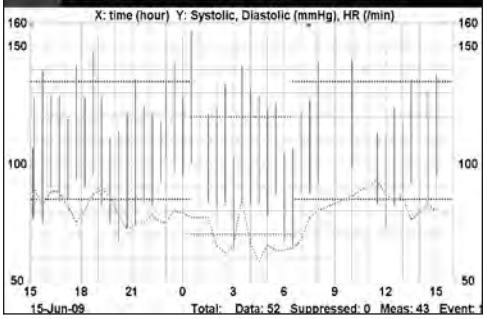
Lessons from this illustration:

- **Be wary of the older female patient with office SHTN.**
- **Encourage HBPM if at all possible.**
- **ABPM can avoid unnecessary Rx and potential ADRs.**

Case: Henry G is a 54 year-old male referred for Rx resistant HTN

Past History: GBD, Remote sports injuries, BPH Elevated OBP: 165/100 PHP: 130/85 to 150/95	**Family hx:** , HTN, CAD, Obesity **CV risks:** Male, + lipids, pre DM **Physical exam:** + abd girth 125 cm Wt: 120 kg, Ht: 185 cm
Investigations: + lipids, - FBS: 6.8. Meds: **Diovan 320, Norvasc 10, HCTZ 25, all in AM. Flomax, Avodart, Lipitor.**	**Pre-ABPM electronic BP: 125/80(R); 121/78(L)-average of 3 measurements.**

Clinical Case: Henry G, Age 54, + abd girth



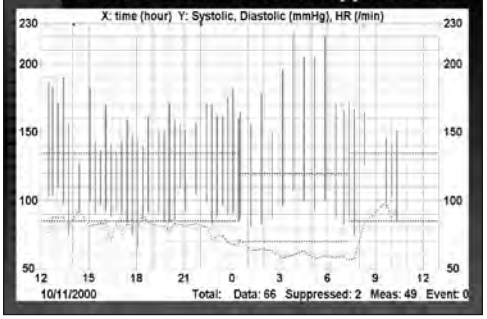
ABPM Results:

- 24 hour average: 128/85 (43)
- Awake period: 129/86 (32)
- Asleep period: 128/82 (11)
- 24 hour PP: 44 mmHg
- Dipper status: SBP:-1% DBP:1%

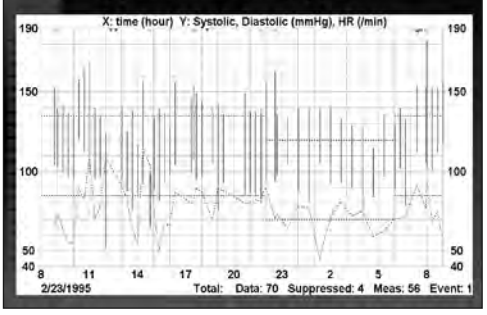
Interpretation

- **Verify activities/sleep quality during ABPM session:**
- **usually very poor sleep less active day than usual.**
- **Dipper index: reverse dipper-STP/ non-dipper-DBP**
- **With abnormal range asleep BPs and reverse dipper for SBP on triple therapy sleep apnea is suspected.**
- **Recommendations: A sleep study is recommended**

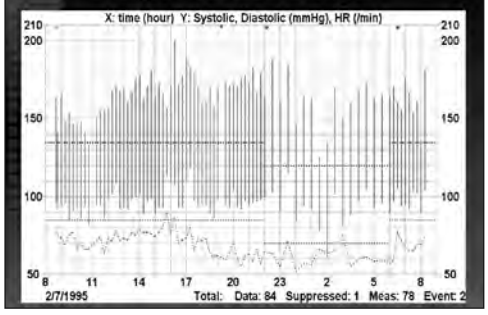
S+D HTN with Nocturnal Hypertension



Hypertensive Non-Dipper



Stage 3 HTN Non-Dipper

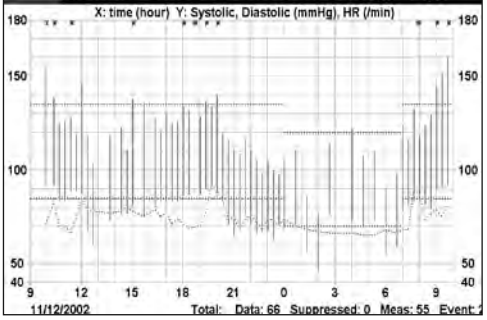


Dippers and Non-Dippers

- Dipper: Day/Night >10/5 mmHg
- Non-Dipper: Day/Night <10/5 mmHg

- Dipper: Stroke 3%
- Non-Dipper: Stroke 23%

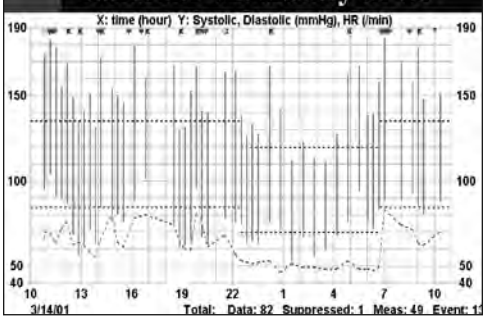
Normal 24 hr ABP with morning surge



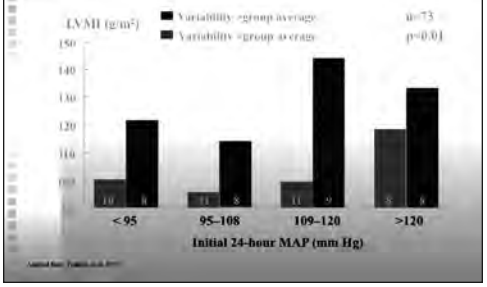
CV Events that are Coincident with Morning Blood Pressure 'Surge'

- **Myocardial ischemia**
- **Myocardial infarction**
- **Sudden cardiac death**
- **Stroke**
  - **Thrombotic**
  - **Hemorrhagic**

Inherent Variability of BP



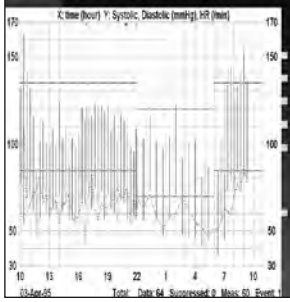
Blood Pressure Variability and Target Organ Damage: A Longitudinal Analysis



Case: Margie S is a 78 year-old female referred for ABPM to evaluate periods of dizziness on Rx

Past History: COPD, OP, MKD, MCI-5 years. Elevated OBP: 165/90 PHP: 110/85 to 140/90	**Family hx:** , Nil **CV risks:** age, PM, VD **Physical exam:** fibin, chest findings Wt: 48 kg, Ht: 140 cm
Investigations: eGFR: 55, K-3.3, Na: 132, Cl: 90, EKG-SVEs CT brain: lacunar infarcts. Meds: **Diovan 160, Adalat XL 20, HCTZ 25, Exelon, Fosamax, Vit D, Ca, ASA 80, Spiriva, Ventolin prn**	**Pre-ABPM electronic BP: 145/90(R); 150/86(L)-average of 3 measurements.**

Overtreatment

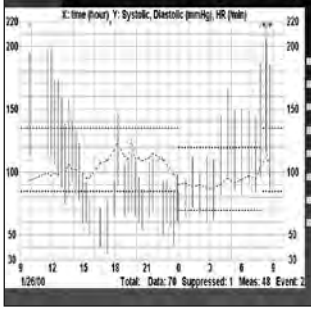


- 24 hour average: 110/60
- Awake period: 115/64
- Asleep period: 102/54
- 24 hour PP: 50 mmHg
- Dipper status: SBP: 12% DBP:16%
- 04:40 - 81/41mmHg

Case: Trudy D is a 65 year-old female

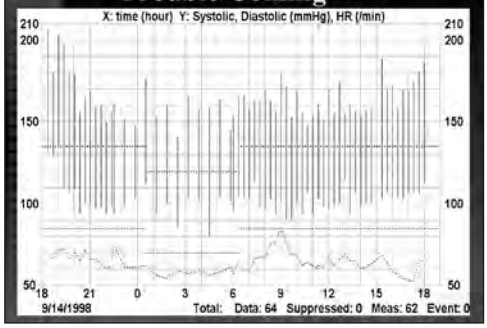
Past History: **TOD/CCVD: CAD, LVH, CABG, CVA, Remote MI. CO-MORBIDITY: DIABETES. RF: AGE, PM, SMOKER, HYPERLIPIDEMIA OBP: 170-180/90**	**Physical exam:** Carotid bruits. Reduced PP's, trophic leg changes, Mild weakness RA. BMI: 29. **Meds: LOPRESSOR 100 MG BID, COZAAR 100 MG QAM,** Glucophage 500 tid, Lipitor 20 qhs, ASA 80 mg QD.
Lab Investigations: TC-6.52, HDL-1.05, LDL-5.1 TG: 3.2, CV Risk Ratio:6.21 Proteinuria >3gm/d, HgB A1C: 0.078. EKG: LVH, Remote inferior MI.	**Referred to evaluate 24 hour control in view of persistantly high OBP** The Dilemma: BP management in light of ABPM results.

Autonomic Dysfunction

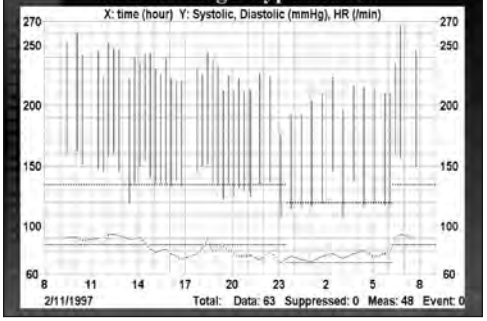


- 24 hour average: 126/74
- Awake period: 121/71
- Asleep period: 129/76
- 24 hour PP: 52 mmHg
- Dipper status:
- SBP:-7% DBP:-8%
- 08:22 216/118
- 16:32 72/42

Trouble Coming



Stroke Range Hypertension



What are principal advantages of ABPM versus OBPM and SBPM

- Revealing:
  - white coat hypertension (20%)
  - masked hypertension (10%)
  - nocturnal hypertension (CV event predictor)
  - 24 hour blood pressure patterns

The Future of ABPM:

standardizing and automating ABPM reporting "dabl"

Dabl slides graciously supplied for this workshop by Professor Eoin O'Brien, University College, Dublin, Ireland
www.dableducational.org

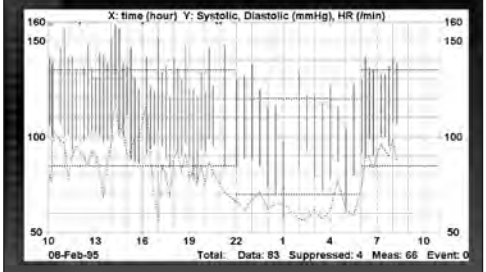
Dabl Disease Management Systems

- **A new concept in ambulatory blood pressure interpretation**
- **On-line instant internationally recognized reporting**
- **Use with any validated ABPM device**
- **No more errors or questions in interpretation**

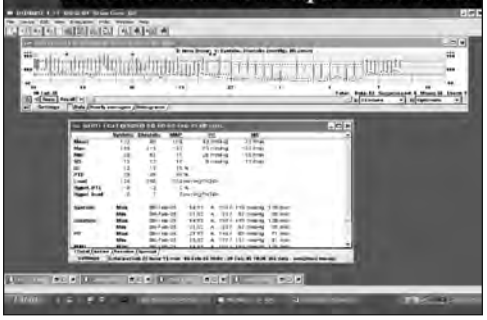
Traditional ABPM Report

- Graphic and Numeric Data Display
- Statistics
- No Interpretation

Traditional ABPM Report: Graph



Traditional ABPM Report: Statistics

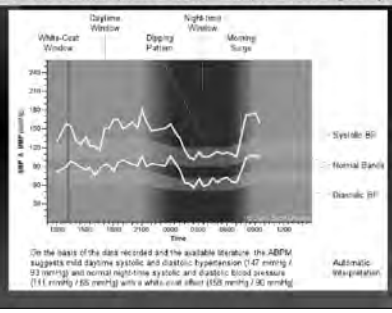


Dabl Sample Reports

ABPM Report

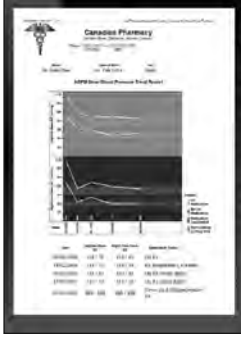
- Personalised Letterhead
- Patient Details
- Statistics
- Plot
- Interpreted Report
- List of blood pressure readings

dabl® Plot & Interpreted Text – Simple & Clearly Displayed



On the basis of the data recorded and the available literature, the ABPM suggests mild daytime systolic and diastolic hypertension (147 mmHg / 93 mmHg) and normal night-time systolic and diastolic blood pressure (111 mmHg / 65 mmHg) with a white-coat effect (158 mmHg / 90 mmHg).

ABPM Trend Report



Benefits include:

Helps physician to easily monitor patient progress.

Record if patient was on/off drug treatment when ABPM was done.

Monitor response/lack of response to drug treatment.

Use report to simply communicate patient risk/improvement.

dabl

- plots
- computer-generated interpretative reports
- eClinical applications

Questions

Supplemental Information

Common Errors in Blood Pressure Evaluation

- Observer Bias
- Technique Failures
- Equipment Error

Observer Bias

- habitually reading higher or lower than actual pressure
- terminal digit preference (150/90)
- bias for normal values
- prejudice against certain values
- variable speed of observer reaction
- differences in interpretation of Korotkoff sounds

Technique Failures

- no rest period prior to BP measure
- one arm, one position measures
- inappropriate cuff size
- poor environmental control: talking, tense.
- inappropriate rate of deflation

Equipment Error

- mercury devices not calibrated or faulty components
- aneroid devices as above
- mechanical and electrical devices: numerous potential problems

Routine Laboratory Tests

Investigation of all patients with hypertension

1. Urinalysis
2. Complete blood count
3. Blood chemistry (potassium, sodium and creatinine)
4. Fasting glucose
5. Fasting total cholesterol and high density lipoprotein cholesterol (HDL), low density lipoprotein cholesterol (LDL), triglycerides
6. Standard 12-leads ECG

Optional Laboratory Tests

Investigation for specific patient subgroups

- For those with diabetes or renal disease: assess urinary protein excretion, since lower blood pressure targets are appropriate if proteinuria is present.
- For those suspected of having an endocrine cause for the high blood pressure, or renovascular hypertension, see following slides.
- Other secondary forms of hypertension require specific testing.

Screening for Hyperaldosteronism

Should be considered for patients with the following characteristics:

- Spontaneous hypokalemia (<3.5 mmol/L)
- Profound diuretic-induced hypokalemia (<3.0 mmol/L)
- Hypertension refractory to treatment with 3 or more drugs
- Incidental adrenal adenomas

Screening for Hyperaldosteronism

Screening for hyperaldosteronism should include plasma aldosterone and plasma renin activity

- measured in morning samples
- taken from patients in a sitting position after resting at least 15 minutes.

Aldosterone antagonists, ARBs, beta-blockers and clonidine should be discontinued prior to testing.

A positive screening test should lead to referral or further testing.

Notes

11:00 - 12:00 Is My Patient Fit to Fly?

Peter Rohan MD, DOH

Program Director, Inter University Occupational &
Environmental Health Clinic, Montreal Chest Institute, MUHC

Research interests: Program Director of the Occupational and Environmental Health Clinic

- In charge of pre and post graduate teaching
- Member of the American Occupational and Environmental Clinics
- Studies and training at the University of Montreal, McGill University and the University of North Carolina

Is My Patient Fit to Fly?

Dr Peter Rohan
Program Director
Inter-University Occupational and Environmental Health Clinic
MCI – MUHC
Aviation medicine consultant
Tel.: 514-934-1934
E-mail: peter.rohan@muhc.mcgill.ca

Objectives

- Learn to identify patients who should not fly with a commercial aircraft
- Learn how to minimize the effect of flying on your patient
- Familiarize yourself with airline guidelines and their applications

OUTLINE

- Effects of altitude
- Case presentations
- Conditions incompatible with flying
- Conditions that require preventive measures
- Conclusions

Medical Problems on Airplanes

- Pressure
- Oxygen
- Immobilization
- Shared air
- Radiation
- Toilets

Atmospheric pressure and Altitude

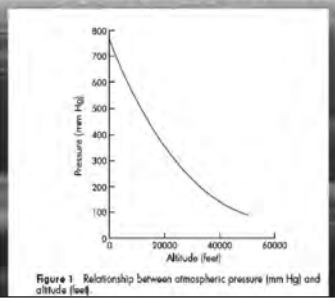


Figure 1 Relationship between atmospheric pressure (mm Hg) and altitude (feet).

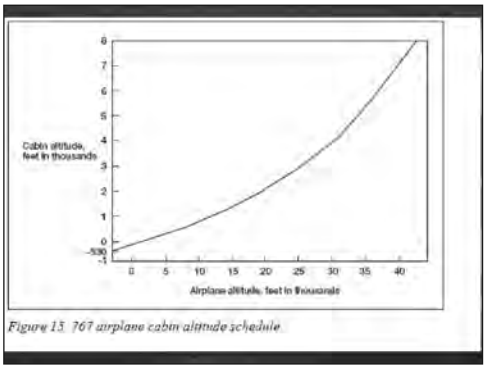
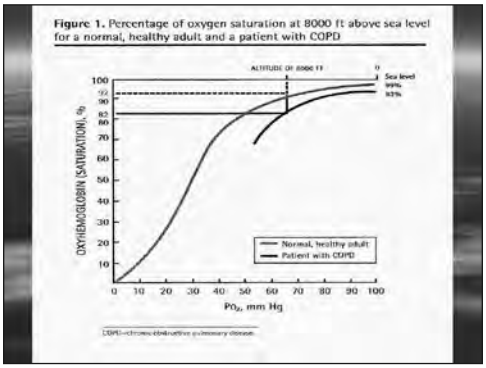
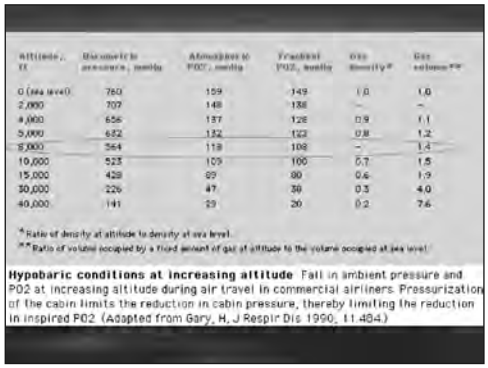


Figure 13. 767 airplane cabin altitude schedule.

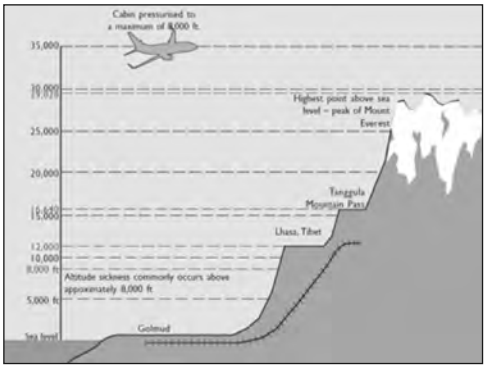




Altitude, ft	Barometric pressure, mmHg	Atmospheric P_{O_2}, mmHg	Fractional P_{O_2}, mmHg	Gas density*	Gas volume**
0 (sea level)	760	159	149	1.0	1.0
2,000	707	148	138	–	–
4,000	656	137	128	0.9	1.1
5,000	632	132	123	0.8	1.2
8,000	564	118	108	–	1.4
10,000	523	109	100	0.7	1.5
15,000	428	89	80	0.6	1.9
30,000	226	47	38	0.3	4.0
40,000	141	29	20	0.2	7.6

*Ratio of density at altitude to density at sea level.
**Ratio of volume occupied by a fixed amount of gas at altitude to the volume occupied at sea level.

Hypobaric conditions at increasing altitude Fall in ambient pressure and PO2 at increasing altitude during air travel in commercial airliners. Pressurization of the cabin limits the reduction in cabin pressure, thereby limiting the reduction in inspired PO2. (Adapted from Gary, H, J Respir Dis 1990; 11:404.)



Case # 1

- *A 52 year old businessman was hospitalized for 3 days for an uncomplicated MI*

  One week later he asks you to fill out a form from the airline enabling him to fly to Japan for a previously planned business trip
- *He is slightly obese, does not smoke, drinks socially (?) and takes Meds:*
  - *Atenolol*
  - *Lisinopril*
  - *Atorvastatin*
  - *ASA 80mg*
- *Cardiac rehabilitation program*

Case # 2

- *A 74 year old retired lawyer wants to visit her daughter in Paris. You have been following this patient for many years for HTN, COPD and has a history of MI 12 y ago.*
- *Meds:*
  - *Hyzaar*
  - *Atenolol*
  - *Enalapril*
  - *ASA*
  - *Lipitor*
  - *Advair*
  - *Ventolin prn*

Case # 3

- A 32 y old severe asthmatic patient had an appendectomy 5 days ago and consults you for an acute URI. She also suffers from colitis. Again, she noticed blood in her stool. Her family comes originally from Greece and she found out yesterday that her aunt has died. She wants to go to the funeral and asks you to sign a form that she can fly.
- Meds:
  - Advair
  - Ventolin prn
  - Asacol
  - Prednisone

Case # 4

- *A 28 y old patient has been treated for a spontaneous Pneumothorax in ER and discharged after 24 hr observation and oxygen therapy. The chest X-ray showed a 2 cm right sided pneumothorax. 4 days later on a follow-up visit the chest x-ray showed good improvement but not a complete resolution. The patient remained asymptomatic.*
- *The patient planned for months a trip to Europe and his flight is in 3 days.*

Case # 5

- A 55 y old patient was hospitalized for a DVT above the knee and subsequent PE. She came out of the hospital and already has a ticket to fly to Vancouver to see her daughter. She asks you to fill out a "Fitness to fly" form for an airline:
- Meds:
  - Warfarin
  - Lipitor

Case # 6

- You saw a 23 y old in ER after a ski accident. He had an ankle fracture which has been reduced and casted. He wants to fly back home to LA.

Case # 7

- A 69 y old with a controlled CHF and HTN had an angioplasty post a non complicated MI. He wants to travel 3 days after the angioplasty to a convention in San Diego.
- *Meds:*
  - Altace
  - Lopressor
  - ASA
  - Lipitor

- *Cardiovascular*
  - Unstable Angina
  - MI Killipp I – 7 days
  - MI complicated - 4 – 6 weeks
  - CHF – uncontroled
  - CVA - 7 days
  - Angioplasty post MI (non-complicated) - 7 days
  - CABG - 4 days for flights < 2 hrs (Hb – 90)
    - 7 days (Hb - 90)
  - Anemia - < 90

- *Respiratory:*
  - Active pneumothorax
  - COPD
    - PaO_2 > 70 mm Hg
  - Acute pneumonia
- *CNS:*
  - CVA - 5 days (first 2 weeks with O_2
  - Sub-arachnoid hemorrhage
  - TIA - 48 hrs after the attack
  - Seizure uncontrolled
  - Craniotomy - 7 days
  - Skull fracture - 7 days (if air present)

- *Gastrointestinal*
  - Intestinal obstruction complete or incomplete
  - Abdominal surgery - 10 days if the lumen has been opened
  - Gastrointestinal hemorrage (if from ulcer 7 days after it stopped)
  - Acute colitis or diverticulitis
- *ENT & Eyes:*
  - Acute otitis or sinusitis
  - Pneumatic retinopexy
    - With SF6 - 2 weeks
    - C3F8 - 6 weeks

- *Contanious diseases:*
  - Chicken pox - 5 days
  - German measles (rubella) - 4 days
  - Red measles (rubeola) - 4 days
  - Mumps - 9 days
  - TB - 2 weeks after beginning treatment
  - MRSA - wound drainage or pneumonia resolved
- *Pregnancy:*
  - Including 36th week - and no signs of imminent delivery
- *Newborns:*
  - 7 days - 48 hrs for short trips

PERSONAL oxygen Concentrators accepted by Air Canada

- AirSep LifeStyle
- Airsep freeStyle
- Inogen One
- SeQual Eclipse
- Respironics EverGo

References

- Medical Guidelines for Air Travel - 2nd Edition – Aerospace Medical Association, Medical Guidelines Task Force Alexandria, VA – Aviation, Space, and Environmental Medicine – Volume 74 Number 5 Section II, Supplement May 2003
- IATA – International Air Transport Association – Medical Manual 1st Edition June 2004
- Assessment of the cardiac patient for fitness to fly: Can J Cardiol Vol 20 No 13 November 2004

Notes

11:00 - 12:00 Heart Failure Management

Richard Sheppard MD
Attending Cardiologist, Division of Cardiology,
SMBD - Jewish General Hospital

Research Interests: Dr. Richard Sheppard is an Attending Cardiologist in the JGH Division of Cardiology, where he is a subspecialist in congestive heart failure. Among his areas of interest are the impact that different forms of genes can have on congestive heart failure; recent-onset cardiomyopathy (deterioration in the function of the heart muscle); and exercise physiology and congestive heart failure.

He obtained his M.D. from McGill University and went on to receive training and certification in internal medicine at the Royal Victoria Hospital (1996-1999), and in cardiovascular medicine at the McGill University Health Centre (1999-2002). In addition, he pursued a Congestive Heart Failure/Cardiac Transplant Fellowship at the University of Pittsburgh Medical Centre (2002-2003), and a Cardiovascular Research Fellowship in congestive heart failure at the Montreal Heart Institute (2003-2004).

Notes

Monday, Nov. 23 – Workshop A-08

11:00 - 12:00 ECG Interpretation

Marcel Fournier MD
Division of Cardiology, MUHC

Dr. Marcel Fournier is a staff cardiologist at the McGill University Health Center as well at St-Mary's hospital in Montreal. He is an associate professor at McGill University and is actively involved in teaching medical students and residents. His other involvements include chairing the council of physicians, dentists and pharmacists at St-Mary's hospital. He is a frequent contributor and presenter at medical grand rounds, as well as many conference venues. Dr. Fournier will continue his quest for the prefect bottle of red wine....a goal he hopes never to achieve !

Notes

Monday, Nov. 23 – Lunch Symposium

12:00 - 12:45 Satellite Symposium

Chair • **Heather Abrahams**

Diabetic Peripheral Neuropathic Pain (DPNP):
Pathways to Optimal Clinical Outcomes

Angela Genge MD

Supported through an unrestricted educational grant from Boehringer-Ingelheim.

Notes

13:30 - 14:00 Is There a Doctor in the Stands?

(Maintaining an Airway in Athletes – What Works?)

J. Scott Delaney MDCM, FRCP(C), FACEP
Research Director, Department of Emergency Medicine, MUHC;
Team Physician, Montreal Alouettes and Impact

Research Interests

Dr. J. Scott Delaney practices emergency medicine and sport medicine at McGill University in Montreal, Quebec. He has a fellowship in sport medicine and is the research director for the McGill University Health Centre Adult Emergency Department. He is an associate professor at McGill University and is a team physician for the Montreal Alouettes, Montreal Impact, McGill Football, McGill Men's and Women's Soccer teams and Cirque du Soleil. He is a member of the editorial board for the Clinical Journal of Sport Medicine and his research interests include concussions and neck injuries in both the athletic and emergency department populations.

Learning Objectives

1) Understand the unique and difficult nature of managing an airway in an unconscious athlete

2) Become familiar with cervical spine immobilization and different log rolling procedures

3) Learn different airway management technique in an unconscious athlete

I- Introduction

The maintenance of an airway and assisted breathing in an athlete wearing protective equipment who has become obtunded or unconscious is a serious concern for sport medicine professionals. Options available include simple airway procedures such as a jaw thrust maneuver, placement of an oral airway to improve ventilation, adjunctive airway devices such as a bag-valve-mask (BVM), laryngeal mask airway (LMA), Combitube™, and finally definitive airway control with endotracheal intubation or a surgical airway. The issues are complicated by minimal research on the subject, the relative infrequency of prolonged airway compromise, the comfort level of sport medicine professionals with the myriad of airway equipment, the presence of sports equipment and the possibility of a cervical spine injury in most athletes who have been knocked unconscious or remain obtunded after a collision.

A. Sports Equipment:

The presence of mouthguards in a variety of sports can lead to occlusion of an airway in an unconscious or obtunded athlete. These mouthguard should always be assumed to be present and should be sought during the "Look, Listen, Feel" assessment of the airway.

In football and ice hockey players, the large shoulder pads worn by most players position them such that when they are injured in a supine position, the helmet and shoulder pads are usually left in place so as to maintain a neutral cervical spine alignment. If the helmet is removed, the head and neck usually fall into an extended position, possibly further complicating an existing cervical spine injury. As such, most experts agree that when a football or ice hockey player has sustained a possible cervical spine injury, the helmet should be left in place while the facemask or visor is removed, or both helmet and shoulder pads should be removed simultaneously. Recently,

helmets in football and ice hockey have become larger in an effort to provide more protection. Thicker padding inside the helmets causes them to project further out on the forehead, possibly obscuring visualization of the airway from the head or top of the athlete. The outer shells of many helmets now extend to cover more of the face and jaw area, often obscuring the angle of the mandible. Inflatable bladders at the ear and side of the face inside newer football helmets allow for better fit and protection, but are not easily removable, and can interfere with access to the angle of the mandible. Shoulder pads have also been getting larger, sometimes encroaching on the jaw and neck area of an unconscious supine athlete. All of these changes may adversely affect airway management in the obtunded or unconscious athlete.

B. Cervical Spine Injury:

The possibility of a cervical spine injury often complicates airway management in an injured athlete, as the cervical spine is ideally splinted in a neutral position. This is most often accomplished with someone, often a therapist, positioned at the head of the supine athlete and holding the helmet or head in a neutral (in-line) position. Unfortunately this necessary procedure allows for less access to the airway, as there is less physical space for the sport medicine professional to maintain or control the airway at the head of the athlete.

II- Bag-Valve-Mask (BVM) Ventilation

BVM ventilation is probably the most important airway skill any physician or therapist can have in his or her airway management armamentarium. This basic but vital airway management technique allows for oxygenation and ventilation of patients until the athlete regains airway control or a more definitive airway can be established. It is also occasionally the only option available when more definitive airway control such as endotracheal intubation has failed or is impossible. In the injured athlete, BVM ventilation requires a good seal and a patent airway. As such, as in any unconscious patient, the airway should be assessed for obstruction and foreign body (ex. is there mouthguard that has fallen into the posterior pharynx) using the "Look, Listen, Feel" assessment of the airway before ventilation is attempted. Having the correct sized mask will help a good seal and thus good ventilation.

Certain factors may predict difficult BVM ventilation in athletes and these include:

-the presence of facial hair
-lack of teeth
-a large body mass index (BMI)
-history of snoring (probably not going to find this out in an emergency situation!)

****A- Techniques of BVM ventilation**

1-**One person BVM ventilation (1-BVM):** this involves holding the jaw, usually at the angle of the mandible with one hand, and thrusting it forward while holding the facial mask over the mouth with the same hand (usually the left). The other hand (usually the right) is used to pump the bag (See Figure1).

2-**Two person BVM ventilation (2-BVM):** this involves one person using two hands to control the jaw and maintain the mask over the mouth while a second person holds the bag and pumps the bag to provide ventilation. This is sometimes called the "4 hand technique" (See Figure 2).

3-**In-line immobilization and ventilation (IIV):** this newly developed technique involves the person controlling the airway crouching behind and to the left side of the person maintaining the in-line immobilization and attempting to place the mask of the BVM in a proper position by him or herself. Again, this involves holding the jaw, usually at the angle of the mandible, and thrusting it forward while holding the mask over the mouth with the same hand (usually the left) so the other hand can pump the bag. The arm of the hand holding the bag in this position is placed around and over the head of the person maintaining the in-line immobilization of the cervical spine (See Figure3).

B- Difficulties in BVM Ventilation:

When difficulties are encountered in BVM ventilation, there are a number of techniques and actions that can be taken to improve the quality of ventilation.

1) Difficult facemask seal:

- proper size facemask – too big better than too small
- proper inflation of mask cuff
- if edentulous: place false teeth back in
- consider inserting guaze sponges into cheeks
- if beard: lubricant to beard to mat down hair
- two hands to maintain mask seal

2) Upper airway obstruction

- rule out foreign body – "Look, Listen, Feel" +/- larynoscope and Magill forceps
- optimize position of head and neck – triple maneuver if c-spine clear (head tilt, jaw lift, mouth opening)
- two-hand technique to maximize jaw thrust and chin lift
- oropharyngeal and or nasal airway (adjuncts such as oral and nasal airways can aid with ventilation by relieving physiologic obstruction and by opening up the hypopharynx)

3) Poor resistance and compliance

- two-person BVM= four hand technique
- try to minimize peak pressures to avoid stomach distension

III- Algorithm for Difficulty "Bagging"

- Remove FB – fingers, Magill forceps
- Triple maneuver if C-spine clear (head tilt, jaw lift, mouth opening)
- Nasal or oropharyngeal airways; aid with ventilation by relieving physiologic obstruction and by opening up the hypopharynx.
- Two-person BVM = four-hand technique
- Do not abandon bagging unless it is impossible with two people and both an OP and NP airway

Remember that the first response to failure of bag-mask intubation is always better bag-mask ventilation

Figure 1: One person BVM ventilation (1-BVM)

Figure 2: Two person BVM ventilation (2-BVM) or "4 hands" ventilation

Figure 3: In-line immobilization and ventilation (IIV)

Notes

14:00 - 14:30 Contraception

Cleve Ziegler MD, FRCSC, CSPQ
Assistant Professor, Department of Ob/Gyn, McGill University;
Attending Physician, Department of Ob/Gyn,
SMBD–Jewish General Hospital

Research interests:

Dr. Ziegler is a member of the department of Obstetrics and Gynecology at the Jewish General Hospital since 1994 and an Assistant Professor in the department of Obstetrics and Gynecology at McGill University.

His interests include contraception, colposcopy and gynecologic surgery, and CME.

Learning Objectives:

1. To be aware of new delivery systems in contraception
2. To understand the pathophysiology and management of breakthrough bleeding with hormonal contraception.
2. Familiarity with extended cycle control and it's potential benefits in treating associated menstrual cycle pathologies.

Update on Contraception: New Choices, New Regimens

Cleve Ziegler. M.D., FRSC
60th Annual Refresher Course
November 23, 2009

Contraception:Beyond The Condom.....

Greeks and Jews:Medicine

Hippocrates | Maimonides

Greeks and Jews:Scholarship

Greeks and Jews: Food

Pikilia | Smoked Meat

Greeks and Jews:Family

Yia-Yia | Bubbe

Greeks and Jews:Dance

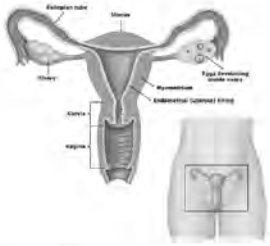
Hora

Hippocrates Refusing Gift
(No Known Jewish Equivalent)

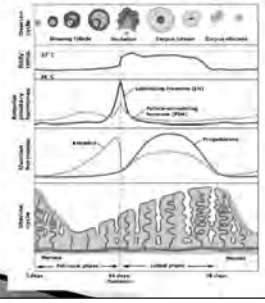
Outline of This Presentation

- Physiology of Menstruation
- Anthropology of Menstruation
- Cultural Attitudes Toward Menstruation
- Update In New Contraceptive Methods
- Concept of Extended Cycle Contraception and Menstrual Suppression

Menstruation:Anatomy



Menstruation:Normal Physiology



Menstruation:Good or Bad?

- Ridding the body of toxins
- Sign of fertility and femininity
- Physiological anemia and reduction in cardiovascular disease

- Dysmenorrhea
- Menorrhagia
- Endometriosis
- Ovarian cancer
- Breast cancer
- Premenstrual syndrome
- Migraine headache
- Epilepsy

Normal Physiological Process | Pathological Entity

Menstrual Disorders:Cost

- Affects 250,000 Canadian women /year
- 10-15% of ER visits in women 15-44
- 40% require regular analgesics
- 25% reduction in productivity during menses
- Economic cost 8-10% of total wages
- 20% of women with abnormal bleeding undergo hysterectomy

Menstruation:Anthropological Perspective

- Pollution and impurity
- Seclusion
- Rite of Passage
- Ultimate sign of femininity
- Attempts to suppress menstruation as a male intrusion

Menstruation:Ethnic Preferences

Cultural Preferences | Geographic Trends

Attitudes Towards Menstruation

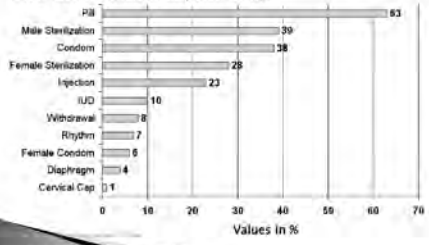
- Culture
- Age
- Parity
- Economic Status
- Educational level
- Presence/absence of menstrually associated symptoms

Preferred Frequency of Menses

	Age category (years)					
	Dutch women			Rokan women (without menstruation-related symptoms)		
	15-19 (n=322)	25-34 (n=325)	45-45 (n=324)	20-29 (n=22)	30-39 (n=171)	40-49 (n=77)
Monthly	50	30	32	9 (41%)	70 (40%)	31 (40%)
Every 3 months	35	24	10	10 (45%)	35 (21%)	15 (19%)
Every 6 months	8	8	4	2 (9%)	7 (4%)	2 (3%)
Yearly	3	4	5	0 (0%)	1 (<1%)	2 (3%)
Never	26	34	51	1 (5%)	49 (28%)	27 (35%)

Opinions About Contraceptive Methods. Percentage of Respondents with "Very Favourable" Opinion,

2002 Canadian Contraception Study.



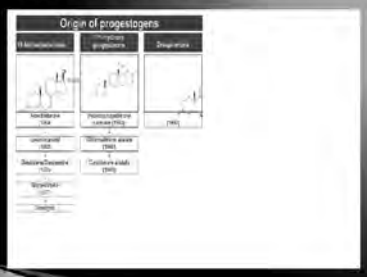
The Oral Contraceptive

21/7 Phasic | 21/7 Phasic

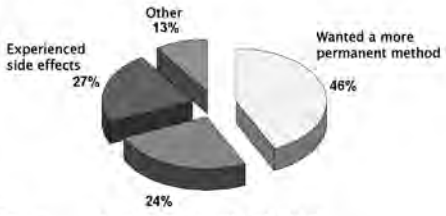
Change in Estrogen and Dose

Thorneycroft IH. *Infect Clin North Am.* 2000;11:515-529.

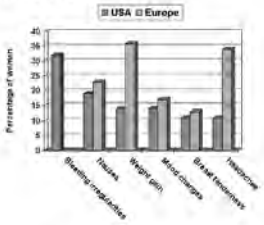
Evolution of progestogens



Reasons for discontinuation of oral contraception



Side effects leading to discontinuation

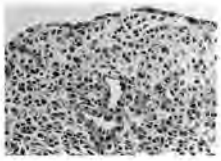


Breakthrough Bleeding on Hormonal Contraception

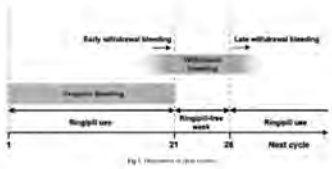
- WHO Definitions:
- ***Breakthrough Bleeding***:bleeding requiring sanitary protection
- ***Spotting***:Not requiring sanitary protection

Histology of Endometrium

- Glandular atrophy
- Stromal atrophy
- Stromal edema
- Thin blood vessels
- Sinusoidal veins
- Vascular ectasia



Cycle Control:Definition



Problems with the 28-Day Cycle

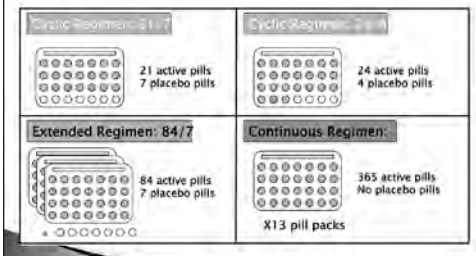
- 262 women on OCs charted symptoms throughout menstrual cycle
- Higher rates of symptoms in 7-day hormone-free interval (HFI)

Symptom (in current pill users)	Users with symptoms during active pill period	Users with symptoms during HFI	*p* value
Pelvic pain	21%	70%	<0.001
Headache	53%	70%	<0.001
Breast tenderness	43%	65%	<0.001
Bloating/swelling	16%	38%	<0.001

Extended Use
Hormonal Contraception

Definition: "Extended" oral contraception refers to the use of combined hormonal contraceptives for extended periods of time (greater than 2 consecutive 21 days) with planned HFIs.

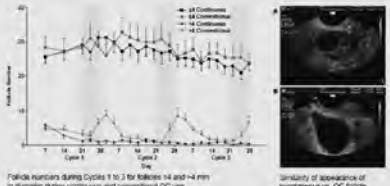
What's the Difference in OC Dosing Regimens?



Transdermal vs. Oral: Follicular Inhibition

- 124 women randomized to the patch or 1 of 3 common oral contraceptives
- Ovarian ultrasounds over 5 months
  Any follicle ≥12 mm followed daily

Follicle Growth During the HFI

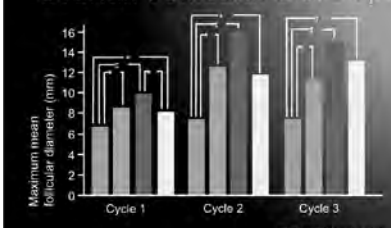


Follicle numbers during Cycles 1 to 3 for follicles >4 and >4 mm in diameter during continuous and conventional OC use. HFI indicated by light grey shading.

Similarity of appearance of spontaneous vs. OC follicle

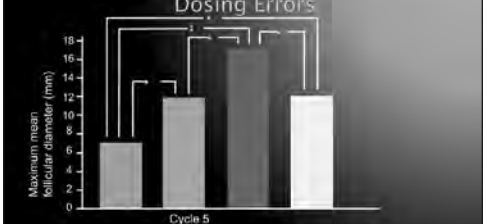
Birch RL et al. *Reprod Biol Endocrinol* 2005; 3:12.
Birch RL et al. *Contraception* 2006; 73(3):235-43.

Ovarian Follicular Development



*p ≤ 0.05; †p < 0.001

Ovarian Follicular Development After Dosing Errors



*p ≤ 0.05; †p < 0.001

Archer DF, Stanczyk FZ. *Contraception* 2003;80:34-42.

YAZ® Is Unique Among OCs...

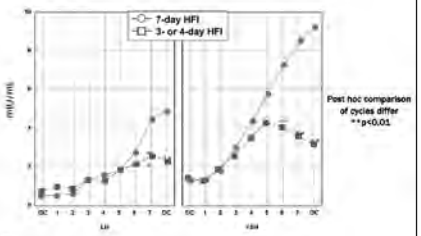
The only 24/4-day OC with drsp:

- Drospirenone (3mg) and low-dose ethinyl estradiol (20µg)
- 3 additional days of EE + drsp with anti-mineralocorticoid and anti-androgenic* activity
- 30-hour half-life of drospirenone extends its activity into the 4 day hormone-free interval

24 active pills/ 4 placebo

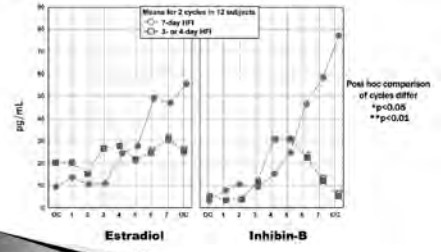
drsp = Drospirenone; EE = ethinyl estradiol
*Anti-androgenic activity seen in preclinical animal and in vitro studies

7-Day vs. 3- or 4-Day Hormone-Free Interval LH and FSH:



Study used 3mg drsp/30mcg EE.
LH = Luteinizing hormone; FSH = Follicle-stimulating hormone

7-Day vs. 3- or 4-day Hormone-Free Interval Ovarian Response:



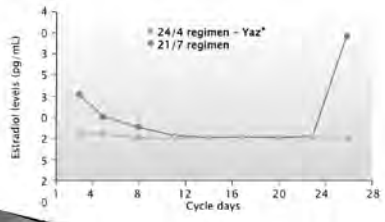
Study used 3mg drsp/30µg EE

Advantages of a 24/4-Day OC Regimen

- Shortening Hormone-free interval
  Decreases hormone fluctuations
  Decreases ovarian follicular activity
- Potential reduction in symptoms by shortening the HFI
- May increase the contraceptive safety margin (i.e. "more forgiving")

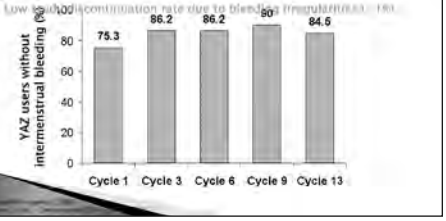
HFI = Hormone-free interval is the time when active pills are not taken.

Shorter Hormone-Free Interval Decreases Estradiol Fluctuation



Proportion of Women With No Intermenstrual Bleeding

Up to 90% of women experienced no intermenstrual bleeding
After Cycle 1, intermenstrual bleeding was predominantly limited to spotting
Low discontinuation rate due to bleeding/irregularities (~1%)



Indications/Considerations for Extended Regimen

- Contraception
- Control of menstrual cycle symptoms[1,2]
- Control of bleeding[3,4]
- Quality of life/preference

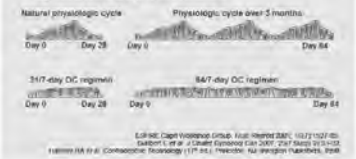
Extended Use
Approved Products

- 30 µg EE + 150 µg levonorgestrel (Seasonale™)
  – Like Min-Ovral® but 84-day cycle

Advantages of approved products
- Compliance/adherence
- Patient support
- Insurance
- Health Canada approval
- Convenience
- Well-known ability to suppress endometrium

Extended Use Regimens
Common Concerns

Doesn't this cause a buildup of blood?
- Ultrasound and endometrial biopsies after extended use regimens show atrophic endometrium



Extended Use Regimens
Common Concerns

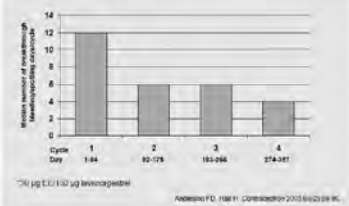
Isn't there more hormone exposure?
- Yes. However cumulative dose is minimally more than 35 µg pills and lower than that with 50 µg EE pills

Total estrogen exposure over 1 year for different marketed OCs compared to newer extended cycle regimens

Name of the combined OC	Type of regimen	Dose of EE
Alesse®	Cyclic	2,520 µg
LoEstrin®, Marvelon®, Min-Ovral®, Ortho-Cept®, Yasmin®	Cyclic	0,480 µg
Cyclen®, Ortho® 1/35, Ortho® 0.5/35	Cyclic	0,560 µg
Seasonale™	Extended	0,600 µg
Ovral®, Ortho-Novum® 1/50	Cyclic	0,600 µg

Adapted from: Guillerd E et al. *J Obstet Gynaecol Can* 2007; 29(7 Suppl 2):S4-S2

Breakthrough Bleeding and Spotting with an 84/7 OC Regimen*



*30 µg EE/150 µg levonorgestrel

Anderson FD. *Int Fertil Conservation* 2003;6(2):84-90.

Cycle Control
Bleeding Days (Mean or Median Number)

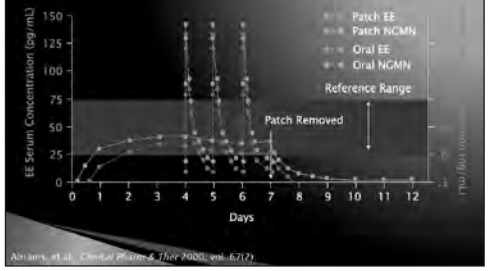
Studies	Cyclic use	Extended use	Duration of study
Miller L, Notter KM. Obstet Gynecol 2001; 98(5 Pt 1):771-8.	Mean: 10.9 days	Mean: 6.4 days	84 days
Kaunitz M et al. Contraception 2003; 67(1):9-13.	Mean: 33.8 days	Mean: 18.4 days	168 days
Anderson FD, Hait H. Contraception 2003; 68(1):89-96.	Median: 40 days	Median: 16 days	364 days
Miller L, Hughes JP. Obstet Gynecol 2003; 101(4):653-61.	Median: 10 days	Median: 3 days	84 days

The EVRA Transdermal System

Composition of EVRA: a 'Matrix' Patch

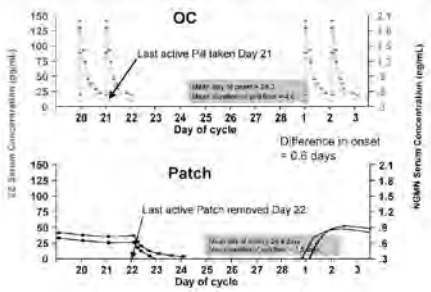
- Backing Film
  - Flexible Polyester Film
- Middle Layer
  - Norelgestromin (6 mg) / Ethinyl Estradiol (0.60 mg)
  - Adhesive
  - Non-woven polyester fabric
- Release Liner
  - Clear polyester S-cut film protecting the adhesive layer

NGMN and EE Levels: Patch vs OC



Abrams, et al. Clinical Pharm & Ther 2000; vol. 67(2)

Onset & Duration of Withdrawal Flow



Extended Use of Transdermal Norelgestromin/Ethinyl Estradiol: A Randomized Trial

EVRA Patch:Extended Use

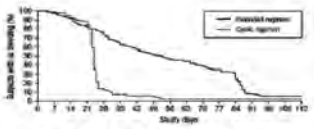


Fig. 3. Kaplan-Meier analysis of time to first bleeding for the extended and cyclic regimens. Data collected during the first week (during menses) were excluded; hence, the study started with 100% of subjects with no bleeding. Median day: extended, 54; cyclic, 25 ($P < .001$).

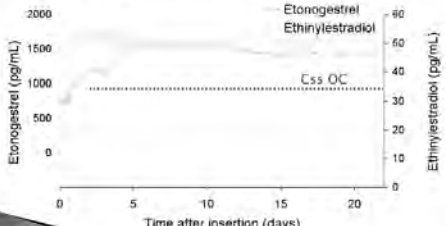
Sirum. Extended Transdermal Contraceptive Therapy. Obstet Gynecol 2005

Confronting the Legal Risks of Prescribing the Contraceptive Patch With Ongoing Litigation

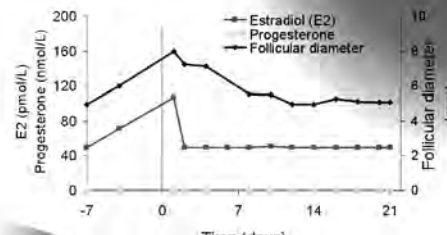
NuvaRing

- 1 ring per cycle
- Regimen:
  - 3 weeks of ring-use
  - 1 ring-free week
- Daily release:
  - 15 µg ethinylestradiol
  - 120 µg etonogestrel

Pharmacokinetic profile
NuvaRing and 30 EE/150 DSG COC



Ovulation inhibition with NuvaRing
Median follicular diameter, E2 and P concentration



Mulders & Dieben, Fertil Steril, 2001;75:865–70

Nuvaring:Extended Cycle use

Nuvaring:Extended Use

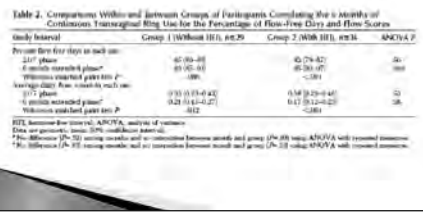
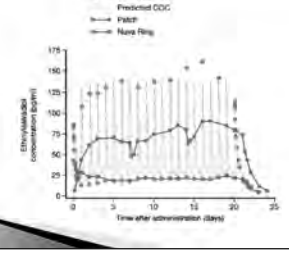


Table 2. Comparisons Within and Between Groups of Participants Combining the 6 Months of Continuous Transvaginal Ring Use for the Percentage of Flow-Free Days and Flow Scores

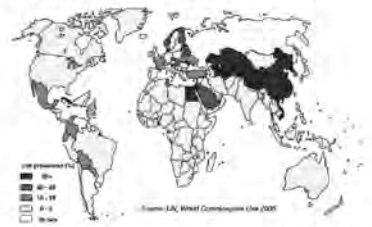
Study Interval	Group 1 (Without HRT), n=8,29	Group 2 (With HRT), n=836	ANOVA P
Percent flow-free days in each set:			
21st phase	67 (50–88)	75 (54–92)	.06
6 months matched phase*	83 (50–93)	85 (50–97)	.004
Wilcoxon matched pairs test, P	<.001	<.001	
Average daily flow scores by each set:			
21st phase	0.33 (0.23–0.42)	0.34 (0.23–0.45)	.52
6 months extended phase*	0.21 (0.12–0.27)	0.17 (0.12–0.23)	.06
Wilcoxon matched pairs test, P	.002	<.001	

HRT, hormone therapy; ANOVA, analysis of variance.
Data are percentile (median; 95% confidence interval).
*No difference ($P = .52$) among months and no interaction between months and group ($P = .69$) using ANOVA with repeated measures.
†No difference ($P = .17$) among months and no interaction between month and group ($P = .10$) using ANOVA with repeated measures.

Serum Hormonal Levels:Pill,Patch and Ring



World Wide Usage of IUD



Prevalence of IUD use in women aged 15–49, married or in union (2005)

Mirena

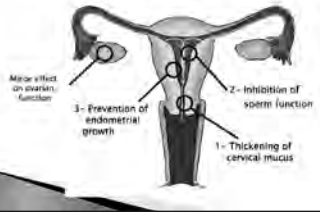
- Intrauterine system (IUS)
- Releases up to 20 µg/day o levonorgestrel (progestin)
- No estrogen
- 5 years of treatment

Indications

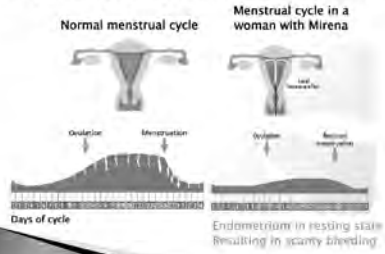
- Contraception

Contraception with LNG-IUS

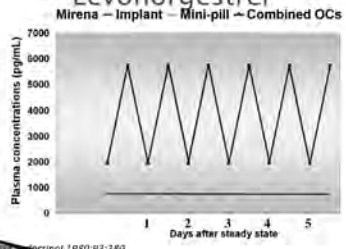
Mirena provides contraception through a combination of 3 main actions:



Effect of LNG-IUS on the Endometrium



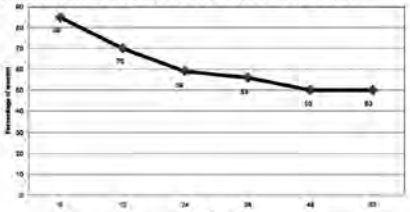
Plasma Concentrations of Levonorgestrel



Gynecological Endocrinology

Levonorgestrel-releasing intrauterine system: why do some women dislike it?

Mirena:Discontinuation Rate



Back to The Future:Depo-provera

Contraception

Review article

The use of depot-medroxyprogesterone acetate in contraception and its potential impact on skeletal health

Depoprovera Consensus 2009

Implanon

Implanon

Thank You!

Efharisto poli,my friend...

Merci, eh!

14:30 - 15:30 ER: ER Procedures

H. Mitchell Shulman MDCM, FRCPC, CSPQ

Assistant Professor, Department of Surgery, McGill University;
Associate Professor, Family Medicine, St. Mary's Hospital Centre;
Attending Physician, Emergency Room, Royal Victoria Hospital

This workshop focuses on some techniques that will get you and your patient out of trouble. We will concentrate on the ABC's:

Airway: cricothyroidotomy and other tricks to deal with a difficult airway;

Breathing: chest tube placement and management;

Circulation: venous cutdowns and intraosseous line placement.

Cricothvroidotomy

Inability to intubate the trachea is the primary indication for creating a surgical airway; specifically where oral/nasal endotracheal intubation is impossible.

For example: obstruction / facial trauma / severe hemorrhage / laryngeal fracture / laryngospasm / laryngeal stenosis / etc.

A) Surgical

Contra-indications:

1. Age: Should not be performed in children less than 5 years of age (needle cricothyroidotomy instead);
   ? in children from 5 to 10 years (note: ATLS says 12);
2. Pre-existing laryngeal pathology: cancer, inflammation, infection;
3. Inexperience with the technique!!!!!
4. Anatomical distortion;
5. Coagulopathy (!!!!!)

Technique

The cricothyroid membrane (approx. 10 mm high; 22 mm. wide) spans the space between the thyroid cartilage superiorly and the cricoid cartilage inferiorly. The vocal cords are at least 1 cm above the membrane. The superior cricothyroid vessels (branches of the superior thyroid artery) run transversely across the upper 1/3 of the membrane, which is why you're supposed to try and enter the membrane through the inferior 1/3. The anterior jugular veins run vertically laterally.

The key is *to be prepared in advance*. Have a kit of some sort already set up, checked on a regular basis and somewhere where you and your staff can find it quickly (for example: with your intubation kit). It need not be very elaborate. A scalpel (#11 blade); a cuffed tracheostomy tube (ID 6.5 or 7 e.g. #4 Shiley) are the minimum. If you want to use one of the prepared kits just make certain you've practiced with it in advance!

Surgically prep the site as time and circumstances allow. Identify the laryngeal prominence "Adam's apple" and the hyoid bone above it. If you are right-handed: stand on the patient's right side and use the thumb and the middle finger of the left hand to grasp the upper poles of the thyroid cartilage. The left index finger rests on cricothyroid membrane. Incise the skin vertically in the midline down to the membrane. Recheck your landmarks.

Leave the left index finger at the most inferior pole of the thyroid cartilage then incise the membrane transversely in the inferior 1/3 of the membrane at least 1.5 cm long. Remove the scalpel, insert a tracheal hook for traction or use a hemostat by introducing it into the incision and then opening it to introduce the tracheostomy tube (whose balloon you of course remembered to check before you started the procedure!). Remove the dilator, inflate the balloon and ventilate the patient. Confirm the tube's position (auscultate, chest x-ray).

B) Percutaneous "Needle" Tracheal Ventilation

Method of choice in children.

Contraindications:

!!Be careful with partial airway obstruction and especially with complete airway obstruction. Pneumothorax and worse can happen if there is complete expiratory obstruction.

Technique:

With the patient supine, identify the landmarks and prep and anesthetize the area as the situation allows. Puncture the skin with a # 12 or #14 gauge catheter-over-the-needle attached to a 10 ml. syringe in the midline, directly over the cricothyroid membrane at an angle of 45 degrees caudally, aspirating as the needle is advanced. The aspiration of air indicates entry into the tracheal lumen. At that moment advance the catheter while gently withdrawing the stylet. Connect to oxygen at 15L/min. Chest movement controls the time that oxygen flows into the patient (a maximum of 1 second allowing 4 seconds for passive exhalation). Secure the catheter and confirm its position (auscultation, chest x-ray).

If coughing is a problem, instill 2 ml 4% lidocaine through the catheter.

Chest Tubes

A) Tube Thoracostomy: to evacuate an abnormal accumulation of air or fluid from the normally closely approximated visceral and parietal pleura. The rate of collection and the amount that has accumulated will determine the extent of the problem!

Important clinical point: a pneumothorax is most easily seen in a full expiration view on CXR.

Indications:

Any pneumothorax if the patient is: on a ventilator / going to surgery / must be transported / open "sucking" chest wound / tension / bilateral / increasing.

Otherwise as per usual criteria.

Contraindications:

Are there really any? Need for immediate thoractomy? Clotting dysfunction? Multiple pleural adhesions / scarring?

Technique:

Set the patient up properly with an IV line in place, breathing 100% oxygen, oxygen saturation / EKG / BP monitoring applied. Measure the distance the tube will have to travel and mark the chest tube in advance (the tip will be at the apex of the lung). This will tell you how far in the tube will need to go. Prepare your water seal / suction apparatus. Use the biggest available tube for trauma (36 – 42 Fr.); an 18 – 22 Fr. for a pneumothorax.

The patient is in the semi-Fowler or in the lateral decubitus position with the arm placed out of the way. Find the 5th intercostal space (usually at the level of the nipple or the inferior scapular border), midaxillary line. Prep and anesthetize the patient as time and circumstances allow. Remember that you can use the anesthetic needle to confirm your diagnosis. Incise the skin and subcutaneous tissues approximately 3 times the width of the chest tube directly over the rib 1 interspace below the rib that the tube will eventually pass over. Use a curved hemostat to dissect the intercostals muscles and to puncture the pleura just above the upper edge of the rib. Use a gloved finger to check the incision (clear any adhesions and avoid puncturing the lung, liver, heart, spleen, etc. etc.). Grasp the end of the chest tube with a curved clamp and advance it posteriorly and superiorly into the pleural space.

Make certain that the last hole of the chest tube is well within the parietal pleura. Check the position of the tube ("fogging", air movement, x-ray). Suture the tube in place and apply a sterile waterproof dressing.

Important things to remember:

Never re-adjust a tube by advancing it (although it can be withdrawn if necessary).

Do not irrigate a tube.

To avoid re-expansion pulmonary edema (risk is increased after the rapid evacuation of a large {>1000 ml}, long-standing {> 72 hours} effusion) remove the fluid in a slow, step-wise fashion.

B) Needle Thoracentesis

Indications: Remember to think of it in the patient who presents in P (ulseless) E (lectrical)

A (ctivity) and /or with chest trauma.

Technique:

Prepare the patient as before. Prep and anesthetize as appropriate. Insert a #14 gauge over-the-needle catheter attached to a syringe just above the rib into the second intercostal space, mid-clavicular line on the side of the pneumothorax. Aspirate as much air as you can (if necessary remove the syringe!). Place a proper chest tube.

If no air escapes or the procedure provides no relief think of:

Pericardial tamponade / myocardial contusion / myocardial infarction / air embolus / etc.

Venous Cutdown

The easiest site is the distal saphenous vein just medial and superior to the medial maleolus.

Contraindications

a. previous use of that vein (vein stripping, CABG);

b. phlebitis;

c. cellulitis at the site;

d. significant trauma to that extremity;

e. severe venous obstructive disease;

Technique

Immobilize the leg. Prep and drape and anesthetize as time and the situation permit. Apply a tourniquet at the mid-leg. Use a transverse skin incision approximately 2 cm long going through all the layers of the skin (subcutaneous fat should be visible through the incision). Insert with the concavity of the clamp upward a small "mosquito" hemostat into the wound and advance scooping up all the tissue. Separate out the vein. At this point you can either catheterize it as you would any vein or place sutures under the vein; make a small incision with scissors or a scalpel and advance a catheter into the vein securing it in place with the proximal suture. Remove the tourniquet. Tie off the distal vein using the other suture. Close the wound and protect the line.

Important clinical point: Flow is directly proportional to the radius of the tube (raised to the 4th power) and inversely related to the tube length and the fluid's viscosity.

Intraosseous

Any substance that can be given intravenously can be given intraosseously. It is wise to dilute hypertonic, alkaline, or otherwise irritaring substances and administer them more slowly if the situation permits.

Contraindications:

Compromised insertion site (infected / burn / ipsilateral facture). Osteogenesis imperfecta / osteopetrosis.

Important clinical point:

For volume infusion its use is limited to children < 5 years of age. The needle tip must be directed away from the joint space and the epiphyseal plate.

Technique

In children there are 2 commonly recommended sites:

a. proximal tibia (anteromedical flat surface) 1 – 3 cm below and medical to the tibial tuberosity;

b. distal femur 2 – 3 cm above the epicondyles in the midline directed cephalad at an angle of 10 – 15 degrees from the vertical.

In adults:

Distal tibia at the ankle (1 cm above the superior margin of the medial maleolus). Remember in an adult to use an external pressure bag or pressure device delivering 300 mm Hg to the solution bag.

Immobilize the leg. Prep and anesthetize as time and the situation allow. Use an intraosseous or bone marrow needle and with the obturator still in place insert at the chosen site. Once the periosteum is reached redirect the needle at a 45 – 60 degree angle away from the adjacent joint and advance with a rotating action. When the needle "pops" into the marrow space remove the obturator, attach an empty syringe and aspirate. The needle should be able to stand securely. Use a second sterile syringe filled with normal saline to flush while you palpate the extremity. If a subcutaneous or subperiosteal fluid collection develops remove the needle, apply pressure to the site and try elsewhere.

When you no longer need the site, remove the catheter and apply local pressure and appropriate wound care as you would normally for any IV site.

Important clinical point:

the aspirated blood / bone marrow can be used for a chemstrip / routine biochemistry / paCO2 / pH / blood culture / type and crossmatch but NOT a complete blood count.

This is a valuable but temporary alternative to intravenous access. It must be replaced.

Notes

14:30 - 15:30 GER: Andropause

Peter Chan MD, CM, MSc, FRCS(C), FACS
Director of Male Reproductive Medicine, McGill University Health Centre;
Associate Professor, Department of Surgery, McGill University Health Centre

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Research Interests: Dr. Peter Chan is the Director of Male Reproductive Medicine at the McGill University Health Center. Dr. Chan has been involved in Andrology basic science research on epididymal sperm maturation since 1988 at McGill University. He received numerous national and international recognitions, including First Prize in Fundamental Research and the Annual Urological Research Award of the Canadian Urologic Oncology Group; Best Video at the 2002 American Society for Reproductive Medicine Annual Meeting; First prize in Audio-Visual Award at the American Urological Association Annual Meetings in 2002 and 2003; First Prize of Scientific Research Presentation from the Quebec Environmental Health Research Network in 2006 and the 2007 Clinical Diagnostic Award from the American Society of Andrology. Upon completing his Urology residency at McGill University, Dr. Chan was named the Canadian Institute for Health Research Scholar and the American Foundation for Urological Diseases Scholar when he received his fellowship training in Male Reproductive Medicine and Microsurgery under Drs. Marc Goldstein and Peter Schlegel at the Cornell Institute for Reproductive Medicine in New York and his fellowship in Molecular Genetics at the Population Council of the Rockefeller University. Dr. Chan is the youngest two-time recipient in the history of McGill University for the Everett C. Reid Award of Excellence in Teaching in Urology in 2003 and 2008. Dr. Chan has published over 60 professional journal articles, book chapters and videos. He is the senior editor of a textbook entitled "Reproductive Medicine Secrets" and the second edition of "The Andrology Handbook" published by the American Society of Andrology. Dr Chan is currently a principle investigator of research grants from the Canadian Institute for Health Research studying the impact of chemotherapy and cancer on male fertility. He is also featured in "Doctorology", a television documentary produced by the Discovery Channel on the various medical subspecialties.

Objectives:

At the conclusion of the session the participants will

1. Understand the association of Andropause various conditions including metabolic syndrome, erectile dysfunction and prostate diseases.

2. Be able to outline the routine investigation for men presenting with andropause and utilize proper laboratory tests for testosterone level determination.

3. Be familiar with the current choice of treatment for andropause, the protocol to follow these patients and the safety of testosterone replacement on prostate health.

TESTOSTERONE REPLACEMENT THERAPY AND PROSTATE HEALTH

Peter Chan M.D., C.M., M.Sc., F.R.C.S.(C)., F.A.C.S.

Director of Male Reproductive Medicine,
Department of Urology, McGill University Health Center

OBJECTIVES

At the conclusion of this workshop, participants should be able to:

1) Describe the presentation and diagnostic strategies of hypogonadism
2) Outline the treatment options and counseling strategies for late-onset hypogonadism
3) Evaluate the risks prostate problems with testosterone replacement therapies.

ANDROPAUSE

ADAM - Androgen Deficiency in Aging Men
LOH – Late Onset Hypogonadism

Recognition from various international organizations

WHO
NIH
International Society for the Study of Aging Male

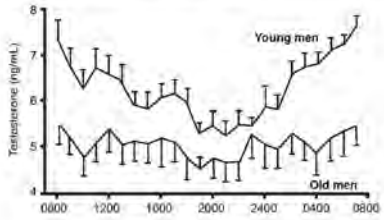
Prescription of testosterone products ↑ 8-fold since 1993!

ANDROPAUSE - FACTS

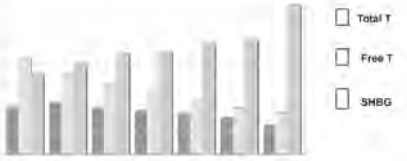
Over 60% of men > 65 yr have low free testosterone

Only 5% of these men are treated

TESTOSTERONE - DIURNAL VARIATION



AGE EFFECT ON TESTOSTERONE



Mean serum levels of testosterone, free testosterone (FT) and sex-hormone binding globulin (SHBG) according to age in a cross-sectional study of 300 healthy men.

ANDROPAUSE - PRESENTATION

Must have a high index of suspicion!

-Frailty/Fatigue
-Decreased energy and work capacity
-Decreased muscle strength and mass
-Increased abdominal fat
-Fractures and back pain (osteoporosis)
-Decreased sexual desire
-Erectile dysfunction

METABOLIC SYNDROME

A clustering of metabolic derangements:

1) Diabetes / Insulin resistance
2) Cardiovascular diseases / HTN
3) Central obesity/dyslipidemia
4) Endothelial dysfunction
5) Systemic inflammation
6) Erectile dysfunction
7) Hypogonadism

Low T levels may predict future onset of Type II Diabetes or Metabolic Syndrome

Author / Publication	N	Age	Results/Conclusions
Laaksonen, et al *Diabetes Care* 2004; 27(5): 1036-1041	702	42 – 60	Men with low testosterone were significantly more likely to develop either metabolic syndrome or diabetes Among men monitored for 11 years, those in the lowest testosterone quartile had a 2.3-fold higher risk of both outcomes
Muller, et al *J Clin Endocrinol Metab* 2005; 90(5):2618-2623	400	40 – 80	Levels of testosterone and SHBG were inversely associated with metabolic syndrome and insulin resistance Each unit increase (1 SD or 5.3 nmol/L) in total testosterone level reduced the risk of metabolic syndrome by 57%
Kupelian, et al *J Clin Endocrinol Metab* 2006; 91(3):843-850	1709	40 – 70	Low total testosterone and low serum SHBG are associated with increased risk of developing MetS over time, particularly non-overweight, middle-aged men (BMI<25)

DEFINITION OF MetS

Central obesity: Waist circumference—ethnicity specific
Plus any two:

Raised triglycerides
>150 mg/dL (1·7 mmol/L)
Specific treatment for this lipid abnormality
Reduced HDL-cholesterol
<40 mg/dL (1·03 mmol/L) in men
<50 mg/dL (1·29 mmol/L) in women
Specific treatment for this lipid abnormality

Raised blood pressure
Systolic ≥ 130 mm Hg
Diastolic ≥ 85 mm Hg
Treatment of previously diagnosed hypertension

Raised fasting plasma glucose
Fasting plasma glucose ≥100 mg/dL (5·6 mmol/L)
Previously diagnosed type 2 diabetes
If above 5·6 mmol/L or 100 mg/dL, oral glucose tolerance test is strongly recommended, but is not necessary to define presence of syndrome

Lancet, Volume 366, Issue 9491, Pages 1059-1062 (24 September 2005-30 September 2005)

ANDROPAUSE – SCREENING TOOL

ADAM Questionnaire

1. Do you have a decrease in sex drive?
2. Do you have a lack of energy?
3. Do you have a decrease in strength and/or endurance?
4. Have you lost height?
5. Have you noticed a decreased enjoyment of life?

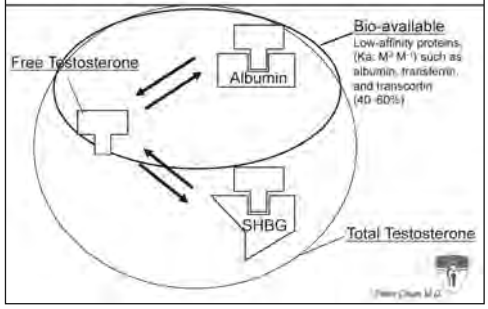
ANDROPAUSE – SCREENING TOOL

ADAM Questionnaire

6. Are you sad and/or grumpy?
7. Are your erections less strong?
8. Has it been more difficult to maintain your erection ?
9. Are you falling asleep after dinner?
10. Has your work performance deteriorated recently?

Q1 and Q7 plus 3 more yes → Do blood test!

TESTOSTERONE TESTS



SO HE'S IN ANDROPAUSE, NOW WHAT?

1. Make sure there is no contraindications for treatment
2. Pre-treatment baseline evaluations
3. Treatment
4. Follow-up

CONTRAINDICATIONS

Absolute:
1. Known or suspected prostate cancer
2. Known or suspected male breast cancer
3. BPH with severe voiding symptoms
4. Desire fertility

Relative:
1. Polycythemia
2. Uncontrolled HTN
3. High risk of prostate cancer
4. Psychological instability
5. Uncontrolled cardiac insufficiency
6. Liver dysfunction

TREATMENTS FOR ANDROPAUSE

•Therapeutic choices include:

- **Intramuscular** preparations
  - (Delatestryl®, Depo-Testosterone Cypionate)
- **Oral capsule** (Andriol®)
- **Transdermal patch** (Androderm™)
- **Transdermal gel** (AndroGel™, Testim™)

SIDE EFFECTS

- Acne[1]
- Lab test abnormalities (polycythemia, PSA)[2]
- Gynecomastia[2]
- Weight gain[1]
- Worsening sleep apnea[2]
- Gastrointestinal upset (testosterone undecanoate)[1]
- Skin reactions (transdermal patches > gel)[3]

1. Bain J. *Can Fam Physician* 2001
2. Tenover JL. *Endocrinol Metab Clin North Am* 1998
3. Wang C. *J Clin Endocrinol Metab* 2000

FOLLOW-UPS

3 months, 6 months, 12 months, Q year

1. Re-evaluate andropause symptoms
2. BP, DRE, Lower limb edema, Weight gain
3. Blood tests

DOES TESTOSTERONE REPLACEMENT WORK?

Large volume of recent literature:

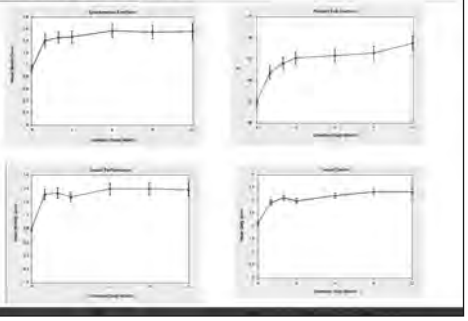
- Improves sexual function (libido and erection)
- Cognitive function
- Mood
- Muscle strength
- Body composition, Bone mineral density

Study Results

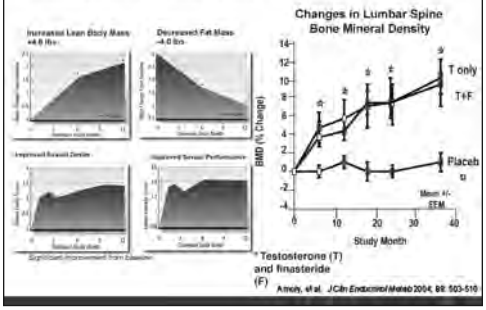
- Significant percentage change increase in bone mineral density
- Significant improvements in body composition parameters, including Lean Body Mass, Fat Mass, and %Fat
- Significant improvements in mood
- Significant improvements in sexual function parameters
  - Sexual Performance
  - Sexual Motivation
  - Sexual Desire
  - Satisfaction with Duration of Erection
  - Percentage of Full Erection
  - Spontaneous Erections

Green J. et al. *Reviews in Urology* 2005; 7: S37-S44

Study Results



Proven Positive effects of TRT



Amory, et al. *JClin Endocrinol Metab* 2004; 89: 503-510

Testosterone replacement therapy effects

- RCT with Chronic stable angina, 3 mo of TRT ↓exercise-induced MI
  English et al. *Circulation* 2000
- Obese, hypogonadal men with DM, TRT normalized BP
  Boyanov et al. *Aging*
- Obese, hypogonadal men with DM, TRT
  ↓ fasting glu
  ↓ HbA1c
  ↑insulin sensitivity
  Marin. *Obes Res Suppl* 1995; Boyanov et al. *Aging Male* 2003

Androgens and the Prostate

- TRT increases prostate volume and PSA to that noted in age-matched controls with normal T levels
- The rise in prostate volume and PSA with TRT is usually modest- approx 15%
- No changes in uroflow, postvoid residual, or prostate symptom scores

Randomized Controlled Trial

- Hypogonadal men 44-78 yrs
- Randomized to 6-mo TRT vs placebo
- TRUS-Bx of prostate pre and post TRT

IPSS voiding Sx score:	no sig changes
Flow rate:	no sig changes
PSA	1.55 →2.29
PCa:	10% in TRT vs 21% in placebo
High-PIN	10% in TRT vs 14% in placebo

Marks et al. *JAMA* 2006

Prostate Cancer: MMAS

Are Serum Hormones Associated With The Risk Of Prostate Cancer? Prospective Results From The Massachusetts Male Aging Study

- **N = 1,576 men - Approximately 8 year follow-up**
- **70 men (4%) developed prostate cancer**
- **No correlation with:**
  - **Total testosterone**
  - **Free testosterone**
  - **SHBG**
  - **Androstenedione**
  - **Estradiol**

TESTOSTERONE AND PROSTATE

No increased risks of prostate cancer in the current literature

At eugonadal T levels, no increase in BPH

PSA may increase → close follow-up (may require investigations)

Future trend: organ confined prostate cancer that is treated may be ok to receive testosterone for hypogonadism

Agarwal & Gelfand. 2005

SUMMARY

- Evidence that aging is associated with decline in testosterone production
- Significant impact on quality of life and physical health
- Association with Metabolic Syndrome
- Careful screening, testing necessary
- Alleviation of symptoms possible with appropriate treatment
- Testosterone replacement is effective and safe

Notes

Monday, Nov. 23 – Workshop B-03

14:30 - 15:30 PEDS: Ortho in Newborn and Very Young

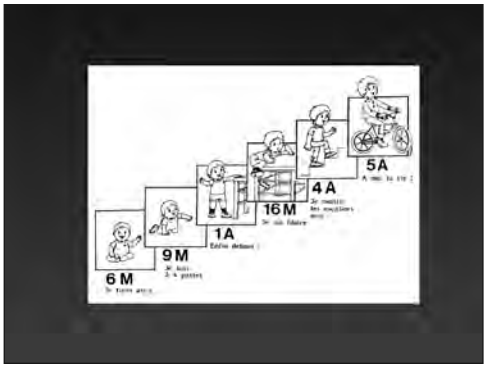
Thierry E. Benaroch MD, FRCS(C), FAAOS
The Montreal Children's Hopsital – MUHC

Research interest:

Thierry Benaroch is an Assistant Professor at McGill University in the Department of Surgery. He specializes in Pediatric Orthopedic Surgery and is presently the Director of Orthopedic Surgery at the Montreal Children's Hospital. He is also an active staff member at the Shriners Hospital for Children. As well, he is extensively involved in rehabilitation centres; such as, the Mackay Centre and Peter Hall School.

Newborn to Child Basic Office Orthopedic Principles

Thierry Benaroch, MD, FRCS(C)
MUHC



Neuro-orthopedics

- Walk with the head… not the feet!
- Normal gait development can take up to 3 years…

Neuro-orthopedics

- Delayed walk is very rarely due to an orthopedic problem
- Check birth history:
  - Term, conditions
  - Birth weight
  - Apgar score
- Evaluate development stage
- Neurologic exam

Neuro-orthopedics

- A child hops on one foot at age 4-5

The Newborn Hip: When to Refer

History

- The 4 "**F**'s"

History

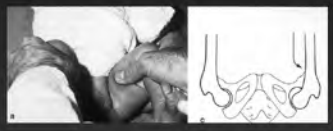
- **F**irst born
- **F**emale (13:1)
- **F**rank breech (hips flexed, knees extended)
- **F**amily history

Physical Exam

- Baby must be relaxed
- If crying, examine hip later
- Gentle exam

Physical Exam

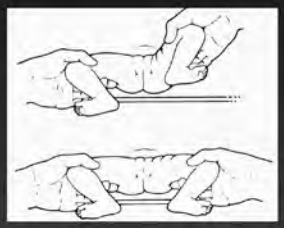
Barlow – dislocate reduced hip



Physical Exam

Ortolani $^{+ve}$ – reduce a dislocated hip

Ortolani $^{-ve}$ – not able to reduce a dislocated hip



Physical Exam

Click:

- Benign
- Not a "clunk"
- No significance

Physical Exam

Barlow, Ortolani → up to 4 – 6 weeks of age

Click → up to 4 – 6 months of age

Physical Exam

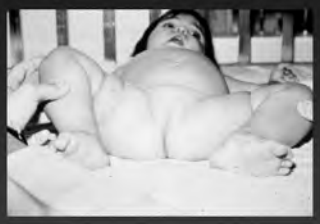
If dislocated hip not picked up by 4 – 6 weeks of age then generally lose Barlow, Ortolani manoeuvre.

Late physical signs of dislocated hip appear, but only by 4 – 5 months of age.

Physical Exam
Late Signs

Decreased hip abduction

Limitation of abduction



Physical Exam
Late Signs

Apparent short leg - Galeazzi sign

- asymmetrical thigh folds

Bottom Line

Detect unstable hip (Barlow, Ortolani)

Refer to pediatric orthopedic surgeon

Bottom Line

Hip click – stable exam

Re-examine at 6 weeks of age

-ve click -ve click

Hip u/s Fired

+ve -ve

Refer to Pediatric Orthopedic Surgeon Fired

Grey Area
6 weeks to 3 – 4 months

- Too late to detect reducibility (absent Ortolani, Barlow)
- Too early to detect late physical signs (decreased abduction, LLD)

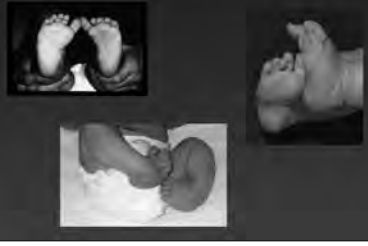
Bottom Line
Grey Area

- If exam does not "feel right"
- If 2 or more "**F**'s" present

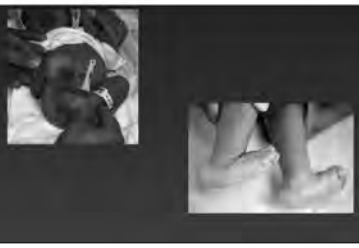
Send for hip ultrasound

Foot Deformities

You are called to the nursery…



You are called to the nursery…

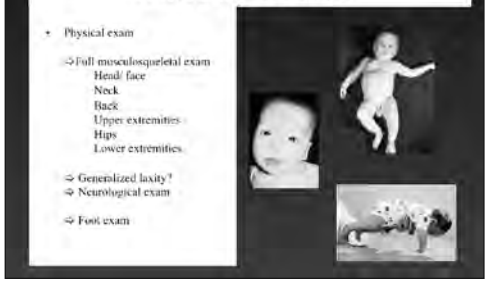


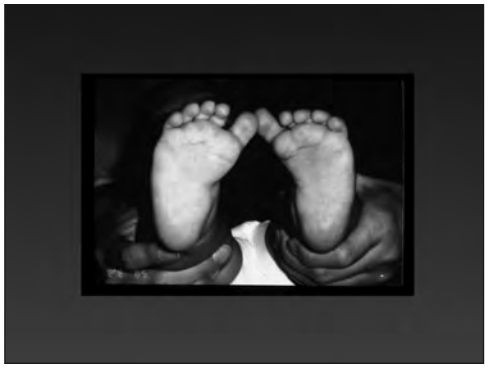
Let's talk the same language!

- Postural deformity
- Congenital malformation

Where do we start?

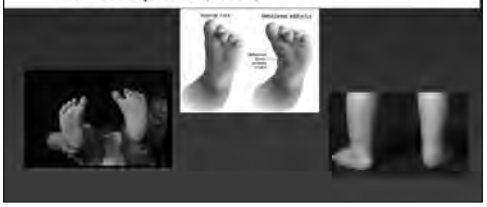
- Physical exam
  - ⇒ Full musculosquelelal exam
    - Head/ face
    - Neck
    - Back
    - Upper extremities
    - Hips
    - Lower extremities
  - ⇒ Generalized laxity?
  - ⇒ Neurological exam
  - ⇒ Foot exam





Metatarsus adductus

- Most common foot deformity in the newborn period (3.1%)

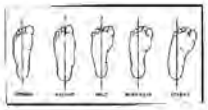


Metatarsus adductus

- Often bilateral (50%)

Metatarsus adductus

- Classification



- Natural history is benign: spontaneous resolution ≥85%

Metatarsus adductus treatment

- Mild deformity:

observe

If no correction ≥ 3 months

Derotation Bar= NO!

Congenital clubfoot

- Rigid deformityof the hindfoot (equinus and varus) and of the forefoot (adductus, supinatus and cavus)

Congenital clubfoot

- Incidence: 1-2/1000
- Familial predisposition
- More frequent in boys (2:1)
- Bilateral: 50%

Congenital clubfoot

- Etiology unknown, multifactorial

- Idiopathic
- Associated with a malformation syndrome (teratologic)
- neuromuscular

Congenital clubfoot

- No spontaneous correction

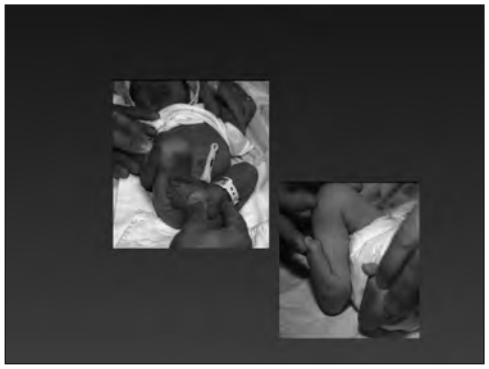
- Early reference to the orthopaedic surgeon!

Congenital clubfoot treatment

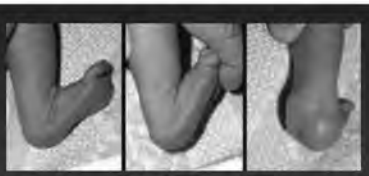
- Goals: functional, non painful, flexible plantigrade foot
- Serial castings

Congenital clubfoot treatment

- Percutaneous tenotomy at ~3 months
- Casts (2 to 4 weeks)
- Braces or derotation bar



Talipes calcaneovalgus



Talipes calcaneovalgus

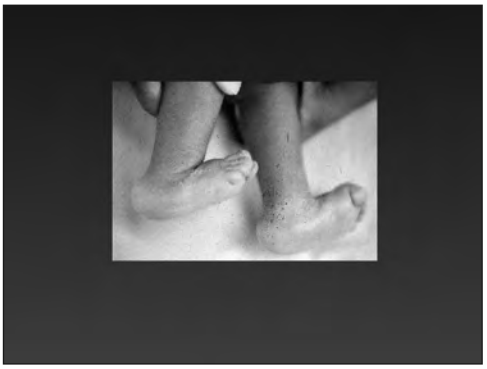
- Very frequent postural deformity (1-2 /100)
- Familial predisposition
- More frequent in girls, and first born babies

Talipes calcaneovalgus

- Differential diagnosis

Talipes calcaneovalgus

- Spontaneous resolution in most cases in 3 to 6 months: no treatment usually required
- If no correction or if very rigid, look for a cause! (neurologic, vertical talus)



Vertical talus

- Rigid deformity of the hindfoot (equinus/valgus) and of the forefoot (dorsiflexion/abduction) : rockerbottom aspect of the foot

Vertical talus

- Idiopathic; rare
- Neurogenic: 60% (arthrogryposis, spinal dysraphism)

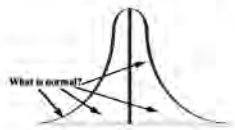
Vertical talus

- Early orthopaedic intervention required
  - ⇨Serial castings in plantar flexion
  - ⇨Surgical correction
- Prognosis?

Angular and Torsional Deformities of the Lower Extremities

Rule of the Universe

- **Life is a bell curve**



Charlie's Corollary

- Severity of deformity in child is inversely proportional to the number of grandparents in the room

Intoeing "Pigeon Toe"

- Most common complaint seen in pediatric orthopedic office
- Most, if not all, do not need to be seen by pedipod

Intoeing

(i) Hip

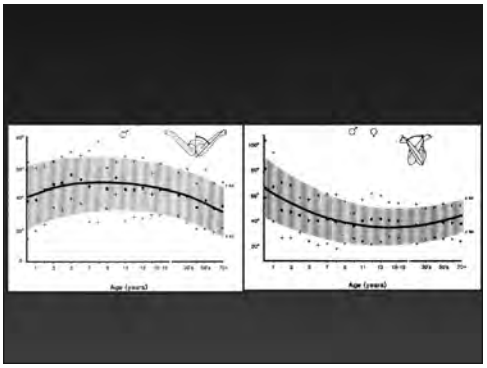
(ii) Tibia

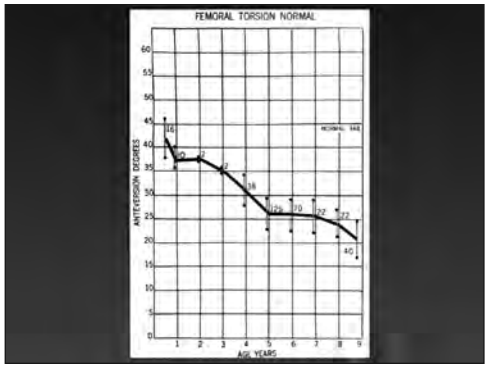
(iii) Foot

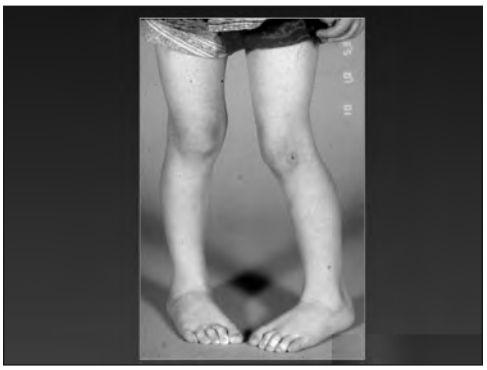
or combination

Femoral Anteversion

- ↑ Hip internal rotation
- ↓ Hip external rotation
- Familial
- Female
- Age: ~ 3 - 10







Femoral Anteversion Treatment

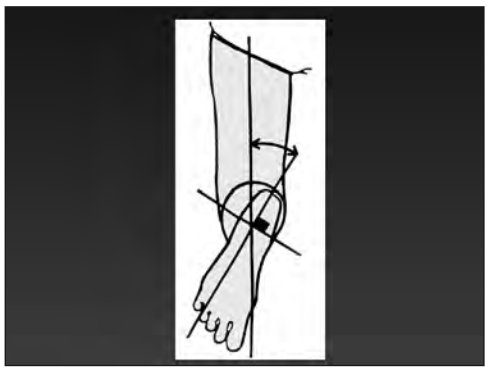
- Mother Nature!!
- Most cases of femoral anteversion will remodel by age 10 unless mom and dad still have it

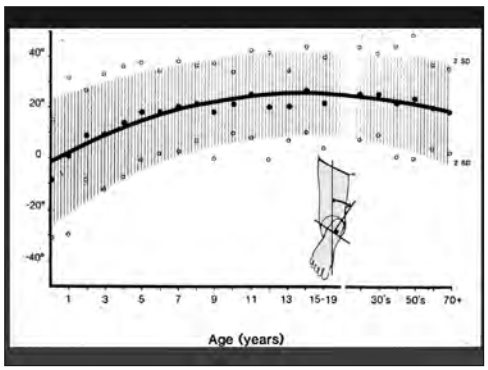
Femoral Anteversion

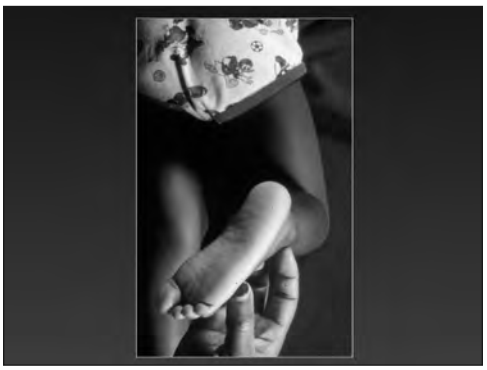
- Cosmetic concern only
- No functional implications in later life!!!

Internal Tibial Torsion

Most common cause of intoeing
< 3 years of age

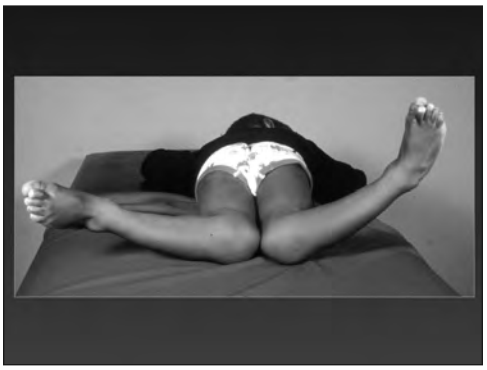






Internal Tibial Torsion

- Usually symmetric
- Most cases will remodel by age 4
- May be associated with femoral anteversion



Internal Tibial Torsion

- Treatment
- Mother nature
- Cosmetic concern
- No functional concerns

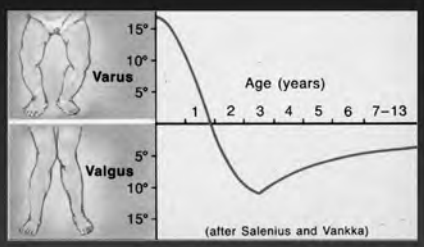
Angular Deformities in Children

- Very common reason for consultation
- Can be of great concern to parents
- Usually physiological, needs no treatment
- But… do not miss pathological causes

Angular Deformities

- Physiological
  - Genu varum
  - Genu valgum
- Pathological
  - Congenital (e.g. absence of fibula)
  - Post-traumatic
  - Post-infectious
  - Skeletal Dysplasia
  - Metabolic (O.I, Rickets)
  - Others (Blount)

How to Differentiate Physiological from Pathological Angulation in Children?



Approach to a Child with Angular Deformity

Approach to a Child with Angular Deformity

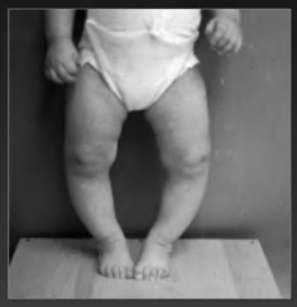
- Family history
- History of present condition
  - Onset of deformity: birth? Before or after walking age?
  - Progression
  - Any associated symptoms?
- Physical examination:
  - General (features of sk. dyspl.)
  - Gait, standing, table top examination

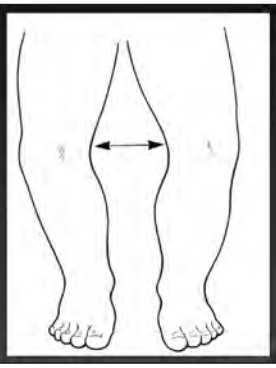
Clinical Evaluation

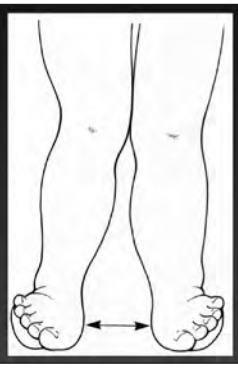
- No evidence of pathological bone disorder
- Age of the child
  - Genu Varum = 1 – 3 years
  - Genu Valgum = 3 – 7 years

Therefore, it is physiological – you do not need to refer the patient

- Follow-up appointment
- Clinical photographs







When should you refer a child with angular deformities?

- Deformities falling outside the age for physiological genu varum and valgum
- Unilateral
- Asymetrical
- Severe
- Progressive
- Any suspicion of pathological disorder

When should you refer a child with angular deformities?

- Deformities falling outside the age for physiological genu varum and valgum

When should you refer a child with angular deformities?

- Unilateral

When should you refer a child with angular deformities?

- Asymetrical

When should you refer a child with angular deformities?

- Severe

When should you refer a child with angular deformities?

- Progressive

When should you refer a child with angular deformities?

- Any suspicion of pathological disorder

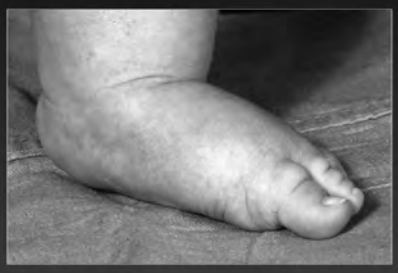
When should you refer a child with angular deformities?

- Deformities falling outside the age for physiological genu varum and valgum
- Unilateral
- Asymetrical
- Severe
- Progressive
- Any suspicion of pathological disorder

The Flat Foot: A Myth or a Problem?

Flat Feet

- Abnormally low longitudinal arch due to ligament laxity



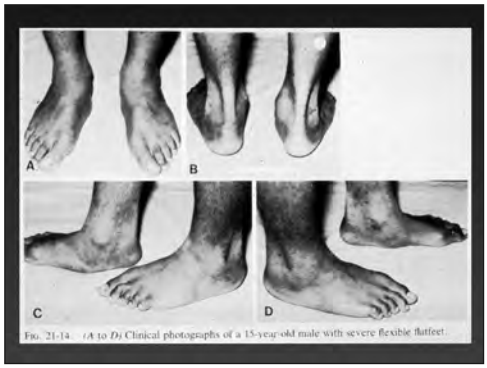
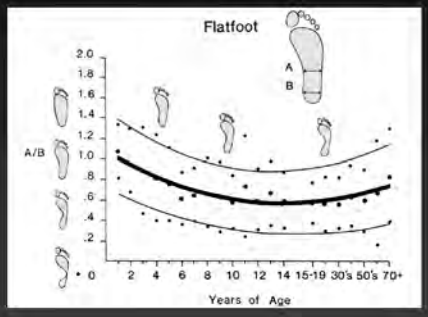


FIG. 21-14. *A* to *D*, Clinical photographs of a 15-year-old male with severe flexible flatfeet.

Flat Feet

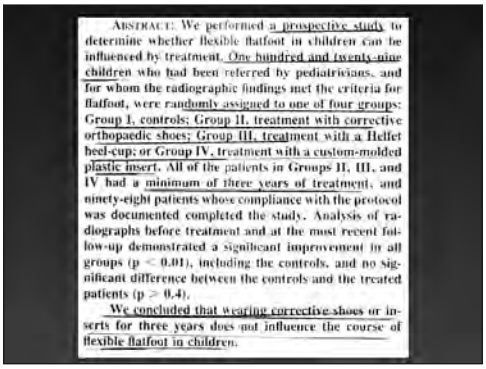
- Most always asymptomatic
- No correlation to back pain
- Major source of concern to parents



Corrective Shoes and Inserts as Treatment for Flexible Flatfoot in Infants and Children*

BY DENNIS R. WENGER, M.D.†, SAN DIEGO, DONALD MAULDIN, M.D.‡, GAIL SPECK, M.D.‡, DEAN MORGAN, C.PED.‡, DALLAS, TEXAS, AND RICHARD L. LIEBER, PH.D.†, SAN DIEGO, CALIFORNIA

From the Texas Scottish Rite Hospital, Dallas, and the Division of Orthopaedics, University of California at San Diego, San Diego



ABSTRACT: We performed a prospective study to determine whether flexible flatfoot in children can be influenced by treatment. One hundred and twenty-nine children who had been referred by pediatricians, and for whom the radiographic findings met the criteria for flatfoot, were randomly assigned to one of four groups: Group I, controls; Group II, treatment with corrective orthopaedic shoes; Group III, treatment with a Helfet heel-cup; or Group IV, treatment with a custom-molded plastic insert. All of the patients in Groups II, III, and IV had a minimum of three years of treatment, and ninety-eight patients whose compliance with the protocol was documented completed the study. Analysis of radiographs before treatment and at the most recent follow-up demonstrated a significant improvement in all groups ($p < 0.01$), including the controls, and no significant difference between the controls and the treated patients ($p > 0.4$).

We concluded that wearing corrective shoes or inserts for three years does not influence the course of flexible flatfoot in children.

Treatment

- Only if symptomatic (rare) - almost never < 13 years
- Molded arch supports

Toe Walking

- Persistent tiptoe gait after 2 y.o. without discernable neuro or orthopaedic abnormality :

  « Idiopathic Toe Walking »

- DDx:
  - Cerebral palsy
  - Muscular dystrophies
  - Tethered cord syndrome
  - Diastematomyelia
  - Other neuromuscular diseases

- Dx is made after other diseases have been excluded.
- Typically,there is a tiptoe gait at the initiation of walking which occurs at a N age

Hx: - Perinatal Hx, Development
- Family Hx
- R/O recent onset
- % of time on tip toes

P/E: Neuro: sensory,motor,reflexes
Gower sign, clonus, spasticity
Ortho: LLD, gait, ROM, spine, squat test

- Ankle DF to be assessed with knee in EXT.

DF= -20 degres

DF= 0 degres

- Treatment:
  Any ANOMALY on exam → REFER
- If left untreated, will persist or worsen
- Modalities Physio: Stretching
  Braces
  Serial casts ± Botox
  Surgery

Thank you!

Notes

Monday, Nov. 23 – Workshop B-04

14:30 - 15:30 HANDS ON Shoulder Exam

J. Scott Delaney MDCM, FRCP(C), FACEP
Research Director, Department of Emergency Medicine, MUHC;
Team Physician, Montreal Alouettes and Impact

Research Interests: Dr. J. Scott Delaney practices emergency medicine and sport medicine at McGill University in Montreal, Quebec. He has a fellowship in sport medicine and is the research director for the McGill University Health Centre Adult Emergency Department. He is an associate professor at McGill University and is a team physician for the Montreal Alouettes, Montreal Impact, McGill Football, McGill Men's and Women's Soccer teams and Cirque du Soleil. He is a member of the editorial board for the Clinical Journal of Sport Medicine and his research interests include concussions and neck injuries in both the athletic and emergency department populations.

Learning Objectives

1) Gain an overall approach to examining the injured shoulder
2) Become familiar with the diagnosis of common shoulder pathologies including injuries to the rotator cuff/ acromioclavicular joint/ labrum and shoulder instability

Today's workshop will deal mainly with the painful shoulder and will concentrate on the physical examination. The physical examination leading to the diagnosis of shoulder tendinitis, rotator cuff tears, labral cuff tears, and impingement syndromes will be emphasized. Due to time constraints, the diagnosis of the various fractures and dislocations around the shoulder will not be covered today.

Overview

1) Anatomy of Shoulder Joint

3 joints : sternoclavicular
1 articulation : scapulothoracic

acromioclavicular

glenohumeral

Muscles around shoulder : see ROM

Bursa : subacromial

2) History

age

occupation / recreational activities

location of pain (+/- radiation)

onset of pain : acute or chronic

duration

aggravating / alleviating factors

associated symptoms

other medical history

3) Physical Examination

Note : - always include an exam of the cervical spine!!

- disrobe (women place bra in halter or tube top style)

A- ***Inspection*** : from the front and back of the patient

- asymmetry (compare to other side)
- gross deformities
- wasting / atrophy of musculature
- abnormal movement

B- Palpation : may do from the front and / or back of the patient

- bones
- joints / articulations
- muscles / tendons / bursa
- axilla

C- Range of Motion

Note : - do active range of motion first

- only need to do passive range of motion if there is limitation with active
- usually check both sides at once
- not so important to remember numbers : compare to unaffected side

- abduction = 180o
  - need to externally rotate humerus after 120o
  - after first 20o : 2o of glenohumeral motion for every 1o of scapulothoracic motion
- adduction = 45o
- flexion = 180o
- extension = 60o
- internal rotation = 70o
- external rotation = 80o
- combinations :

i- Reach behind back and touch opposite scapula (" undoing your bra") = internal rotation and adduction

ii- Reach behind head and touch upper part of opposite scapula (Apley Scratch test) = external rotation and abduction

D- Strength Testing

- abduction : medial deltoid supraspinatus : especially first 15o of motion
- adduction : pec major
  lat dorsi
- flexion : anterior deltoid
  coracobrachialis
  pec major
- extension : lat dorsi
  teres major
  posterior deltoid
- internal rotation : subscapularis
  pec major
  lat dorsi
  teres major
- external rotation : infraspinatus
  teres minor
- scapular elevation : trapezius
  levator scapulae
- scapular retraction : rhomboid major and minor
- scapular protraction : serratus anterior

(remember scapular winging when serratus anterior is weak)

-***Rotator Cuff Testing***

i) Supraspinatus (Abduction)

- abduct shoulder to 90o at side, internally rotate with thumbs pointing down to the ground and move arm 30o forward from coronal plane (emptying a can position) : now abduct against resistance

ii) Infraspinatus and Teres Minor (External Rotation)

- elbows flexed to 90o at side with thumbs pointing up : now externally rotate against resistance

iii) Subscapularis (Internal Rotation)

- arm behind back with hand around belt level (as if starting to undo bra) and palm facing backwards towards examiner: now push palm back against resistance

- Abdominal compression test

Note: Dynamic testing of the rotator cuff or lag signs of the rotator cuff muscles are also useful examination tools for assessing rotator cuff tears. A) The lower fibers of the infraspinatous and teres minor are tested by abducting the patient's arm and maximally externally rotating. The patient is asked to keep the arm in this position. A positive test is if the patient is unable to keep the arm in this position and the arm springs back. This is also known as the positive horn-blower's sign.

B) Supraspinatous and infraspinatous insufficiency is tested by maximally externally rotating the arm at the side and asking the patient to keep the arm in this position. If the patient is unable to keep the arm in this position, the test is considered positive.

iv) Long head Bicep tendinitis:

I- *The Yergenson's test* is performed with the patient's elbow flexed to 90 degrees and stabilized against the thorax with the forearm pronated. The examiner resists supination while the patient also laterally rotates the arm against resistance. A positive result elicits tenderness in the bicipital groove and is indicative of bicipital tendinitis.

II- *The Speed's test* is a more effective way of eliciting bicipital tendinitis. The examiner resists shoulder forward flexion by placing his hand on the patient's supinated forearm while the patient is elevating the arm and keeping the elbow extended. For a more effective method of pain reproduction, the examiner places fingers on the bicipital groove while performing this maneuver.

E- Neurologic Testing

- motor testing done as above

- sensory : pinprick to area

i- lateral arm = C5 (axillary nerve - important in anterior shoulder dislocation / subluxations)

ii- thumb and index finger = C6

iii- long finger = C7

iv- ring and index finger = C8

v- medial forearm = T1

vi- axilla = T2

vii- nipple = T4

F- Special Tests

i- Acromioclavicular Joint Separation

I- Scarf Test : Forward flex the arm to 90o, internally rotate the arm so the palm is facing the ground, and adduct the arm across the chest at shoulder height. Pain in the AC area indicates a positive test.

ii- Impingement Tests

- These tests try to pinch a tendon (usually the supraspinatus) under the coracoacromial complex . This is accomplished by placing the greater tuberosity underneath the coracoacromial ligament by internally rotating the arm. The greater tuberosity will then pinch the tendon against the coracoacromial ligament causing pain.

I- Hawkins Test : Forward flex the arm to 90o, flex elbow to 90o, and forcibly internally rotate the arm.

II- Neer Test : Internal rotation of the arm so palm is facing the ground and forward flex the arm passively or actively

iii- Instability

- There are several types of instability.

- Anterior instability is classic with the humeral head subluxing out of the glenoid usually when the arm is abducted, externally rotated, and extended.

- Posterior instability refers to the glenoid subluxing posteriorly out of the glenoid. This is less common than anterior instability.

- Multidirectional instability refers to a lax shoulder joint (usually bilateral) that may sublux or dislocate in any direction.

- Grades of Instability:

- Grade 1 instability denotes translation up to the labrum without an ability to dislocate.

- Grade II instability is the ability of the examiner to dislocate the shoulder while a spontaneous reduction occurs at the time of the maneuver.

- Grade III instability is the dislocation of the shoulder the requires a subsequent reduction by the examiner.

I- Sulcus Sign : Patient stands with arms at side and muscles relaxed. Examiner pulls down on forearms. The presence of a sulcus lateral and inferior to the acromion indicates inferior instability. If the patient demonstrates inferior instability, he/she has multidirectional instability.

II- Load and Shift Maneuver : Examiner stands behind patient. One hand stabilizes the scapula at the acromion. The other hand loads the humeral head into the glenoid and then pushes the humeral head posterior to gauge the posterior laxity or excursion. The humeral head may start to ride over the rim of the glenoid if laxity is present. The humeral head is then pushed anteriorly to gauge the anterior laxity or excursion Compare with the other side.

III- The Jerk test is used to assess posterior instability. It can be performed with the patient sitting or standing. The arm is forward flexed to 90 degrees and internally rotated approximately 90 degrees. The examiner grasps the elbow and axially loads the humerus. While maintaining this load, the examiner adducts the arm across the body. A positive test occurs when the examiner feels a sudden jerk occurs as the head slides posteriorly. A second jerk can be felt as the arm is returned to a 90 degree abduction posttion. This is an indication of the shoulder reducing back into joint.

III- Apprehension (Crank) Test and Relocation Test : The patient lies supine on the edge of the bed. The examiner abducts and externally rotates the arm slowly. This will tend to push the humeral head out of the glenoid anteriorly in patients with anterior instability. A positive Apprehension or Crank Test is indicated by a look of apprehension or fear on the patient's face. The patient may state that the sensation is similar to a previous dislocation. If a posterior force is now applied to the proximal humerus so as to push the humerus back into its proper position in the glenoid, the patient may lose the apprehension and feel less anxious. This is termed a positive Relocation Test.

iv- Labral Tear tests

I- Modified Labral "Clunk" Test : This test is akin to the McMurray's test of the knee. The examiner stands behind the patient. One hand rests posteriorly on the humeral head. The other holds the arm at the elbow. The arm is abducted to different degrees while the humeral head is pushed anteriorly. The other hand loads the humeral head into the glenoid and externally rotates the arm. A painful clunk is a positive test and is indicative of a labral cuff tear. This test may also cause apprehension in a patient with anterior instability.

II- *The Obrien's tes*t is performed by placing the arm patient's arm in cross chest adduction (horizontal flexion) of the affected shoulder with the elbow extended and forearm pronated (thumb down toward the ground). A resisted downward movement is applied to the arm by the examiner. A positive maneuver produces either apprehension, pain referable to the bicipital groove, and an audible or palpable click. The test is repeated with the forearm supinated, which must cause diminution of the pain. Mechanically, elbow extension and forearm pronation places traction on the long head biceps tendon. When anterior scapular protraction is limited by the clavicle, further adduction entraps the unstable biceps tendon and superior glenoid labrum between the glenoid fossa and head thus causing the pain produced by this test

III- *The SLAP test* is performed with the arm held in a 90 degree abducted position with the forearm fully supinated. The examiner places one hand on the shoulder with the thumb in the axilla. The opposite hand is used to exert a downward force on the outstretched hand of the patient, this creates a fulcrum of the thumb, shifting the humeral head in a superior direction. This maneuver is positive if crepitation or pain is produced.

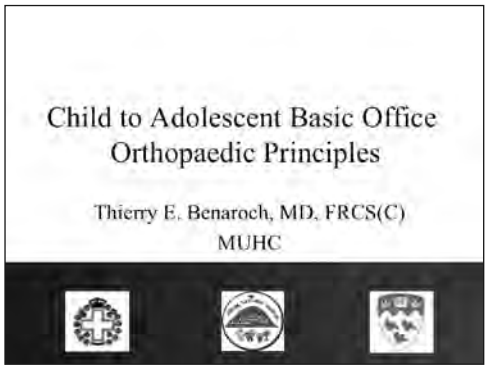
14:30 - 15:30 Contraception - Practical Approach

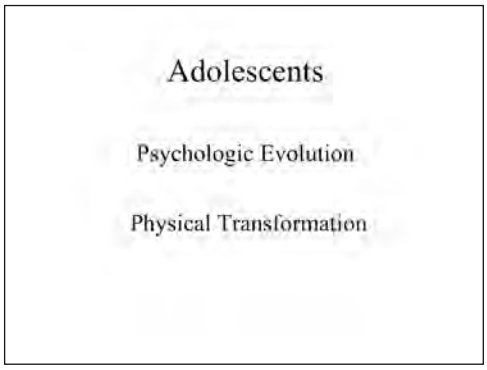
Cleve Ziegler MD, FRCSC, CSPQ

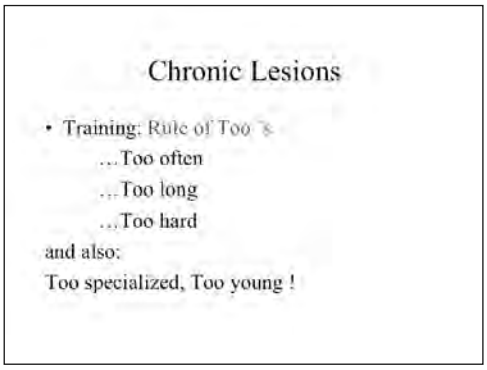
Assistant Professor, Department of Ob/Gyn, McGill University
Attending Physician, Department of Ob/Gyn, SMBD–Jewish General Hospital

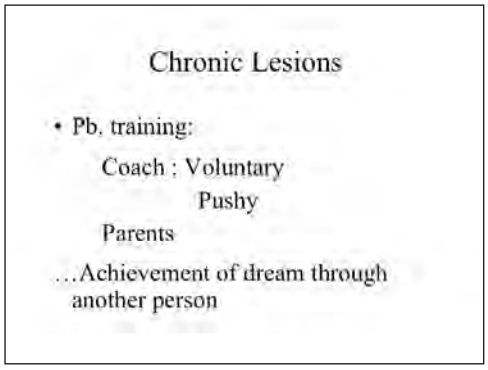
Research interests: Dr. Ziegler is a member of the department of Obstetrics and Gynecology at the Jewish General Hospital since 1994 and an Assistant Professor in the department of Obstetrics and Gynecology at McGill University.

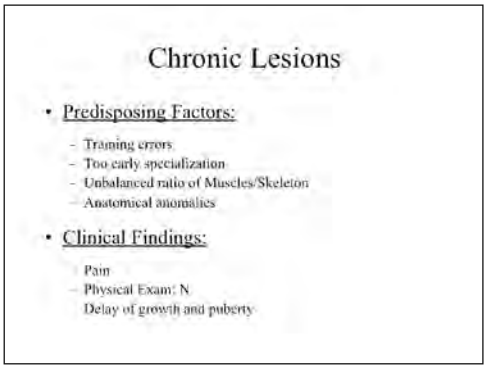
His interests include contraception, colposcopy and gynecologic surgery, and CME.





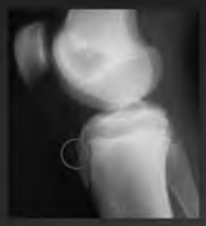






Chronic Lesions

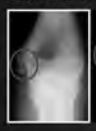
- Apophysitis:
  - Osgood-Schlatter
  - Sever
  - Tibial post.
  - Iselins

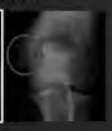


Chronic Lesions

« Kissing spine »

Pitcher's Elbow



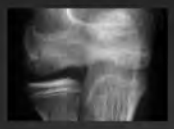


Chronic Lesions : Osteochondritis dissecans

- Treat the patient, not the X-Rays
- Check the other side

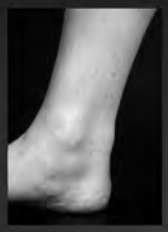
Other lesions

Osteonecrosis



Tendinitis

Very rare before puberty !



Physiological Differences 3

- Growth
  - Increase in length of bones
    - Muscle Stretching

ADOLESCENTS ARE NOT FLEXIBLE !!

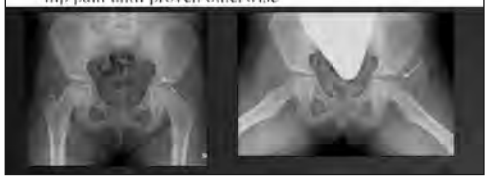
Adolescent Knee Pain

Any flags?

Anterior Knee Pain

- Common site of complaint
- Acute trauma
- Repetitive minor trauma

Knee pain in skeletally immature patient – referred hip pain until proven otherwise



Anterior Knee Pain

HISTORY:

- Poorly localised
- Usually bilateral
- Grab sign
- associated with prolonged sitting, stairs, + theater sign
- Pseudolocking

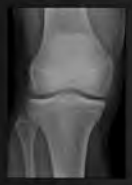
Anterior Knee Pain

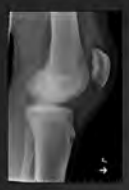
EX:

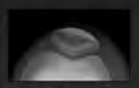
- Gait, ROM hips, knees, ankles, feet, knee stability, patellar tracking, meniscal tests, tenderness patella, patellar tendon, joint lines
- Atrophy, strength
- Quads/hamstrings flexibility

Anterior Knee Pain

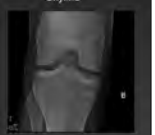
- X-rays: 4 Views







Skyline



A/P Lat Tunnel

Anterior Knee Pain

- Once other sources of anterior knee pain are R/O, 80% will respond to nonsurgical treatment

Physio: quad, hamstrings, flexibility/strength

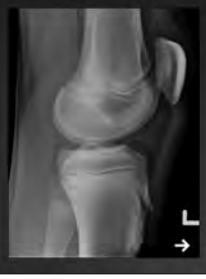
Knee brace

Adolescent anterior knee pain is a mythical disease,equivalent to a headache of the knee.

Surgical intervention has no more rationale than skull burr holes for headache tx.

Anterior Knee Pain

- Osgood-Schlatter



14:30 - 15:30 Exercise Prescription

Ivan Rohan MD, CCFP

Assistant Professor, Director of CME Division
Family Medicine, McGill University

IVAN ROHAN M.D., CCFP

Assistant Professor, Director of CME Division

Family Medicine, McGill University

Family Physician, graduate of Family Medicine Program at McGill, Involved in **teaching** and **CME** . Course Director for the Refresher Course for Family Physicians for over ten years and collaborated on many CME projects at the McGill Faculty of Medicine.

Research: in Diabetes, Sport Medicine and Public health(influenza)

Geriatrics: Treating physician of CHSLD Father Dowd and President of the Council of physicians of CHSLD St. Andrew, St.Margaret and Father Dowd.

Sport's Medicine giving lectures at Exercise Science, Concordia University, workshops on exercise at different conferences.

Teaching: St.Mary's Hospital Family Medicine

"Anyone who sits around idle and takes no exercise will be subject to physical discomfort and failing strength."

Ivan Rohan M.D., CCFP
McGill University
Montréal

EXERCISE -Prescription
How can family doctor improve compliance

Educational goal:
To familiarize the participants with:
The benefits, risks, recommendations, guidelines and monitoring of exercise.

Look at the strategies to improve the compliance with exercise.

To review some specific conditions:
Cardiac, diabetic, elderly, women, children.

EXERCISE -PRO'S AND CON'S
How can family doctor improve compliance

- BENEFITS OF EXERCISE
- Risks of exercise
- Recommendation – Guidelines type, frequency, intensity
- Monitoring of exercise
- Specific conditions – Cardiac, Diabetes, Elderly, Women, Children
- Compliance issues

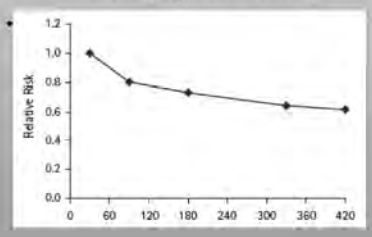
BENEFITS OF EXERCISE

- All-cause morbidity and mortality
- Cardiovascular health
- Obesity
- Diabetes mellitus
- Psychological
- Cognition
- Other - cancer prevention…

Health benefits

- 60 min/week - benefits start
- 150 min/week - most benefits
- 300 min/week - more benefits

All cause mortality declines with more physical activity



CARDIOVASCULAR BENEFITS

- All CV disease incidence and mortality
- Coronary heart disease
- Multiple metabolic risk factors
- Lipids
- Hypertension
- Stroke

Psychological benefits

- Depression
- Anxiety, stress
- Well-being, self image
- Adjunct in alcohol and substance abuse

Complications and risks of exercise

BENEFITS FAR OUTWEIGH THE RISKS

Complications and risks of exercise

- Injuries
- Overuse syndromes
- Exhaustion, heat stroke, dehydration
- Hypoglycemia in diabetics
- Myocardial infarction irregular vigorous exercise
- Sudden death - rare

Adverse Events

- **Moderate-intensity physical activity**, such as brisk walking, has a **low risk** of such **adverse events.**
- **The risk of musculoskeletal injury** increases with the total amount of physical activity. However, people who are physically active may have fewer injuries from other causes, such as motor vehicle collisions or work-related injuries.
- Participation in **contact or collision sports**, such as soccer or football, has a higher risk of injury than participation in non-contact physical activity, such as swimming or walking.
- **Cardiac events, such as a heart attack or sudden death** during physical activity, are **rare**. However, the risk of such cardiac events does increase when a person suddenly becomes much more active than usual. The greatest risk occurs when an adult who is usually inactive engages in **vigorous-intensity activity** (such as shovelling snow).

MI and exercise

MI risk during heavy exertion:

- 2.4x increase in regular exercisers
- 60 - 107x increase in irregular exercisers
- Higher risk in diabetics and the difference not fully accounted for the lack of regular exercise

Sudden death

- In young
- Middle age and older

1 death / 50,000 participants
1 death / 215,000 hours of competition
1 death / 396,000 exercise hours
1 cardiac arrest / 4,800,000 exercise hours

Prevention of cardiac events

- Screening is generally poor
- Teaching of cardiac symptoms typical and even less typical
- CAD patients should be encouraged to exercise
- But they should avoid vigorous exercise

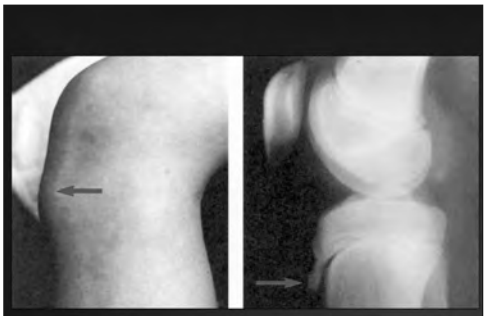
Recommendations, Guidelines for Exercise

- TYPE
- FREQUENCY
- INTENSITY

3 Main kinds of exercise

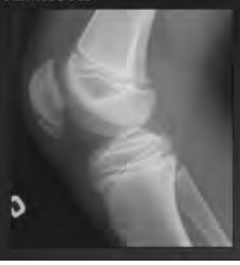
- Aerobic
- Muscle strengthening
- Bone strengthening

- 2 other activity – Balance, Stretching



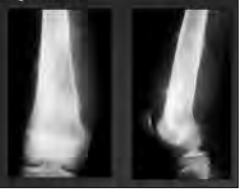
Anterior Knee Pain

- Sinding-Larsen-Johansson



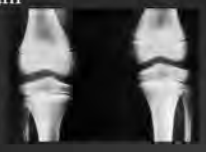
Red Flags

- Hx: Unilateral knee pain
  Swelling, locking, giving way
  No improvement post tx



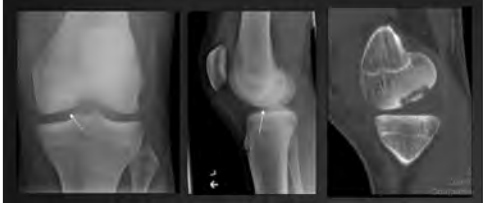
Red Flags

P/E: Swelling
Atrophy
Pain: fem.condyles , joint lines
Abnormal Lig.exam
+ Mc Murray



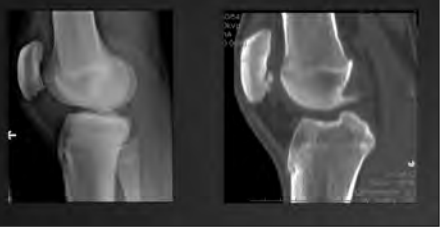
Red Flags

Osteochondritis Dissecans: Femoral Condyle



Red Flags

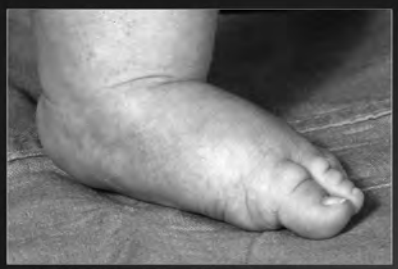
- Osteochondritis Dissecans:Patella

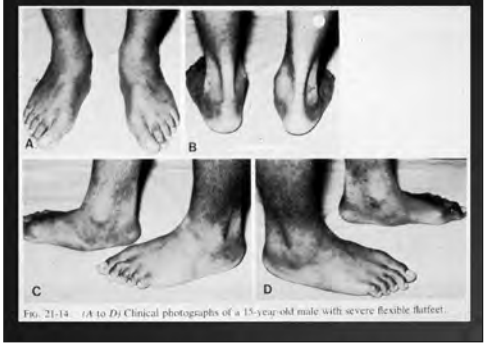


The Flat Foot: A Myth or a Problem?

Flat Feet

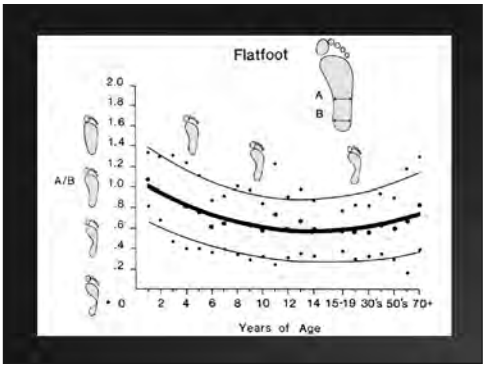
- Abnormally low longitudinal arch due to ligament laxity

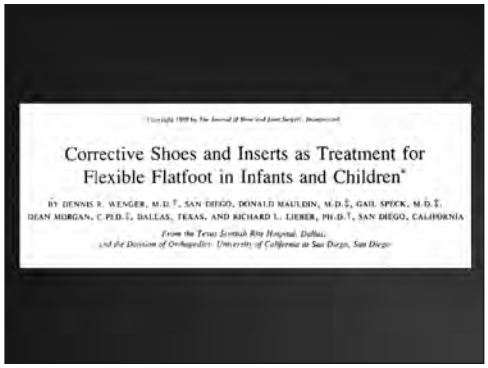




Flat Feet

- Most always asymptomatic
- No correlation to back pain
- Major source of concern to parents

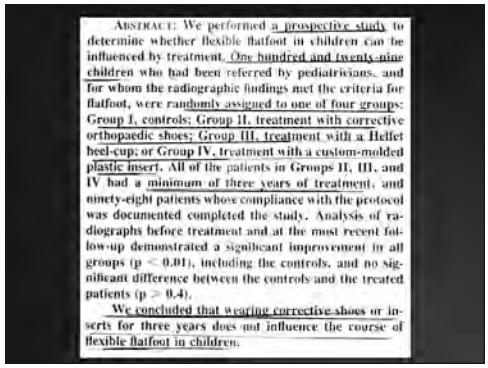




Corrective Shoes and Inserts as Treatment for Flexible Flatfoot in Infants and Children*

BY DENNIS R. WENGER, M.D.†, SAN DIEGO, DONALD MAULDIN, M.D.‡, GAIL SPECK, M.D.‡, DEAN MORGAN, C.P.D.T., DALLAS, TEXAS, AND RICHARD L. LIEBER, PH.D.†, SAN DIEGO, CALIFORNIA

From the Texas Scottish Rite Hospital, Dallas, and the Division of Orthopedics, University of California at San Diego, San Diego



ABSTRACT: We performed a prospective study to determine whether flexible flatfoot in children can be influenced by treatment. One hundred and twenty-nine children who had been referred by pediatricians, and for whom the radiographic findings met the criteria for flatfoot, were randomly assigned to one of four groups: Group I, controls; Group II, treatment with corrective orthopaedic shoes; Group III, treatment with a Helfet heel-cup; or Group IV, treatment with a custom-molded plastic insert. All of the patients in Groups II, III, and IV had a minimum of three years of treatment, and ninety-eight patients whose compliance with the protocol was documented completed the study. Analysis of radiographs before treatment and at the most recent follow-up demonstrated a significant improvement in all groups ($p < 0.01$), including the controls, and no significant difference between the controls and the treated patients ($p > 0.4$).

We concluded that wearing corrective shoes or inserts for three years does not influence the course of flexible flatfoot in children.

Treatment

- Only if symptomatic (rare) - almost never < 13 years
- Molded arch supports

Tarsal Coalition

- Congenital abnormality with a varying degree of union between 2 or more tarsal bones producing a rigid flat foot

Symptoms

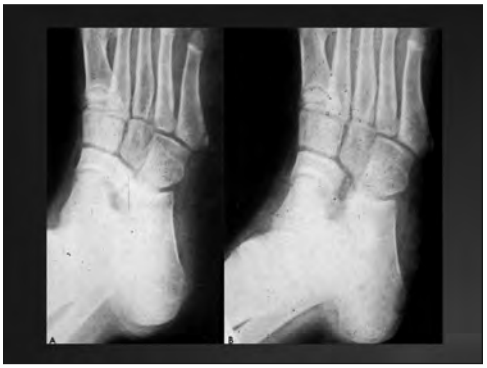
- Pain age 8 - 12 years
- Ossification of bar
- Decreased subtalar motion

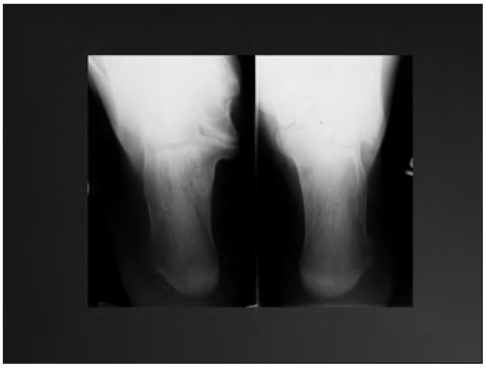
Most Common

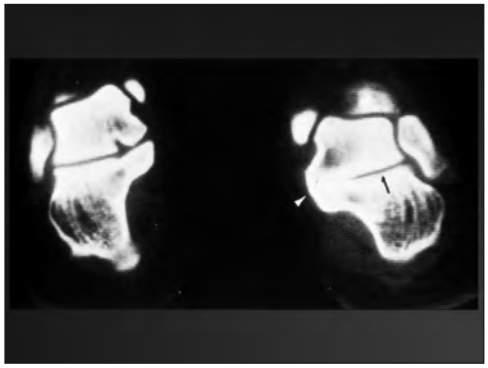
- Calcaneonavicular
- Talo calcaneal
- 50% bilateral

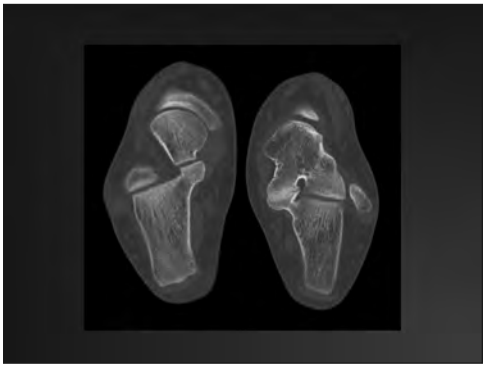
Radiographs

- 45 degree oblique
- Harris Axial Calcaneal View
- CT scan









LIMPING

The challenge is to find where and what is the problem with the smallest number of tests, and to determine if further observation or immediate referral is necessary.

You do not want to miss serious conditions such as septic hip.

Look for Five Presentations

- Pain
- Limb length discrepancy
- Stiffness and contractures (sp. Rheumatoid arthritis)
- Neuromuscular conditions
- Hysteria

EXERCISE LITERATURE

1. Dunn EJ, Blair SN, Physical Activity Behaviour Change (Review) 70 ref. Health Psychology 19 (suppl); 32-41, 2000 Jan
2. Lee IM, Paffenbarger RS Jr, Association of light moderate and vigorous intensity physical activity with longevity. The Harvard Alumni Health Study. American Journal of Epidemiology 151;(30):293-9,2000 Feb.
3. Mittleman MA et al. Triggering of acute myocardial infarction by heavy physical exertion-protection against triggering by regular exertion. N Eng J Med 1993;329:1677-83..
4. Thompson PD, The cardiovascular complications of vigorous physical activity. Review. Arch Int Med 1996;156(20):2297-2302...
5. PetrellaRJ, Lattanzio CN, Does counseling help patients get active? Can Fam Physician 2002;e48:72-80.
6. Pipe A, Get active about physical activity. Can Fam Physician;2002;48:13-14.
7. Bhaskarabhatia K, Birer R, Physical activity and Type 2 Diabetes. Phys Sportmed 2004;32:13-17.
8. Frere JA, Maharami lG, VanCamp SP. The risk of Death in running Races. Phys Sportmed 2004;32:33-40..
9. Weuve et al. Physical activity including walking and cognitive function in older women. JAMA 2004 Sep22;292(12):1454-61.
10. Lee IM, Oguma Y, Paffenbarger RS Jr, The "weekend warrior" and risk of mortality.
Am J Epidemiol. 2004 Oct 1;160(7):636-641.
11. Rohan I Benefits and Risks of Exercise in Elderly Patients. J CME 1994 Sep;6(9) 49-67.
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14. Herman WH, HoergerTJ, Brandle M,et al: The co-effectiveness of lifestyle modification or metformin in preventing type 2 diabetes in adults with impaired glucose tolerance. Ann Intern Med 2005;142(5):323-332.
15. Lee I-M., Sesso HD, Oguma Y et al: The weekend warrior and risk of mortality. Am J Epidemiol 2004:160(7):636-41.
16. Dennis Y. Wen: Pre-participation Cardiovascular Screening of Young Athletes. An Epidemiological perspective. Physician Sports Med 2005;33(12):31-42.
17. Weiss Kelly A, Practical Exercise Advise During Pregnancy Guidelines for active and inactive women. Phys Sportmed 2005: 33(6): 24-30.
18. Hou L, Ji B-T, Blair A, et al: Commuting physical activity and risk of colon cancer in Shanghai,China. Am J Epidemiology 2004;160(9):860-867

Useful WEB sites:

Health Canada, Santé Canada: www.hc-sc.gc.ca
Canadian Academy of Sports Medicine: www.casm-acsm.org
American College of Sports Medicine: www.acsm.org
US Department of Health and Human services http://www.health.gov/paguidelines

14:30 - 15:30 Avoiding Amputation in the Diabetic Patient

Philip Weech Ph.D.

Physiotherapist, Jewish Rehabilitation Hospital, Laval

Research interests:

1974 B.Sc. Biochemistry and Zoology, University of Wales Cardiff, United Kingdom

1977 Ph.D. Biochemistry, Middlesex Hospital Medical School, University of London, United Kingdom

1977-1996 Research scientist:

Oklahoma Medical Research Foundation, Oklahoma City, U.S.A.

Institut National de la Santé et de Recherche Médicale, Hôpital Henri Mondor, Créteil, Paris, France

Institut de recherches cliniques de Montréal, Montréal, Canada

Merck Frosst Centre for Therapeutic Research, Merck Frosst Canada Inc., Montréal, Canada.

Adjunct professor: Université de Montréal, Université du Québec à Trois Rivières, Université du Québec à Montréal.

60 publications on biochemistry of Plasma Lipoprotein structure and metabolism in relation to vascular disease; Phospholipase A2 and prostaglandins in relation to inflammatory mediators.

1999 B.Sc. Physical Therapy, McGill University, Montréal, Canada

1999-present Physiotherapist, Amputee rehabilitation program, Jewish Rehabilitation Hospital, Laval

Session lecturer on amputee rehabilitation, to physical and occupational therapy B.Sc. and M.Sc. courses, McGill University.

Current clinical interests are amputee rehabilitation, prevention and wound care, and research on the incidence of balance disorders associated with diabetes in amputees and their impact on mobility.

Monday, Nov. 23 – Workshop B-07

Objectives

- Participants will be able to make a quick, logical evaluation of the foot.
- Participants will understand the effects of diabetes that contribute to foot ulcers.
- Participants will be able to make a comprehensive care and referral plan for clients at risk of ulceration, with a goal that amputation of the leg is avoided.

Syllabus

Participants in this workshop will learn a quick (2-3 minute) protocol for evaluation of the feet.

The evaluation is structured on a disease model that integrates four major factors that together are responsible for injury and poor healing of diabetic foot ulcers.

A model of care emerges from the four-point model, that has been supported by clinical guidelines in several countries.

Clinical case studies of diabetic feet will be discussed with the workshop participants, to explore the model and its use in primary care.

Participants will discuss the simple options for foot care, protection, wound treatment, and referral. The evidence base will be presented.

The personal and health-care costs of amputation are so great that education and simple treatment should be given to diabetics as soon as a risk of foot disease is manifest.

Suggested readings and resources

American Diabetes Association: http://care.diabetesjournals.org/content/32/Supplement_1/S13.full.pdf+html

American Diabetes Association: Consensus Development Conference on Diabetic Foot Wound Care. Diabetes Care, Volume 22 (8): 1354-1360, 1999

Agbor Ndip, Jude E.B. Emerging Evidence for Neuroischemic Diabetic Foot Ulcers: Model of Care and How to Adapt Practice. Int. J. Low Extrem. Wounds, Volume 8 (2): 82-94, 2009

Basic Guidelines for Diabetes Care Packet (revised August 2009):

www.diabetescoalitionofcalifornia.org www.caldiabetes.org

Canadian Diabetes Association: www.diabetes.ca/about-diabetes /living/complications/foot-care

14:30 - 15:30 Hot Topics in Adolescent Care

Michael Malus MD, CCFP, FCFP

Chief, Department of Family Medicine, SMBD-Jewish General Hospital;
Director of the Herzl Family Practice McGill University Teaching Unit;
Associate Professor, Department of Family Medicine, McGill University

Research interests: Dr. Malus is the Chief of the Department of Family Medicine at the Jewish General Hospital and Director of the Herzl Family Practice McGill University Teaching Unit. He is also Director of the Centre's Teenage Health Unit. This is an outreach program featuring weekly visits for classroom discussions with teenagers in forty local high schools in Montreal with an accompanying offer for 24 hour care. He also has an interest in Native Health Care and has worked extensively with and in Native Communities in Canada and United States.

Hot Topics in Adolescent Health Care

Dr. Michael Malus, C.C.F.P., F.C.C.P
Chief, Department of Family Medicine
Director, Teenage Health Unit
Jewish General Hospital

Constantly Hot Topics

Relate to teenage morbidity & mortality

- MVA's
- Depression
- STD's

Newly hot

Strength-based interviewing

Classical approach

HEADS:

- still valid
- but risk oriented

Add elements of strength-based interviewing:

- affirming

Strength-Based Interviewing

- Conventional approach that emphasizes risks can result in adolescents becoming defensive and closing themselves off

Visit is an opportunity to increase their awareness of their developing strengths and motivate them to take responsibility for their own health and well being.

If already using a HEADS type of interviewing strategy you can simply add a few more questions in the various categories.

Questions involving:

Independence:

- Do you feel you are allowed to make more decisions as you grow older?
- Do you have a part time job?

Mastery:

- Is there something you enjoy doing and feel you do well?

- If you do know the adolescent over time be observant of any strengths and skills they have developed and comment positively on it.

" . . . I've noticed in the last year you've improved on . . . "

Questions involving:

Belonging

- to family
- to community

Generosity

Reference:

"Strength Based Interviewing" by Frankowski et al in Adolescent Medicine: State of the Art reviews" April 2009 published by the American Academy of Pediatrics.

Adolescent obesity

Definition: BMI >95th

Increased incidence

- In 1970 in a survey by the CDC's the incidence of obesity was 5% among children and adolescents aged 2 to 19 years.
- In 2006 same survey by the CDC's the figure has risen to 16.3%.
- 25% of children and adolescents are now overweight; this figure has doubled in the last ten years.

Consequences

DM adolescent & adult

- Presently 1/3 lifetime incidence for people born in 2000.
- Adult cardiovascular disease
  - study tracing age of MI vs weight.

Metabolic Syndrome in Children and Adolescents

- Increased incidence paralleling ↑ incidence of overweight and obesity
- Definition: 3 or more of 5 risk factors:
  1. Central obesity
  2. High blood pressure
  3. High triglyceride
  4. Low HDL
  5. Elevated fasting glucose levels

Metabolic Syndrome (cont'd)

- Key factor is insulin resistance causing ineffective function of insulin hormone at normal serum levels.
- Recent studies show that high fasting serum insulin was the best predictor of metabolic syndrome and subsequent cardiovascular disease in adulthood.

Prevention & treatment of obesity

Monitor weight vs height

- from birth to 2 years by height and weight percentiles
- from 2 years of age to late teenage with BMI from CDC charts.

BMI

- 85th – 95th percentile = overweight
- > 95th = obesity

Prevention

- Promote breast feeding
- 5210:
  - 5 fruits and vegetables per day
  - nor more than 2 hours of sedentary activity apart from homework
  - 1 hour of exercise
  - 0 sugar-containing drinks

Eat Breakfast

- eat breakfast
- eat supper together
- ↓ fast food meals
- family approach

Laboratory Investigation

>95 BMI

- Fasting glucose
- Lipids
- LFTS

>95 BMI

Keep in mind:

- Sleep apnea
- Polycystic Ovarian Syndrome
- Non-alchoholic Fatty Liver Disease

Drug RX

- Orlistat: diarrhea
- Meridia: increased BP

Bariatric Surgery

Treatment

- Intensification of the preventive measures.
- Growth spurt working for the overweight teenager:

  - Just don't gain.

Management of the Adolescent Concussion Victim

Incidence:

- 20% of high school football players have concussion if they play 4 years
- 40% of college football players

Possible consequences of too early return to sport

- cognitive deterioration
- post concussion syndrome: psychological instability, anxiety, depression
- chronic headache

Danger of too early return to sporting activity (cont'd)

- Fewer than 50% of concussion victims recover within 1 week
- 60% within 2 weeks
- 80% by 3 weeks

i.e. no sports with a risk of injury for 1 month

Silent epidemic

Underdiagnosed:

- 1 study (2003) reported in the Clinical Journal of Sports Medicine only finds concussion diagnosed 20% of the time when it occurs
- Loss of consciousness in only 8%-20%
- Enough to be confused, dazed, moving clumsily, etc.

Second impact syndrome

- Even trivial injury after a recent concussion can lead to permanent brain damage or death

Primary Immunodeficiency Diseases (PIDDS)

- Incidence: 1 in 1200; higher than cystic fibrosis
- Most severe cases apparent in infancy.
- Even those cases discovered later in life can have significant morbidity and mortality.

PIDDS (cont'd)

- Watch for repeated pulmonary and sinus infections, frequent hospitalizations for infections
- Most frequent is Common Variable Immunodeficiency (CVID)
- 20% of these patients have other autoimmune diseases

eg:
- Immune Thrombocytopenia (ITP)
- Autoimmune Hepatitis
- Vasculitis

PIDDS (cont'd)

- Higher incidence of lymphoma than the general population.

DX: Serum protein electrophoresis
RX: I.V. immunoglobulin monthly

Notes

Monday, Nov. 23 – Workshop C-01

16:00 - 17:00 ER: ER Procedures (repeat of B-01)

H. Mitchell Shulman MDCM, FRCPC, CSPQ

Assistant Professor, Department of Surgery, McGill University;
Associate Professor, Family Medicine, St. Mary's Hospital Centre;
Attending Physician, Emergency Room, Royal Victoria Hospital

(see pages 87 to 89 for the handout)

Notes

Monday, Nov. 23 – Workshop C-02

16:00 - 17:00 GER: Delirium Evaluation

Robert Bailey MD, FRCP

Director, Division of Geriatrics, St. Mary's Hospital Centre

Notes

Monday, Nov. 23 – Workshop C-03

16:00 - 17:00 PEDS: Ortho Problems in Teenagers

Thierry E. Benaroch MD, FRCS(C), FAAOS

The Montreal Children's Hopsital – MUHC

Research interest: Thierry Benaroch is an Assistant Professor at McGill University in the Department of Surgery. He specializes in Pediatric Orthopedic Surgery and is presently the Director of Orthopedic Surgery at the Montreal Children's Hospital. He is also an active staff member at the Shriners Hospital for Children. As well, he is extensively involved in rehabilitation centres; such as, the Mackay Centre and Peter Hall School.

Child to Adolescent Basic Office Orthopaedic Principles

Thierry E. Benaroch, MD, FRCS(C)
MUHC

Adolescents

Psychologic Evolution

Physical Transformation

Chronic Lesions

- Training: Rule of Too 's
  - …Too often
  - …Too long
  - …Too hard

and also:
Too specialized, Too young !

Chronic Lesions

- Pb. training:
  - Coach : Voluntary
  - Pushy
  - Parents

…Achievement of dream through another person

Chronic Lesions

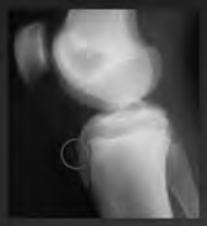
- Very tiny limit between juvenile sports and child abuse

Chronic Lesions

- Predisposing Factors:
  - Training errors
  - Too early specialization
  - Unbalanced ratio of Muscles/Skeleton
  - Anatomical anomalies
- Clinical Findings:
  - Pain
  - Physical Exam: N
  - Delay of growth and puberty

Chronic Lesions

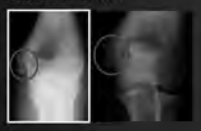
- Apophysitis:
  - Osgood-Schlatter
  - Sever
  - Tibial post.
  - Iselins



Chronic Lesions

« Kissing spine »

Pitcher's Elbow

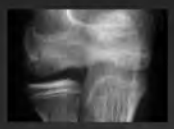


Chronic Lesions : Osteochondritis dissecans

- Treat the patient, not the X-Rays
- Check the other side

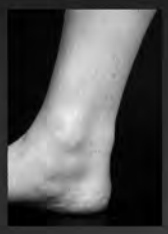
Other lesions

Osteonecrosis



Tendinitis

Very rare before puberty !



Physiological Differences 3

- Growth
  - Increase in length of bones
    - Muscle Stretching

ADOLESCENTS ARE NOT FLEXIBLE !!

Adolescent Knee Pain

Any flags?

Anterior Knee Pain

- Common site of complaint
- Acute trauma
- Repetitive minor trauma

Knee pain in skeletally immature patient – referred hip pain until proven otherwise

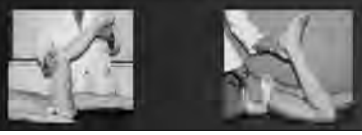
Anterior Knee Pain

HISTORY:
- Poorly localised
- Usually bilateral
- Grab sign
- associated with prolonged sitting, stairs, + theater sign
- Pseudolocking

Anterior Knee Pain

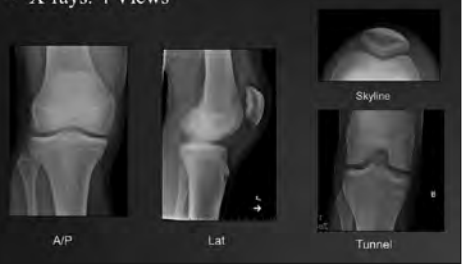
EX:

- Gait, ROM hips, knees, ankles, feet, knee stability, patellar tracking, meniscal tests, tenderness patella, patellar tendon, joint lines
- Atrophy, strength
- Quads/hamstrings flexibility



Anterior Knee Pain

- X-rays: 4 Views



Anterior Knee Pain

- Once other sources of anterior knee pain are R/O, 80% will respond to nonsurgical treatment

Physio: quad, hamstrings, flexibility/strength

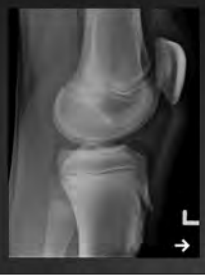
Knee brace

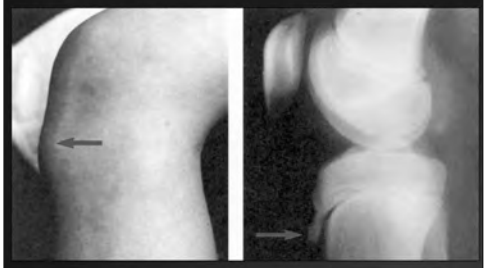
Adolescent anterior knee pain is a mythical disease,equivalent to a headache of the knee.

Surgical intervention has no more rationale than skull burr holes for headache tx.

Anterior Knee Pain

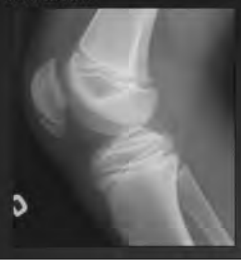
- Osgood-Schlatter





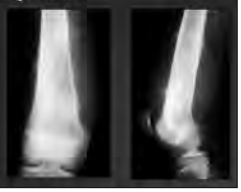
Anterior Knee Pain

- Sinding-Larsen-Johansson



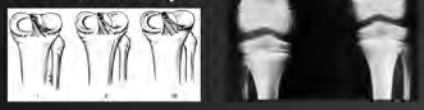
Red Flags

- Hx: Unilateral knee pain
  Swelling, locking, giving way
  No improvement post tx



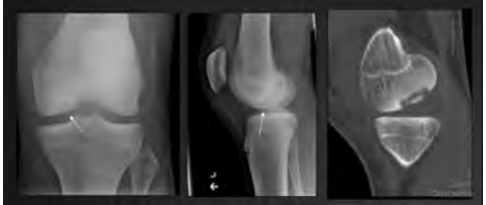
Red Flags

P/E: Swelling
Atrophy
Pain: fem.condyles , joint lines
Abnormal Lig.exam
+ Mc Murray



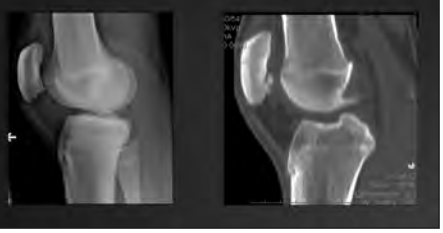
Red Flags

Osteochondritis Dissecans: Femoral Condyle



Red Flags

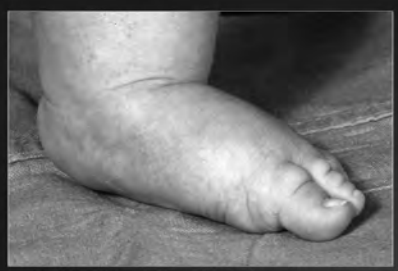
- Osteochondritis Dissecans:Patella

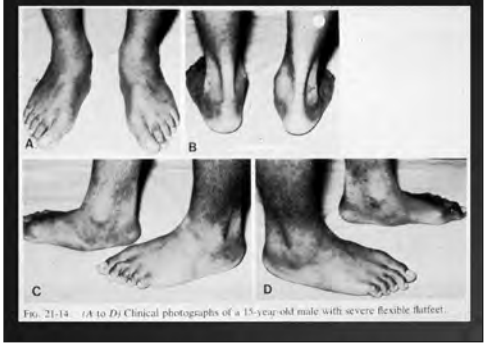


The Flat Foot: A Myth or a Problem?

Flat Feet

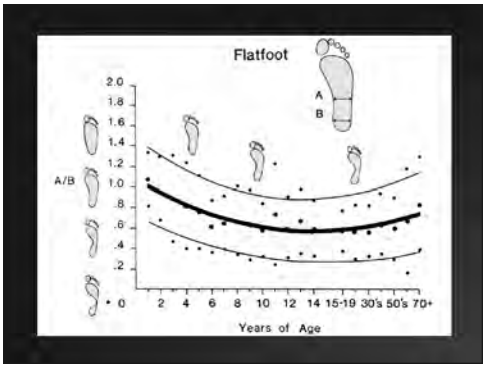
- Abnormally low longitudinal arch due to ligament laxity

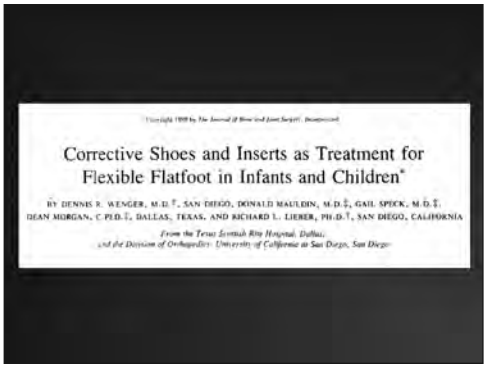




Flat Feet

- Most always asymptomatic
- No correlation to back pain
- Major source of concern to parents

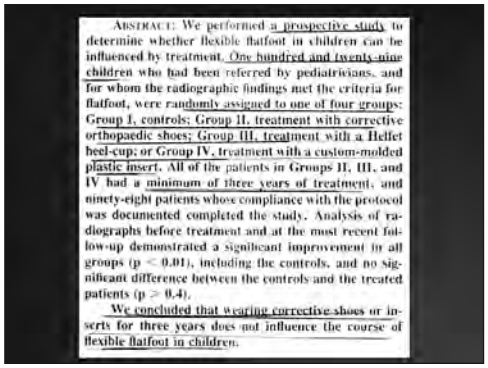




Corrective Shoes and Inserts as Treatment for Flexible Flatfoot in Infants and Children*

BY DENNIS R. WENGER, M.D.†, SAN DIEGO, DONALD MAULDIN, M.D.‡, GAIL SPECK, M.D.§, DEAN MORGAN, C.PED.‖, DALLAS, TEXAS, AND RICHARD L. LIEBER, PH.D.¶, SAN DIEGO, CALIFORNIA

From the Texas Scottish Rite Hospital, Dallas, and the Division of Orthopaedics, University of California at San Diego, San Diego



ABSTRACT: We performed a prospective study to determine whether flexible flatfoot in children can be influenced by treatment. One hundred and twenty-nine children who had been referred by pediatricians, and for whom the radiographic findings met the criteria for flatfoot, were randomly assigned to one of four groups: Group I, controls; Group II, treatment with corrective orthopaedic shoes; Group III, treatment with a Helfet heel-cup; or Group IV, treatment with a custom-molded plastic insert. All of the patients in Groups II, III, and IV had a minimum of three years of treatment, and ninety-eight patients whose compliance with the protocol was documented completed the study. Analysis of radiographs before treatment and at the most recent follow-up demonstrated a significant improvement in all groups ($p < 0.01$), including the controls, and no significant difference between the controls and the treated patients ($p > 0.4$).

We concluded that wearing corrective shoes or inserts for three years does not influence the course of flexible flatfoot in children.

Treatment

- Only if symptomatic (rare) - almost never < 13 years
- Molded arch supports

Tarsal Coalition

- Congenital abnormality with a varying degree of union between 2 or more tarsal bones producing a rigid flat foot

Symptoms

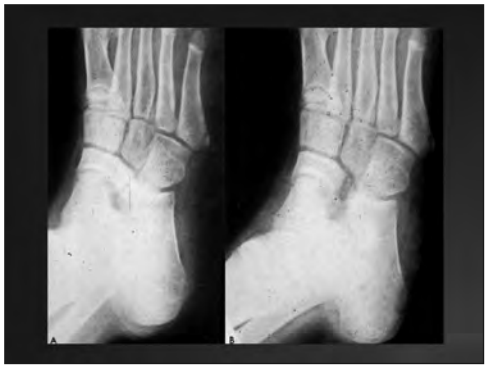
- Pain age 8 - 12 years
- Ossification of bar
- Decreased subtalar motion

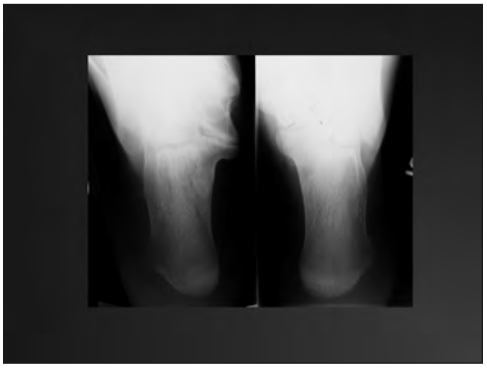
Most Common

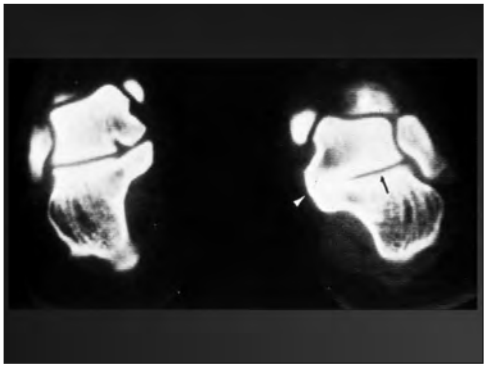
- Calcaneonavicular
- Talo calcaneal
- 50% bilateral

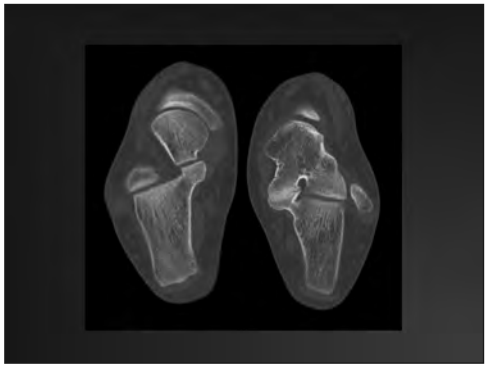
Radiographs

- 45 degree oblique
- Harris Axial Calcaneal View
- CT scan









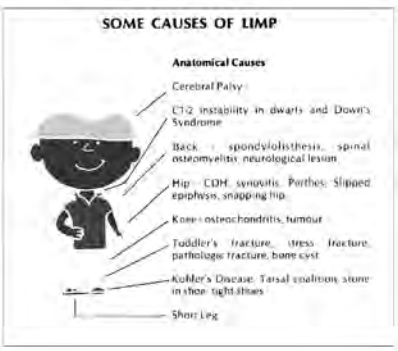
LIMPING

The challenge is to find where and what is the problem with the smallest number of tests, and to determine if further observation or immediate referral is necessary.

You do not want to miss serious conditions such as septic hip.

Look for Five Presentations

- Pain
- Limb length discrepancy
- Stiffness and contractures (sp. Rheumatoid arthritis)
- Neuromuscular conditions
- Hysteria



Approach to a Limping Child

History taking

Details of limping :

- Onset : acute or gradual
- Caused by any specific event (trauma)
- Duration
- Precipitating factors
- Relieving factors
- Systemic manifestations
- Associated signs and symptoms

History taking

1. Is it associated with ***pain*** ?

- Not every limping child is complaining of pain
- Many children will alter their activities in response to a painful stimulus , *before* complaining of pain

History taking

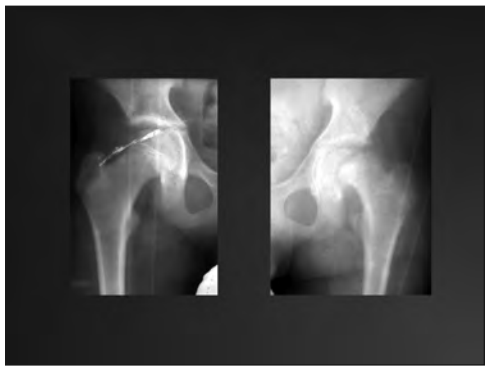
- Night pain (associated with limp) is always worrisome
- History of limp worse in the morning,.... suggests a rheumatological cause

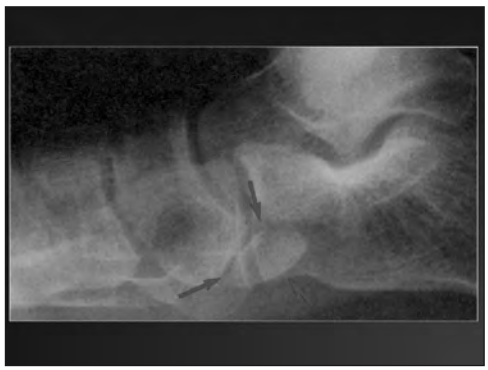
History taking

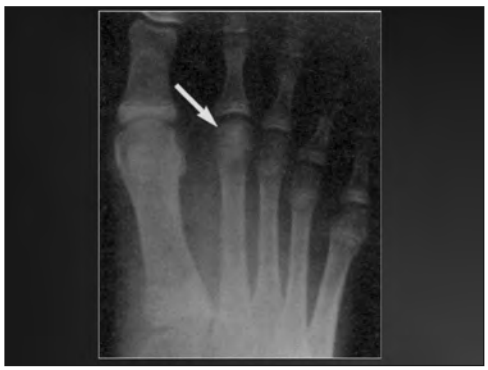
- Referred pain :

Any child or adolescent with thigh or knee pain ,.....

ALWAYS EXAMINE THE HIP







GROWING PAINS

- Usually the pain is bilateral
- Usually the pain occurs only at night
- Usually the child has no limp, pain or symptoms during the day

Examination of a limping child

- Must always examine
  - Spine
  - Pelvis
  - Lower limbs
    - Knees
    - Ankles
    - Feet

You do not want to miss serious, but less evident causes: such as, diskitis, psoas abscess ...

Examination of a limping child

- Three components :

1. Gait examination
2. Standing
3. Table top examination

Abnormal physical signs are always present in a child with a limp which has a serious cause

Back Pain in Children

Objectives

- Demystify back pain in pediatric population
- Define "Red Flag" of back pain in children

History & physical exam

Formulate a step wise management plan including required investigations

Demystify

It is true that back pain in children is not as common as in adults, but neither is it uncommon.

- 40% of children experience some back pain by the age of 15 (1)
- only 2% actually seak Medical attention for their pain (2)

Demystify

It is true that back pain in children is not as common as in adults, but neither is it uncommon

- Children that carried [illegible] were 52% more likely to complain of LBP girls > boys

Demystify

It is true that back pain in children is not as common as in adults, but neither is it uncommon.

However,

22% to had a explaining the pain

Demystify

How do we distinguish the back pain that may represent that hidden disaster?

⇩

Thorough history & physical exam

Clinical Cases 1

History:

7 yr old, otherwise healthy c/o:
Intermittent non-focal neck pain.
Seems to be activity related.
Also complains of a few headaches with no radicular pains.

Benign symptoms:
non-specific, mechanical in nature with no specific neurology

Clinical Cases 1

History:

7 yr old, otherwise healthy c/o:
Intermittent non-focal neck pain.
Seems to be activity related.
Also complains of a few headaches with no radicular pains.

Clinical Cases 1

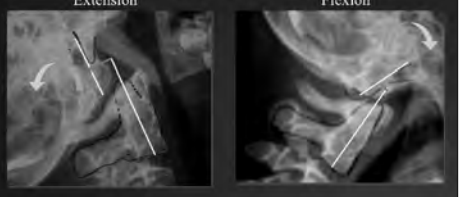
Physical Exam:

Healthy looking child, cute, bashful, head tilt
Full painless ROM except turns left more than right.
Normal neurology exam.
Balance MSK exam, normal.

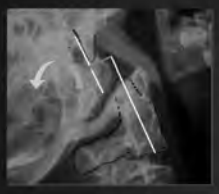
Clinical Cases 1

Investigation:

Extension Flexion



Clinical Cases 1



Diagnosis:

Occipital – Atlas Instability
Requiring spinal stabilization

Clinical Cases 2

History:

9 yr old, gymnast otherwise healthy c/o:
Focal unilat. right low back pain for last 2 - 3 yrs.
Aggravated with activity.
Also complains of tight hamstrings with no radicular pains.

Benign symptoms:
Long standing, mechanical
in nature with no specific neurology

Clinical Cases 2

History:

9 yr old, gymnast otherwise healthy c/o:
Focal unilat. right low back pain for last 2 - 3 yrs.
Aggravated with activity.
Also complains of tight hamstrings with no radicular pains.

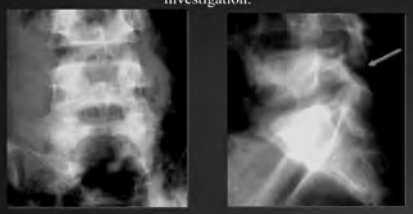
Clinical Cases 2

Physical Exam:

Healthy looking child,
Full painless ROM except extension
Reproduction of pain pattern with extension and lateral bend
Normal neurological exam
Balance MSK exam, normal

Clinical Cases 2

Investigation:

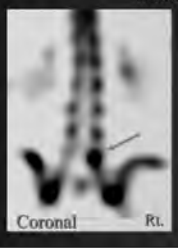


Clinical Cases 2

Investigation: SPECT

Single Photon Emision Computerized Tomogram

3D Bonafied Bone Scan

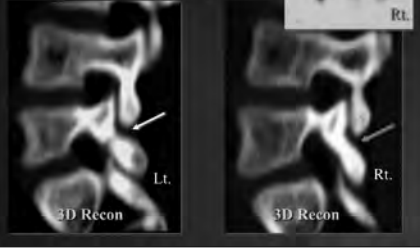


Coronal Rt.

Axial Rt.

Clinical Cases 2

Investigation:



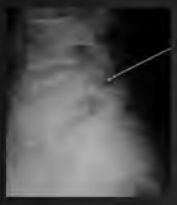
Lt. 3D Recon

Rt. 3D Recon

Rt.

Clinical Cases 2

Diagnosis:



Pars Lysis

Unilateral Spondylolysis

Symptomatic Management
- Activity Modification
- Bracing
- Surgical Repair or Fusion

Clinical Cases 3

History:

3 yr old, otherwise healthy c/o:
Constant focal back pain.
Worse at night for 3 weeks.
3 day history refusal to walk
No focal neurological symptoms
No constitutional symptoms

Clinical Cases 3

Physical Exam:

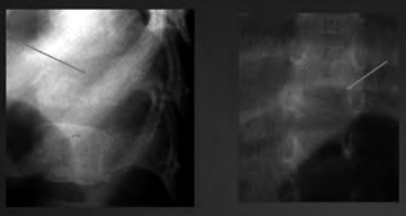
Healthy looking child:
Stiff spine global decrease ROM
Normal neurological exam except
brisker reflex of lower extremity.
Tender thoracolumbar junction
Balance MSK exam. normal.

Clinical Cases 3

Investigation:

Minimum:
Plain x-ray: AP/Lat lumbar spine
Blood Work: CBC, Blood Smear ESR, CRP...
SPECT Scan ascertain activity of lesion
CT scan define bony anatomy
MRI assess soft tissue component
to make tissue diagnosis

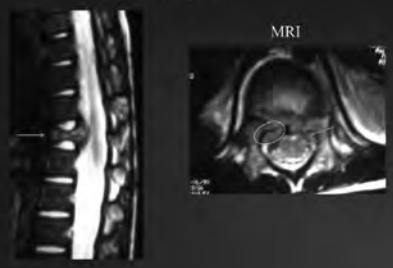
Investigation:



2 weeks later

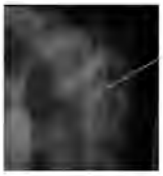
Investigation:

MRI



Clinical Cases 3

Diagnosis:



Vertebra Plana

Eosinophilic Granuloma
Histiocytosis X

Symptomatic Management
- Observation / bracing
- Chemotherapy
- Surgical debridement

How do we distinguish the back pain that may represent that hidden disaster?

Thorough History & Physical Exam

of Back pain in Children

Nature of Pain:

Symptoms	Dx.
• Acute	Fracture
• Unremitting Night Pain	Tumour Infection
• Age < 4	Tumour Inf.
>10	Overuse

Associated Symptoms:

Symptoms	Dx.
• Constitutional (Fever, Chills, Malaise)	Tumour Infection
• Stiff Spine	
• Neurological (Numbness, Disc Herniation Weakness, Apophysis Fracture, Gait abnormality)	Tumour

Radiological of Back pain

Findings	Dx.
•Absent or sclerotic pedicle	Tumour
•Loss of disc space	Infection
•Displaced Post. Corner	Apophyseal #
•Loss of Lordosis	Pain

Standard Work-up

Minimum Radiological Investigation:

Benign History	Worrisome History
• Plain x-ray AP/LAT Spine Involved area.	• Spine Involved area.

Standard Work-up

Minimum Radiological Investigation:

Benign History	Worrisome History
• Plain x-ray AP/LAT Spine Involved area.	• Spine Involved area.

Contrary to adults:
Plain X-ray is the best screening test in pediatric population*
* Feldman, Orthop Trau, 96;16:592

Standard Work-up

Radiological Investigation:

Benign History	Worrisome History
• Plain x-ray AP/LAT Spine Involved area.	• AP/LAT Spine Involved area. • If negative: (Single Photon Emission Computerized Tomogram - 3D Bonafied Bone Scan)

Standard Work-up

Radiological Investigation:

Benign History	Worrisome History
• Plain x-ray AP/LAT Spine Involved area. • If pain > 3 - 6 month (Single Photon Emission Computerized Tomogram - 3D Bonafied Bone Scan)	• AP/LAT Spine Involved area. • If negative: (Single Photon Emission Computerized Tomogram - 3D Bonafied Bone Scan)

Standard Work-up

Radiological Investigation:

Benign History	Worrisome History
• Plain x-ray AP/LAT Spine Involved area.	• AP/LAT Spine Involved area. • If negative: (Single Photon Emission Computerized Tomogram - 3D Bonafied Bone Scan) • of area of activity

Standard Work-up

Radiological Investigation:

Benign History	Worrisome History
• Plain x-ray AP/LAT Spine Involved area.	• AP/LAT Spine Involved area. • If negative: (Single Photon Emission Computerized Tomogram - 3D Bonafied Bone Scan) • of area of activity • , if any signs of neurology

Standard Work-up

Basic Blood Investigation:

Benign History	Worrisome History
• Not standard only if inflammatory process is suspected	•

Notes

16:00 - 17:00 PEDS: Pediatric Eye Exam

Rosanne Superstein MD, FRCSC
Royal Victoria Hospital - MUHC

Biography:

Rosanne Superstein graduated from medical school at McGill University in 1994. She did her ophthalmology residency at McGill University and fellowship at the University of Michigan in Pediatric Ophthalmology and Adult Strabismus in 1999-2000. She is assistant professor at McGill University and Universite de Montreal. She currently sees children at Ste Justine hospital and sees adults with strabismus at the Sir Mortimer B Davis Jewish General Hospital. She has a special interest in retinoblastoma and is a member of the Children's Oncology Group. She enjoys teaching medical students, residents, and fellows.

Learning objectives:

To review the American Academy of pediatrics 2003 eye examination guidelines.

To review a practical approach to examining children.

To review the concept of amblyopia

To review common pediatric eye diseases.

AMERICAN ACADEMY OF PEDIATRICS
Committee on Practice and Ambulatory Medicine and Section on Ophthalmology
AMERICAN ASSOCIATION OF CERTIFIED ORTHOPTISTS
AMERICAN ASSOCIATION FOR PEDIATRIC OPHTHALMOLOGY AND STRABISMUS
AMERICAN ACADEMY OF OPHTHALMOLOGY

POLICY STATEMENT

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of All Children

Eye Examination in Infants, Children, and Young Adults by Pediatricians

ABSTRACT. Early detection and prompt treatment of ocular disorders in children is important to avoid lifelong visual impairment. Examination of the eyes should be performed beginning in the newborn period and at all well-child visits. Newborns should be examined for ocular structural abnormalities, such as cataract, corneal opacity, and ptosis, which are known to result in visual problems. Vision assessment beginning at birth has been endorsed by the American Academy of Pediatrics, the American Association for Pediatric Ophthalmology and Strabismus, and the American Academy of Ophthalmology. All children who are found to have an ocular abnormality or who fail vision assessment should be referred to a pediatric ophthalmologist or an eye care specialist appropriately trained to treat pediatric patients.

INTRODUCTION

Eye examination and vision assessment are vital for the detection of conditions that result in blindness, signify serious systemic disease, lead to problems with school performance, or at worst, threaten the child's life. Through careful evaluation of the ocular system, retinal abnormalities, cataracts, glaucoma, retinoblastoma, strabismus, and neurologic disorders can be identified, and prompt treatment of these conditions can save a child's vision or even life. Examination of the eyes should be performed beginning in the newborn period and at all well-child visits. Visual acuity measurement should be performed at the earliest possible age that is practical (usually at approximately 3 years of age). Early detection and prompt treatment of ocular disorders in children is important to avoid lifelong permanent visual impairment.

TIMING OF EXAMINATION AND SCREENING

Children should have an assessment for eye problems in the newborn period and then at all subsequent routine health supervision visits. These should be age-appropriate evaluations as described in subsequent sections. Infants and children at high risk of eye problems should be referred for specialized eye examination by an ophthalmologist experienced in treating children. This includes children who are very premature; those with family histories of congenital cataracts, retinoblastoma, and metabolic or genetic diseases; those who have significant developmental delay or neurologic difficulties; and those with systemic disease associated with eye abnormalities. Because children do not complain of visual difficulties, visual acuity measurement (vision screening) is an important part of complete pediatric eye care and should begin at 3 years of age. To achieve the most accurate testing possible, the most sophisticated test that the child is capable of performing should be used (Table 1).[1,2] The frequency of examinations recommended is in accordance with the American Academy of Pediatrics "Recommendations for Preventive Pediatric Health Care."[2] Any child unable to be tested after 2 attempts or in whom an abnormality is suspected or detected should be referred for an initial eye evaluation by an ophthalmologist experienced in the care of children.

PROCEDURES FOR EYE EVALUATION

Eye evaluation in the physician's office should include the following:

Birth to 3 Years of Age

1. Ocular history
2. Vision assessment
3. External inspection of the eyes and lids
4. Ocular motility assessment
5. Pupil examination
6. Red reflex examination

3 Years and Older

1 through 6, plus:

7. Age-appropriate visual acuity measurement
8. Attempt at ophthalmoscopy

TABLE 1. Eye Examination Guidelines*

Function	Recommended Tests	Referral Criteria	Comments
Ages 3–5 Years			
Distance visual acuity	Snellen letters Snellen numbers Tumbling E HOTV Picture tests –Allen figures –LEA symbols	1. Fewer than 4 of 6 correct on 20-ft line with either eye tested at 10 ft monocularly (ie, less than 10/20 or 20/40) or 2. Two-line difference between eyes, even within the passing range (ie, 10/12.5 and 10/20 or 20/25 and 20/40)	1. Tests are listed in decreasing order of cognitive difficulty; the highest test that the child is capable of performing should be used; in general, the tumbling E or the HOTV test should be used for children 3–5 years of age and Snellen letters or numbers for children 6 years and older. 2. Testing distance of 10 ft is recommended for all visual acuity tests. 3. A line of figures is preferred over single figures. 4. The nontested eye should be covered by an occluder held by the examiner or by an adhesive occluder patch applied to eye; the examiner must ensure that it is not possible to peek with the nontested eye.
Ocular alignment	Cross cover test at 10 ft (3 m)	Any eye movement	Child must be fixing on a target while cross cover test is performed.
	Random dot E stereo test at 40 cm	Fewer than 4 of 6 correct	
	Simultaneous red reflex test (Bruckner test)	Any asymmetry of pupil color, size, brightness	Direct ophthalmoscope used to view both red reflexes simultaneously in a darkened room from 2 to 3 feet away; detects asymmetric refractive errors as well.
Ocular media clarity (cataracts, tumors, etc)	Red reflex	White pupil, dark spots, absent reflex	Direct ophthalmoscope, darkened room. View eyes separately at 12 to 18 inches; white reflex indicates possible retinoblastoma.
6 years and older			
Distance visual acuity	Snellen letters Snellen numbers Tumbling E HOTV Picture tests -Allen figures -LEA symbols	1. Fewer than 4 of 6 correct on 15-ft line with either eye tested at 10 ft monocularly (ie, less than 10/15 or 20/30) or 2. Two-line difference between eyes, even within the passing range (ie, 10/10 and 10/15 or 20/20 and 20/30)	1. Tests are listed in decreasing order of cognitive difficulty; the highest test that the child is capable of performing should be used; in general, the tumbling E or the HOTV test should be used for children 3–5 years of age and Snellen letters or numbers for children 6 years and older. 2. Testing distance of 10 ft is recommended for all visual acuity tests. 3. A line of figures is preferred over single figures. 4. The nontested eye should be covered by an occluder held by the examiner or by an adhesive occluder patch applied to eye; the examiner must ensure that it is not possible to peek with the nontested eye.
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Ocular media clarity (cataracts, tumors, etc)	Red reflex	White pupil, dark spots, absent reflex	Direct ophthalmoscope, darkened room. View eyes separately at 12 to 18 inches; white reflex indicates possible retinoblastoma.

* Assessing visual acuity (vision screening) represents one of the most sensitive techniques for the detection of eye abnormalities in children. The American Academy of Pediatrics Section on Ophthalmology, in cooperation with the American Association for Pediatric Ophthalmology and Strabismus and the American Academy of Ophthalmology, has developed these guidelines to be used by physicians, nurses, educational institutions, public health departments, and other professionals who perform vision evaluation services.

Ocular History

Parents' observations are valuable. Questions that can be asked include:

- Does your child seem to see well?
- Does your child hold objects close to his or her face when trying to focus?
- Do your child's eyes appear straight or do they seem to cross or drift or seem lazy?
- Do your child's eyes appear unusual?
- Do your child's eyelids droop or does 1 eyelid tend to close?
- Have your child's eye(s) ever been injured?

Relevant family histories regarding eye disorders or preschool or early childhood use of glasses in parents or siblings should be explored.

Vision Assessment

Age 0 to 3 Years

Vision assessment in children younger than 3 years or any nonverbal child is accomplished by evaluating the child's ability to fix and follow objects.[3,4] A standard assessment strategy is to determine whether each eye can fixate on an object, maintain fixation, and then follow the object into various gaze positions. Failure to perform these maneuvers indicates significant visual impairment. The assessment should be performed binocularly and then monocularly. If poor fix and following is noted binocularly after 3 months of age, a significant bilateral eye or brain abnormality is suspected, and referral for more formal vision assessment is advisable.[5] It is important to ensure that the child is awake and alert, because disinterest or poor cooperation can mimic a poor vision response.

Visual Acuity Measurement or Vision Screening (Older Than 3 Years)

Various tests are available to the pediatrician for measuring visual acuity in older children. Different picture tests, such as LH symbols (LEA symbols) and Allen cards, can be used for children 2 to 4 years of age. Tests for children older than 4 years include wall charts containing Snellen letters, Snellen numbers, the tumbling E test, and the HOTV test (a letter-matching test involving these 4 letters).[6] A study of 102 pediatric practices revealed that 53% use vision testing machines.[3] Because testing with these machines can be difficult for younger children (3–4 years of age), pediatricians should have picture cards and wall charts available.

Photoscreening

Using this technique, a photograph is produced by a calibrated camera under prescribed lighting conditions, which shows a red reflex in both pupils. A trained observer can identify ocular abnormalities by recognizing characteristic changes in the photographed pupillary reflex.[7] When performed properly, the technique is fast, efficient, reproducible, and highly reliable. Photoscreening is not a substitute for accurate visual acuity measurement but can provide significant information about the presence of sight-threatening conditions, such as strabismus, refractive errors, media opacities (cataract), and retinal abnormalities (retinoblastoma). Photoscreening techniques are still evolving. (For further information, see also the American Academy of Pediatrics policy statement, "Use of Photoscreening for Children's Vision Screening."[8])

External Examination (Lids/Orbit/Cornea/Iris)

External examination of the eye consists of a penlight evaluation of the lids, conjunctiva, sclera, cornea, and iris. Persistent discharge or tearing may be attributable to ocular infection, allergy, or glaucoma, but the most common cause is lacrimal duct obstruction. It often manifests during the first 3 months as persistent purulent discharge out of 1 or both eyes. Topical or oral antibiotics should be given, and lacrimal sac massage should be attempted. Because these same findings are often seen in congenital glaucoma, failure to promptly resolve after treatment or the presence of cloudy or asymmetrically enlarged corneas should prompt ophthalmologic referral for additional evaluation.

Unilateral ptosis can cause amblyopia by inducing astigmatism, even if the pupil is not occluded. Patients with this condition require ophthalmic evaluation. Bilateral ptosis may be associated with significant neurologic disease, such as myasthenia. Additional investigation by a child neurologist and pediatric ophthalmologist is warranted.

Ocular Motility

The assessment of ocular alignment in the preschool and early school-aged child is of considerable importance. The development of strabismus in children may occur at any age and can represent serious orbital, intraocular, or intracranial disease. The corneal reflex test, cross cover test, and random dot E stereo test are useful in differentiating true strabismus from pseudostrabismus (see Appendix 1). The most common cause of pseudostrabismus is prominent epicanthal lid folds that cover the medial portion of the sclera on both eyes, giving the impression of crossed eyes (esotropia). Detection of an eye muscle imbalance or inability to differentiate strabismus from pseudostrabismus necessitates a referral.

Pupils

The pupils should be equal, round, and reactive to light in both eyes. Slow or poorly reactive pupils may indicate significant retinal or optic nerve dysfunction. Asymmetry of pupil size, with 1 pupil larger than the other, can be attributable to a sympathetic disorder (Horner syndrome) or a parasympathetic abnormality (third nerve palsy, Adie syndrome). Small differences can occur normally and should be noted in the chart for reference in case of subsequent head injury. Larger pupil asymmetries (>1 mm) can be attributable to serious neurologic disorders and need additional investigation.

Red Reflex Test (Monocular and Binocular, Bruckner Test)

The red reflex test can be used to detect opacities in the visual axis, such as a cataract or corneal abnor-

mality, and abnormalities of the back of the eye, such as retinoblastoma or retinal detachment. When both eyes are viewed simultaneously, potentially amblyogenic conditions, such as asymmetric refractive errors and strabismus, also can be identified. The test should be performed in a darkened room (to maximize pupil dilation). The direct ophthalmoscope is focused on each pupil individually approximately 12 to 18 inches away from the eye, and then both eyes are viewed simultaneously at approximately 3 feet away. The red reflex seen in each eye individually should be bright reddish-yellow (or light gray in darkly pigmented, brown-eyed patients) and identical in both eyes. Dark spots in the red reflex, a blunted dull red reflex, lack of a red reflex, or presence of a white reflex are all indications for referral. After assessing each eye separately, the eyes are viewed together with the child focusing on the ophthalmoscope light (Bruckner test, see Appendix 1). As before, any asymmetry in color, brightness, or size is an indication for referral, because asymmetry may indicate an amblyogenic condition.

Visual Acuity Measurement (Vision Screening)

Visual acuity testing is recommended for all children starting at 3 years of age.[6] In the event that the child is unable to cooperate for vision testing, a second attempt should be made 4 to 6 months later. For children 4 years and older, the second attempt should be made in 1 month. Children who cannot be tested after repeated attempts should be referred to an ophthalmologist experienced in the care of children for an eye evaluation. Appendix 1 provides a detailed explanation of the techniques available for visual acuity measurement in children.

Ophthalmoscopy

Ophthalmoscopy may be possible in very cooperative 3- to 4-year-olds who are willing to fixate on a toy while the ophthalmoscope is used to evaluate the optic nerve and retinal vasculature in the posterior pole of the eye.

RECOMMENDATIONS

1. All pediatricians and other providers of health care to children should be familiar with the joint eye examination guidelines of the American Association for Pediatric Ophthalmology and Strabismus, the American Academy of Ophthalmology, and the American Academy of Pediatrics.
2. Every effort should be made to ensure that eye examinations are performed using appropriate testing conditions, instruments, and techniques.
3. Newborns should be evaluated for ocular structural abnormalities, such as cataract, corneal opacities, and ptosis, which are known to result in vision problems, and all children should have their eyes examined on a regular basis.[1]
4. The results of vision assessments, visual acuity measurements, and eye evaluations, along with instructions for follow-up care, should be clearly communicated to parents.[2]
5. All children who are found to have an ocular abnormality or who fail vision screening should be referred to a pediatric ophthalmologist or an eye care specialist appropriately trained to treat pediatric patients.

APPENDIX 1. TESTING PROCEDURES FOR ASSESSING VISUAL ACUITY

The child should be comfortable and in good health at the time of the examination. It is often convenient to have younger children sit on a parent's lap. If possible, some preparation before the actual testing situation is helpful, and parents can assist by demonstrating the anticipated testing procedures for their child. Children who have eyeglasses generally should have their vision tested while wearing the eyeglasses. Eyeglasses prescribed for use only

while reading should not be worn when distance acuity is being tested.

Consideration must be given to obtaining good occlusion of the untested eye; cardboard and paddle occluders have been found inadequate for covering the eye because they allow "peeking." Commercially available occluder patches provide complete occlusion necessary for appropriate testing.[1] Vision testing should be performed at 10 feet (except Allen cards) and in a well-lit area. When ordering wall charts, be sure to indicate that a 10-foot testing distance will be used.

Visual Acuity Tests

Snellen Acuity Chart

When performing visual acuity testing, test the child's right eye first by covering the left. A child who has corrective eyeglasses should be screened wearing the eyeglasses. Tell the child to keep both eyes open during testing. If the child fails the practice line, move up the chart to the next larger line. If the child fails this line, continue up the chart until a line is found that the child can pass. Then move down the chart again until the child fails to read a line. After the child has correctly identified 2 symbols on the 10/25 line, move to the critical line (10/20 or 20/40 equivalent). To pass a line, a child must identify at least 4 of the 6 symbols on the line correctly. Repeat the above procedure covering the right eye.

Tumbling E

For children who may be unable to perform vision testing by letters and numbers, the tumbling E or HOTV test may be used. Literature is available from the American Academy of Ophthalmology (*Home Eye Test*, American Academy of Ophthalmology, PO Box 7424, San Francisco, CA 94109, 415/561-8500 or http://www.aao.org) and Prevent Blindness America (*Preschoolers Home Eye Test*, Prevent Blindness America, 500 East Remington Rd, Schaumburg, IL 60173, 847/843-2020 or http://www.preventblindness.com) for home use by parents to prepare children for the tumbling E test. This literature contains the practice Es, a tumbling E wall chart, and specific instructions for parents.

HOTV Test (Matching Test)

An excellent test for children who are unable to perform vision testing by verbally identifying letters and numbers is the HOTV matching test. This test consists of a wall chart composed only of Hs, Os, Ts, and Vs. The child is provided an 8½ × 11-inch board containing a large H, O, T, and V. The examiner points to a letter on the wall chart, and the child points to (matches) the correct letter on the testing board. This can be especially useful in the 3- to 5-year-old who is unfamiliar with the alphabet.

Allen Cards

The Allen card test consists of 4 flash cards containing 7 schematic figures: a truck, house, birthday cake, bear, telephone, horse, and tree. When viewed at 20 feet, these figures represent 20/30 vision. It is important that a child identify verbally or by matching all 7 pictures before actual visual testing. Testing should only be performed with the figures that the child readily identified. Perform initial testing with the child having both eyes open, viewing the cards at 2 to 3 feet away. Present 1 or 2 figures to ensure that the child understands the testing procedure. Then begin walking backward 2 to 3 feet at a time, presenting different pictures to the child. Continue to move backward as long as the child directly calls out the figures presented. When the child begins to miss the figures, move forward several feet to confirm that the child is able to identify the figures at the shorter distance. To calculate an acuity score, the furthest distance at which the child is able to identify the pictures accurately is the numerator and 30 is the denominator. Therefore, if a child were able to identify pictures accurately at 15 feet, the visual acuity would be recorded as 15/30. This is equivalent to 30/60, 20/40, or 10/20. To perform this test in the same way as for HOTV testing, a "matching panel" of all of the Allen figures may be prepared on a copy machine.

LH Symbols (LEA Symbols)

The LH symbol test is slightly different from the Allen card test in that it is made up of flash cards held together by a spiral binding. The flash cards contain large examples of a house, apple, circle, and square; these should be presented to the child before formal vision testing to see if they can be correctly identified. Unlike the Allen cards, the LH symbol test contains flash cards with more than 1 figure per card and with smaller figure sizes so that testing may be performed at 10 feet. Recorded on each card is the symbol size and visual acuity value for a 10-foot testing distance. The visual acuity is determined by the smallest symbols that the child is able to identify accurately at 10 feet. For example, if the child is able to identify the 10/15 symbol at 10 feet, the child's visual acuity is 10/15 or 20/30.

If it is not possible to perform testing at 10 feet, move closer to the child until he or she correctly identifies the largest symbol. At this point, proceed down in size to the smallest symbols the child is consistently able to correctly identify. The vision is recorded as the smallest symbol identified (bottom number) at the testing distance (top number). For example, correctly identifying the 10/15 symbols at 5 feet is recorded as 5/15 or 20/60. Likewise, identifying the 10/30 symbols at 2 feet is 2/30 or 20/300 (both the bottom and top numbers can be multiplied or divided by the same number to give an equivalent vision.) A "matching panel" is provided with the LH test and may be helpful in testing very young children. At least 3 of 4 figures should be identified for each size or distance.

Testing Procedures for Assessing Ocular Alignment

Corneal Light Reflex Test

A penlight may be used to evaluate light reflection from the cornea. The light is held approximately 2 feet in front of the face to have the child fixate on the light. The corneal light reflex (small white dot) should be present symmetrically and appear to be in the center of both pupils. A reflex that is off center in 1 eye may be an indication of an eye muscle imbalance. A slight nasal displacement of the reflex is normal, but a temporal displacement is almost never seen unless the child has a strabismus (esotropia).

Simultaneous Red Reflex Test (Bruckner Test)

This test can detect amblyogenic conditions, such as unequal refractive errors (unilateral high myopia, hyperopia, or astigmatism), as well as strabismus and cataracts. When both eyes are viewed simultaneously through the direct ophthalmoscope in a darkened room from a distance of approximately 2 to 3 feet with the child fixating on the ophthalmoscope light, the red reflexes seen from each eye should be equal in size, brightness, and color. If 1 reflex is different from the other (lighter, brighter, or bigger), there is a high likelihood that an amblyogenic condition exists. Any child with asymmetry should be referred for additional evaluation. Examples of normal and abnormal Bruckner test appearances are available from the AAP. "See Red" cards are available for purchase at http://www.aap.org/sections/ophthal.htm.

Cross Cover Test

To perform the cross cover test, have the child look straight ahead at an object 10 feet (3 meters) away. This could be an eye chart for older children or a colorful noise-making toy for younger children. As the child looks at a distant object, cover 1 eye with an occluder and look for movement of the uncovered eye. As an example, if the occluder is covering the left eye, movement is looked for in the uncovered right eye. This movement will occur immediately after the cover is placed in front of the left eye. If the right eye moves outward, the eye was deviated inward or esotropic. If the right eye moves inward, it was deviated outward or exotropic. After testing the right eye, test the left eye for movement in a similar manner. If there is no apparent misalignment of either eye, move the cover back and forth between the 2 eyes, waiting about 1 to 2 seconds between movements. If after moving the occluder, the uncovered eye moves in or out to take up fixation, a strabismus is present. Any movement in or out when shifting the cover indicates a strabismus is present, and a referral should be made to an ophthalmologist.

Random Dot E Stereo Test

The random dot E stereo test measures stereopsis. This is different from the light reflex test or the cover test, which detects physical misalignment of the eyes. Stereopsis can be absent in patients with straight eyes. An ophthalmologic evaluation is necessary to detect the causes of poor stereo vision with straight eyes. To perform the

random dot E stereo test, the cards should be held 16 inches from the child's eyes. Explain the test to the child. Show the child the gray side of the card that says "model" on it. Hold the model E in the direction at which the child can read it correctly. Have the child touch the model E to understand better that the picture will stand out. A child should be able to indicate which direction the legs are pointing. Place the stereo glasses on the child. If the child is wearing eyeglasses, place the stereo glasses over the child's glasses. Make sure the glasses stay on the child and the child is looking straight ahead. The child should be shown both the stereo blank card and the raised and recessed E card simultaneously. Hold each card so you can read the back. The blank card should be held so you can read it. The E card should be held so you can read the word "raised." Both cards must be held straight. Do not tilt the cards toward the floor or the ceiling—this will cause darkness and glare. Ask the child to look at both cards and to point to or touch the card with the picture of the E. The E must be presented randomly, switching from side to side. The child is shown the cards up to 6 times. To pass the test, a child must identify the E correctly in 4 of 6 attempts.

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All policy statements from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

Notes

16:00 - 17:00 Laboratory Investigations in Rheumatology and Immunology

Jan Schulz MD, FRCPC, FACP

Associate Professor, Department of Medicine, McGill University

Laboratory investigations in Rheumatology and Immunology

Jan Schulz MD,FRCP,FACP
Associate Professor,
McGill University

I have no disclosures to make

OBJECTIVES : 1. To understand appropriate use of currently available tests in rhematology and immunology

2. Review of new tests,soon to become available

Diagnostic Tests

- Definitive Tests – establish or rule out a disorder with certainty, e.g.:

A. Culture of synovial fluid – septic arthritis
B. Polarized light microscopy – gout, "pseudogout"

- Confirmatory and exclusionary tests

use of these tests presuposes that a clinician has some underlying suspicion, however small, that a specific disorder is present

Case 1

- 38 F, teacher, presents with fatigue, morning stiffness and painful, swollen wrists of 2 months duration.
- O/E : both wrists are warm, with effusion on R and painful and decreased ROM bilat.
- You order all the necessary tests-- which ones ?

Case 1, continued

- Lab Data
- HB 117 ; WBC 8,2 , Plts 145000 ; ESR 37 ; CRP 14,2 ; RF Neg ; anti CCP 97
- X ray hands , wrists : soft tissue swelling, osteopenia , no erosions

Case 2

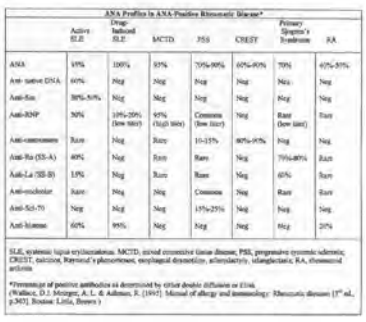
- 32 F, presents with 6 months Hx of alopecia, joint pains, without swelling, and acute appearance of facial rash, chest pain and shortness of breath while vacationing last December in Florida. She took Aleve and was able to obtain some relieve from joint and chest pains but remains very short of breath. You decide to send her to ER, where basic work up is performed. What would you do, to diagnose / treat this pt optimally ?

Case 2 contd

- O/E BP 110/60; P: 98,reg PP 15 O2 sat Ra 96%, adenopathy neck, Malar rash, Lungs : clesr to ausc, Cor : No murmurs, pericardial rub Abd : neg, MSK : No synovitis,

Case 2 continued

- Labs in ER
- Hb 83 ; WBC 3.9 lymph 500; Plts 98 000; Cr 70; LFT : N ; CK : N ; LDH 689 ; U/A N ; ANA 1:640 ; C 3 : 52 ; C 4 : 0.11 ; ENA SM & RNP positive, Antiphospholipid Ab : neg



VASCULITIS-ASSOCIATED AND NONSPECIFIC AUTOANTIBODIES

-ANTINEUTROPHIL CYTOPLASMIC ANTIBODY (ANCA)

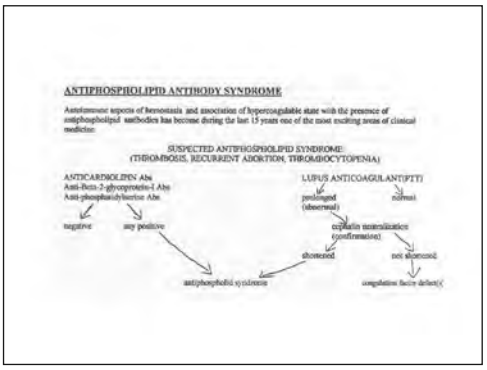
C-ANCA - antibody against proteinase-3 (PR3)
- present in 90% of active generalized Wegener's granulomatosis and in 60% of limited or treated Wegener's

P-ANCA - antibody against myeloperoxidase
-present in idiopathic crescentic glomerulonephritis, microscopic polyarteritis and inflammatory bowel disease

FREQUENCY OF HLA B27 IN RHEUMATIC DISEASES IN CAUCASIANS

Healthy Caucasians *	6-10%
Ankylosing spondylitis **	90%
Reiter's	70-90%
Psoriasi arthritis with sacroiliitis	50-60%
Inflammatory bowel disease with sacroiliitis	50-70%
Juvenile rheumatoid arthritis with sacroiliitis	40-60%
Reactive arthritis - yersinia	80%
-salmonella	80-90%
-shigella	80%
Idiopathic iritis	40-60%

*Healthy US blacks = 3-4%, Healthy orientals ,<1%
**US blacks with spondylitis = 50-60%, orientals with spondylitis = 90%



ANTIPHOSPHOLIPID ANTIBODY SYNDROME

Autoimmune aspects of hemostasis and association of hypercoagulable state with the presence of antiphospholipid antibodies has become during the last 15 years one of the most exciting areas of clinical medicine.

SUSPECTED ANTIPHOSPHOLIPID SYNDROME:
(THROMBOSIS, RECURRENT ABORTION, THROMBOCYTOPENIA)

ANTICARDIOLIPIN Abs
Anti-Beta-2-glycoprotein-I Abs
Anti-phosphatidylserine Abs

LUPUS ANTICOAGULANT(PTT)
prolonged (observed)
normal

negative
any positive
cephalin neutralization (confirmation)
shortened
not shortened
antiphospholipid syndrome
coagulation factor defect(

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3. Jaeschke R, Guyatt G, Sackett DL, for the Evidenced-based Medicine Working Group. Users' guide to the medical literature. III. How to use an article about a diagnostic test. A. Are the results of the study valid? JAMA 1994;271:389–91.
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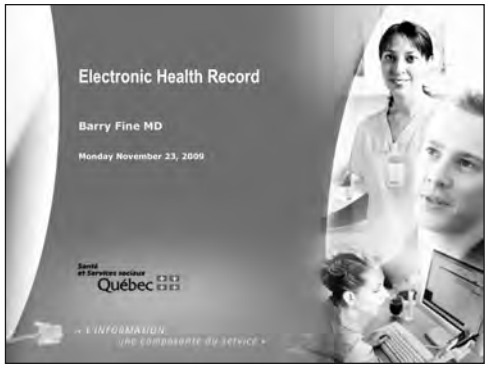
Notes

Monday, Nov. 23 – Workshop C-06

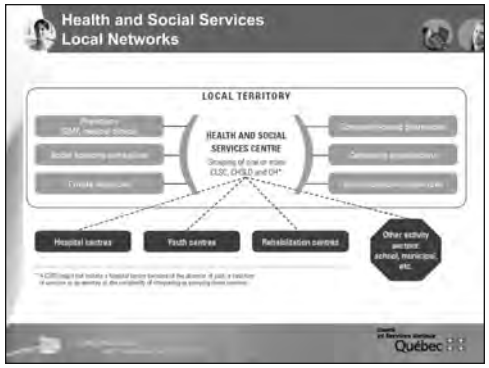
16:00 - 17:00 Electronic Health Record

Barry Fine MD
Lecturer, Faculty of Medicine, McGill University

Barry Fine has been a family physician since 1979. After receiving a BSc from McGill in 1970 he received his medical degree from Université Montepellier in 1977. He did his residency in Family Medicine at St. Mary's Hospital and The Montreal Children's Hospital in 1979. Following his residency he practiced family medicine as a solo practitioner until 2006 after which he became permanent physician for the Cree Health Board at Chisasibi Hopsital in James Bay, Quebec.

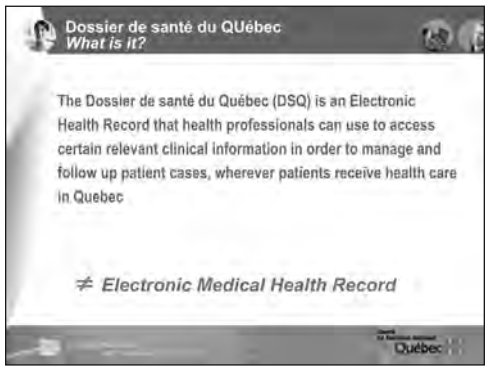

Electronic Health Record
Barry Fine MD
Monday November 23, 2009
Québec

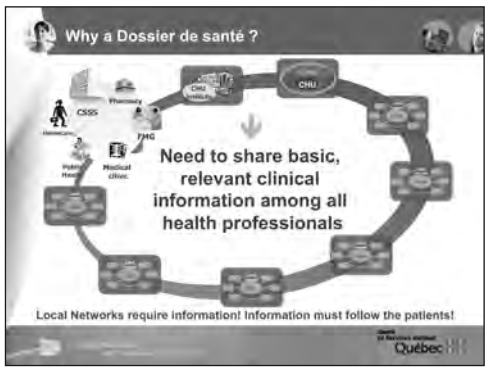

Health and Social Services in Quebec
Québec

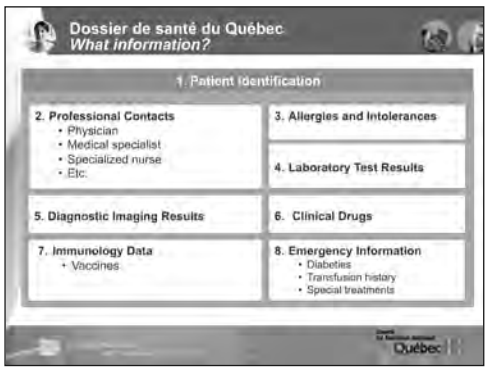

Health and Social Services Local Networks
LOCAL TERRITORY
HEALTH AND SOCIAL SERVICES CENTRE
Hospital centres
Youth centres
Rehabilitation centres
Québec

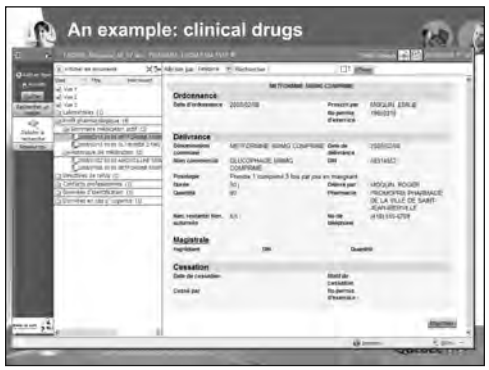

Health and Social Services Governance Structure
MINISTER
Québec

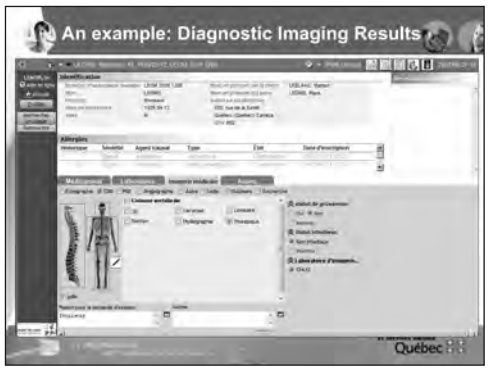

DSQ 101!
Québec


Dossier de santé du QUébec
What is it?
The Dossier de santé du Québec (DSQ) is an Electronic Health Record that health professionals can use to access certain relevant clinical information in order to manage and follow up patient cases, wherever patients receive health care in Quebec
≠ Electronic Medical Health Record
Québec

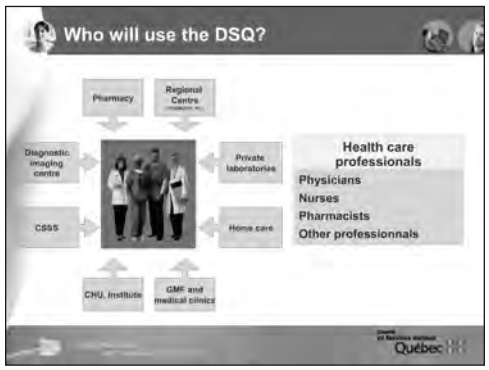

Why a Dossier de santé ?
CHU
CSSS
Pharmacy
Need to share basic, relevant clinical information among all health professionals
Local Networks require information! Information must follow the patients!
Québec

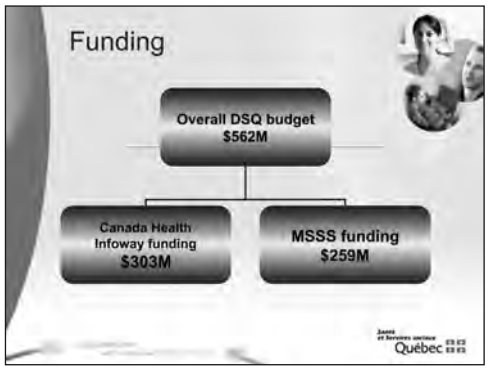

Dossier de santé du Québec
What information?
1. Patient Identification
2. Professional Contacts
• Physician
• Medical specialist
• Specialized nurse
• Etc.
3. Allergies and Intolerances
4. Laboratory Test Results
5. Diagnostic Imaging Results
6. Clinical Drugs
7. Immunology Data
• Vaccines
8. Emergency Information
• Diabetes
• Transfusion history
• Special treatments
Québec


An example: clinical drugs
Ordonnance
Délivrance
Magistrale
Cessation

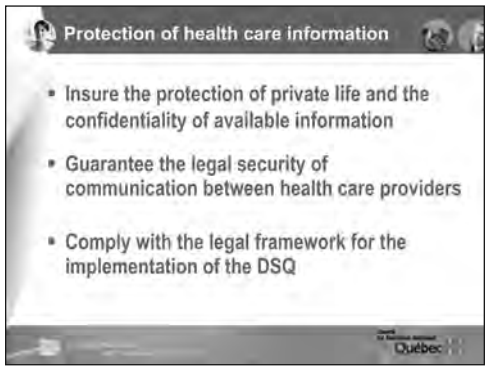

An example: Diagnostic Imaging Results
Québec


Dossier de santé du Québec
For whom?
Any insured person receiving care in Quebec who has not refused to have an EHR
Québec


Who will use the DSQ?
Pharmacy
Regional Centre
Diagnostic imaging centre
Private laboratories
CSSS
Home care
CHU, institute
GMF and medical clinics
Health care professionals
Physicians
Nurses
Pharmacists
Other professionnals
Québec


Funding
Overall DSQ budget $562M
Canada Health Infoway funding $303M
MSSS funding $259M
Québec


LEGAL FRAMEWORK
Québec


Protection of health care information
• Insure the protection of private life and the confidentiality of available information
• Guarantee the legal security of communication between health care providers
• Comply with the legal framework for the implementation of the DSQ
Québec

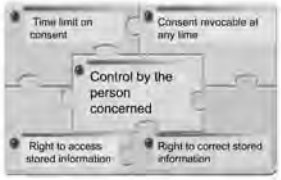
Human rights / consent

Free and informed

Obligation to inform

Limited duration: 5 years

Can be revoked at any time



BENEFITS

To learn more about the benefits evaluation work done for the DSQ, follow this link to the DSQ Internet site:

http://www.dossierdesante.gouv.qc.ca/benefices/

DSQ: clinical benefits

Quality and security

- Reduced patient treatment times
- Fewer medical errors
- Fewer medication side effects
- Fewer prescription errors
- Better access to diagnostic lab tests and DI results

DSQ: clinical benefits

Accessibility

- Increased number of interpretations by specialists on remote site
- Greater self-sufficiency for the regions and the front line
- Help reduce congestion in major centres

DSQ: clinical benefits

Continuity

- Better coordination among service providers
- Keeps patients from having to repeat their medical history to the different service providers they meet throughout an episode of care

DSQ: benefits linked to resource use

Productivity

- Less time required to reconstruct patients' clinical information, including their medical history
- Fewer prescription recalls

Controlling or cutting costs

- More effective management of care episodes
- Fewer duplicate tests and prescriptions
- Lower administrative costs: record and archiving requests, transcripts, telephone calls, mail-outs, transportation

CHALLENGES

Challenges (continued)

- Fostering care-provider involvement
  - Managing changes in the way clinicians and administrative support staff work
- Obtaining consent from a critical mass of users
- Taking into account public opinion with regard to privacy protection
- Fostering the achievement of preliminary work in the regions for implementation of the DSQ.
  - Deploying many workstations
  - Training users
  - Upgrading regional technocentres (24/7, clinical data)
  - Upgrading the telecommunications network (RTSS)
- Respecting scope budget and schedule and Managing expectations
- Ensuring technology integration (interoperability)

Global Plan

Dossier de santé du Québec

Major Milestones

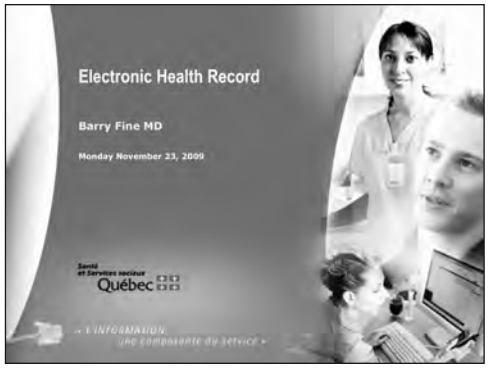
2005	2006	2007-2008	2008-2009	2010.....
Architecture	Competitive Bid Process	Design and Testing	Pilot Project Quebec City	Regional Deployment
Detailed Architecture Planning	- Infrastructure CAIS - QHR - Clinical Drugs	- Adaptation - Development - CAIS, ICP, DSQ, Labos, Médicaments, Imagerie Registres, etc.	- 11 sites - 56 professionals - 28 000 patients	18 regions

Taking better care of you

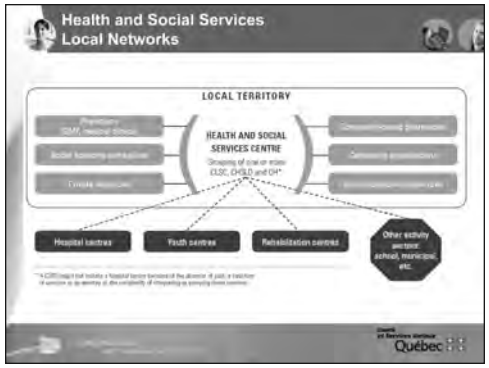
A major project is needed for:

- Patients
- Health professionals
- The regions

Thank you!

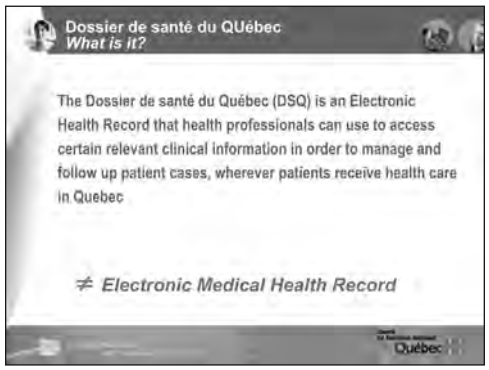

Electronic Health Record
Barry Fine MD
Monday November 23, 2009
Québec

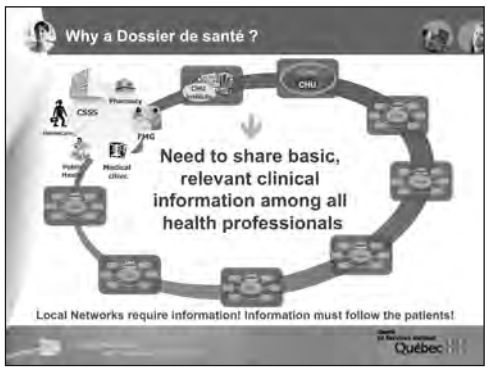

Health and Social Services in Quebec
Québec

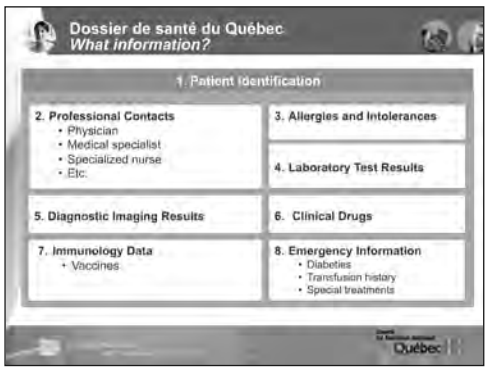

Health and Social Services Local Networks
LOCAL TERRITORY
HEALTH AND SOCIAL SERVICES CENTRE
Hospital centres
Youth centres
Rehabilitation centres
Québec

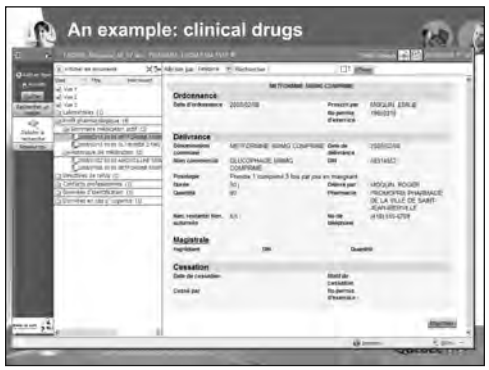

Health and Social Services Governance Structure
MINISTER
Québec

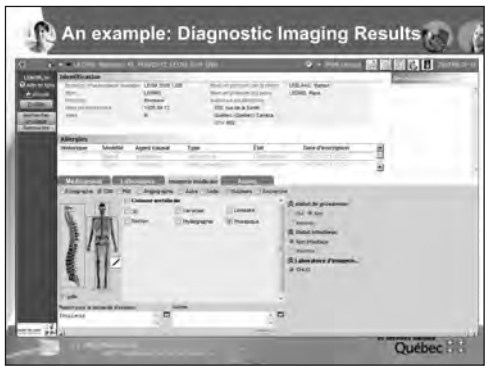

DSQ 101!
Québec


Dossier de santé du QUébec
What is it?
The Dossier de santé du Québec (DSQ) is an Electronic Health Record that health professionals can use to access certain relevant clinical information in order to manage and follow up patient cases, wherever patients receive health care in Quebec
≠ Electronic Medical Health Record
Québec

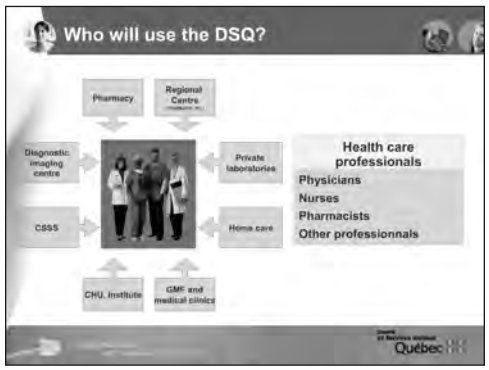

Why a Dossier de santé ?
CHU
CSSS
Pharmacy
Need to share basic, relevant clinical information among all health professionals
Local Networks require information! Information must follow the patients!
Québec

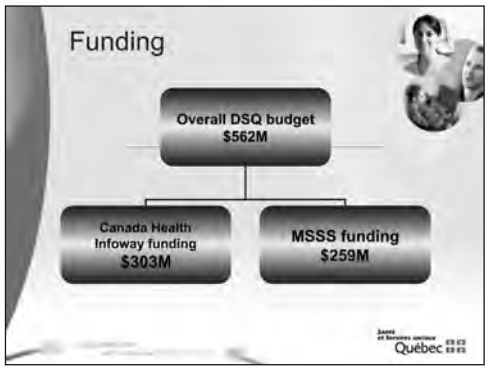

Dossier de santé du Québec
What information?
1. Patient Identification
2. Professional Contacts
• Physician
• Medical specialist
• Specialized nurse
• Etc.
3. Allergies and Intolerances
4. Laboratory Test Results
5. Diagnostic Imaging Results
6. Clinical Drugs
7. Immunology Data
• Vaccines
8. Emergency Information
• Diabetes
• Transfusion history
• Special treatments
Québec


An example: clinical drugs
Ordonnance
Délivrance
Magistrale
Cessation

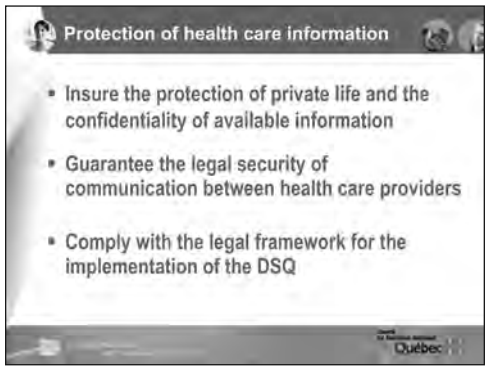

An example: Diagnostic Imaging Results
Québec


Dossier de santé du Québec
For whom?
Any insured person receiving care in Quebec who has not refused to have an EHR
Québec


Who will use the DSQ?
Pharmacy
Regional Centre
Diagnostic imaging centre
Private laboratories
CSSS
Home care
CHU, institute
GMF and medical clinics
Health care professionals
Physicians
Nurses
Pharmacists
Other professionnals
Québec


Funding
Overall DSQ budget $562M
Canada Health Infoway funding $303M
MSSS funding $259M
Québec


LEGAL FRAMEWORK
Québec


Protection of health care information
• Insure the protection of private life and the confidentiality of available information
• Guarantee the legal security of communication between health care providers
• Comply with the legal framework for the implementation of the DSQ
Québec

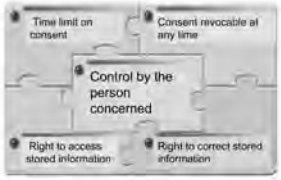
Human rights / consent

Free and informed

Obligation to inform

Limited duration: 5 years

Can be revoked at any time



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Notes

16:00 - 17:00 Effective CME, E-learning

Michael David Rosengarten B.Eng, MD, FRCPSC

Associate Dean of CPHE, Faculty of Medicine, McGill University;
Chair: Standing Committee for CME: AFMC;
Associate Professor of Medicine, McGill University

Luconi Francesca PhD

Professional Associate, Center for Continuing Health Professional Education Faculty of Medicine, McGill University

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Michael David Rosengarten B.Eng, MD, FRCPSC
Associate Dean of CPHE, Faculty of Medicine, McGill University
Chair: Standing Committee for CME: AFMC
Associate Professor of Medicine, McGill University

Research interests: Michael David Rosengarten has a broad technical background. His degree at McGill University in Electrical Engineering included training in information transmission and computer programming, both of which are important in the current context of transmission of material over the web. He is currently the Associate Dean of Continuing Medical Education at McGill.

Luconi Francesca PhD
Professional Associate, Center for Continuing Health Professional Education Faculty of Medicine, McGill University

Learning outcomes: At the end of this workshop, learners will be able to:

a) Describe general trends in continuing medical/professional education

b) Identify strategies to effectively engage in CME/CPD programs

c) Identify principles to select effective CME/CPD programs

d) Discuss how strategies and principles could be applied to specific contexts of clinical practice

Agenda

5' Introduction: Ice-breaker

25' Presentation

- Overview: Trends in face-to-face & online CME/CPD
- Effective strategies for CME/CPD (as learner/as instructor)
- Principles to select effective CME/CPD programs

15' Paired discussion

- Exchange experiences in attending CME/CPD programs

15' Whole group discussion: Conclusions

Major take-home messages

- Family Physicians are increasingly under pressure due to new challenges and emerging simultaneous trends in the health care system.
- CME/CPD is under scrutiny for its lack of impact in clinical practice. A reform is underway in order to ensure quality and knowledge translation.
- CME/CPD can play a supportive role beyond accreditation requirements.
- Strategies to engage in CME/CPD: Readiness to learn, self directed learning and mindful practice
- Effective theory-driven CME/CPD programs: Who is the provider? What type of instructional methods and techniques are used to ensure acquisition/confirmation, retention and application of knowledge and/or skills?

Journal Readings & Resources

Epstein, R. M. (2009). Mindful practice. JAMA. 282(9):833-839.

Fung-Kee-Fung, M. et al. (2009). Regional Collaborations as a Tool for Quality Improvements in Surgery. A Systematic Review of the Literature. Annals of Surgery 249(4): 565-572.

Davis, D., et al. (2009). The Science of Continuing Medical Education. Available at:

http://chestjournal.chestpubs.org/content/135/3_suppl/8S.full.html

Moore, D. E. (2007). How physicians learn and how to design learning experiences from them: An approach based on an interpretive review of evidence. In S. Fletcher (Ed.), Proceedings of the Macy Conference on Continuing Education in the Health Professions. Nov 28-Dec 1, Southampton, Bermuda, 1-44.

Straus, S. & Graham, I. (2009) Knowledge Translation in Health Care: moving from evidence to practice. Hoboken: NJ: Wiley-Blackwell/BMJ.

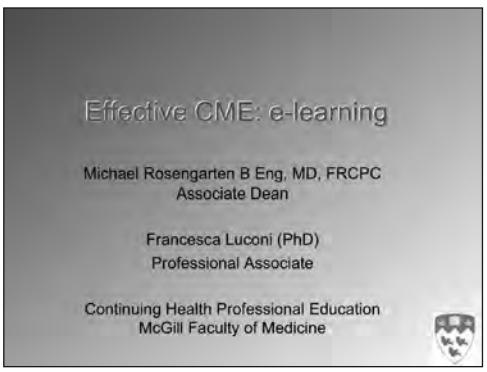
Online accredited CME university providers in Canada

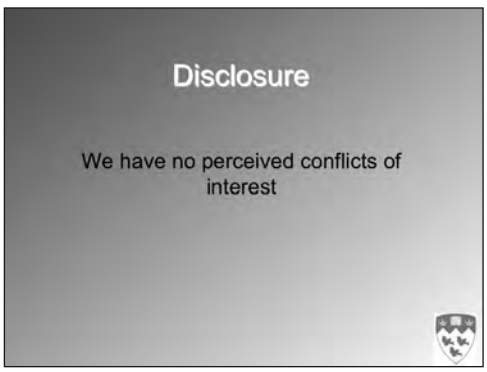
Web Portal of the Canadian Medical Schools: http://www.university-cme.ca/

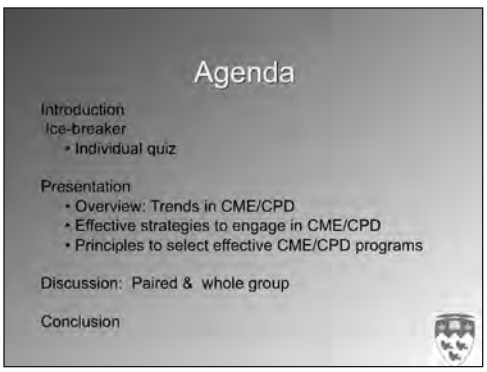
McGill CME: http://cme.med.mcgill.ca

Annotated list of online CME in the US and Canada by Dr Sklar (MD MS). Available at:

http://www.cmelist.com/list.htm







Voltaire

Doctors are men
who prescribe medicines of which they know little
to cure diseases of which they know less
in human beings of whom they know nothing.

Voltaire (1694 - 1778)

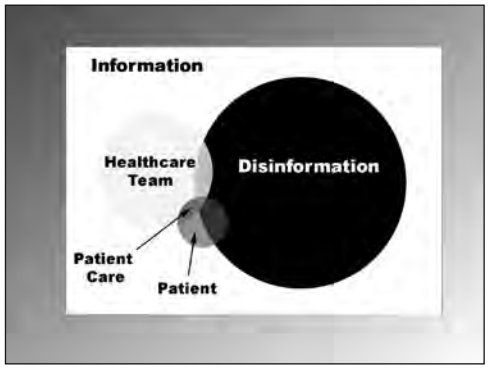
Jerome Kassirer

In all probability, 90% of what you learned in medical school will be proved wrong in 5 years.

Jerome P. Kassirer, M.D., On The Take: How America's complicity with Big business can endanger your health. Oxford University Press 2005

Current trends

- Explosion in medical knowledge & information technology
- Greater demands for accountability & transparency to achieve health care quality
- Expectations inter-professional team work
- Limited resources



The story of Anne

At the breakfast table

Anne, a university professor, sat at our breakfast table. Each of us had freshly poured orange juice, but only Anne's glass did not have a calcium pill beside it.

Odd, as Anne was over 65, and as all who have seen Gustav Klimt's

Three Ages of Woman

realize, osteoporosis is a very real concern for older women.

The Question

I asked:

Do you know if your bones are strong?

Have you had a bone density test?

Anne said no, her doctor said that it was not indicated.

Fact Check

Everyone over the age of 65 should have a bone density test.

2005 GPAC: Guidelines and Protocols Advisory Committee

Quality of Care

It has been estimated for 2008 that:

30 - 40% patients did not get treatments of proven effectiveness.

20 - 25% patients got care that was not needed or potentially harmful.

Conference: Doctors, Decision Makers, and Optimal Prescribing. 2009 Ottawa

How and what do we learn?

What about the Information in the large white box?

The organizational structures of web contents and electronic information resources must adapt to the demands of a growing volume of information and user requirements.

Otherwise the information society will be threatened by disinformation.

Hersh W, Crowley RS, Miller J, Nyhan M. **Dealing with an information overload of health science data:** structured utilization of libraries, distributed knowledge in databases and Web content. Stud Health Technol Inform. 2006;124:549-54.

Young doctors learn bad habits

Tom Blackwell, National Post Monday, March 23, 2009

- Medical students and residents at an Alberta hospital were using a faulty technique for inserting ET tubes.
- Many said they had learned the procedure from watching medical dramas.
- An analysis of the show *ER* revealed its fictional MDs and nurses performed intubations incorrectly almost every time.

Tom Blackwell, National Post Monday, March 23, 2009

Disinformation

Conventional medicine's explanations of HIV and AIDS are a medical myth at best; and outright quackery at worst.

There is no such thing as a virus that "causes" AIDS.....

http://www.NaturalNews.com

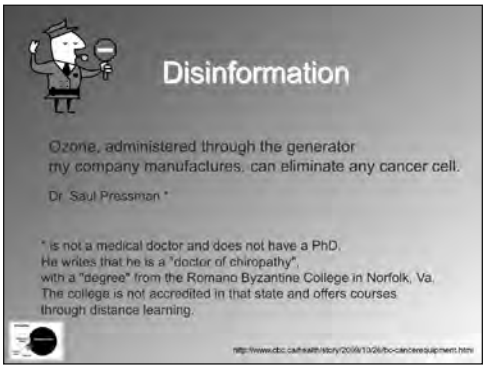
Disinformation

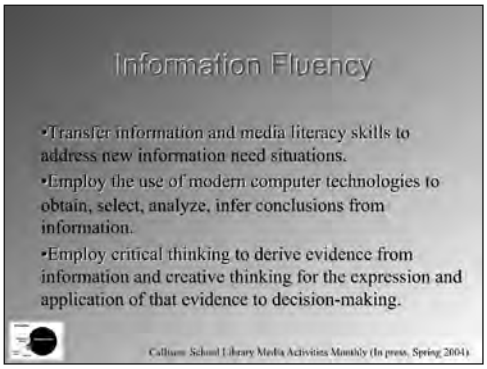
... both the FDA and CDC are pushing a collection of swine flu vaccines that have never been subjected to any scientific testing to determine whether they actually work.

These vaccines are, in technical terms, a "hoax."

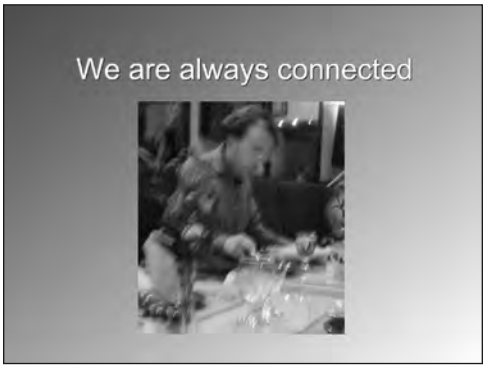
Lacking any scientific evidence of efficacy, they are a scam that should be avoided by all health consumers.

http://www.NaturalNews.com

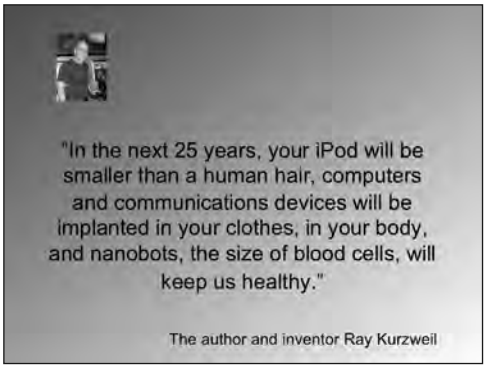

Disinformation
Ozone, administered through the generator my company manufactures, can eliminate any cancer cell.
Dr. Saul Pressman *
* is not a medical doctor and does not have a PhD. He writes that he is a "doctor of chiropathy", with a "degree" from the Romano Byzantine College in Norfolk, Va. The college is not accredited in that state and offers courses through distance learning.


Information Fluency
•Transfer information and media literacy skills to address new information need situations.
•Employ the use of modern computer technologies to obtain, select, analyze, infer conclusions from information.
•Employ critical thinking to derive evidence from information and creative thinking for the expression and application of that evidence to decision-making.
Calhoun School Library Media Activities Monthly (In press, Spring 2004)

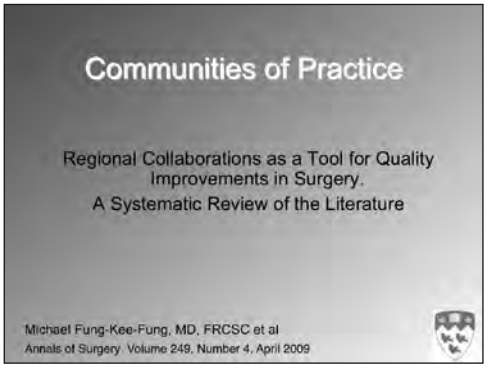

The library card index room at UBC has changed!

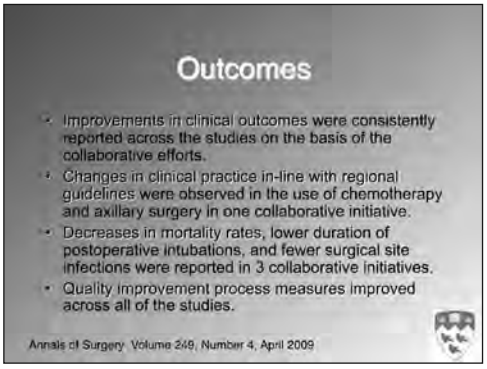

We are always connected


We have new ways to relax in the country


"In the next 25 years, your iPod will be smaller than a human hair, computers and communications devices will be implanted in your clothes, in your body, and nanobots, the size of blood cells, will keep us healthy."
The author and inventor Ray Kurzweil

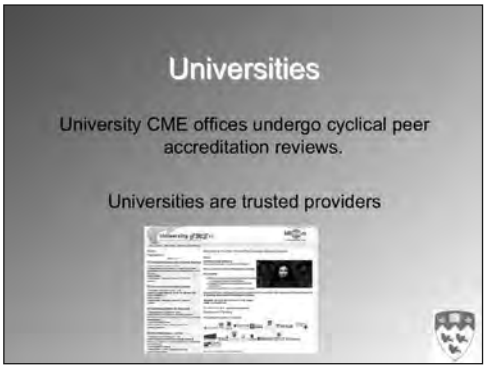

Maintaining competence.
"An effective method to maintain the level of a practice is to work as a group"
Dr. Martin Dawes: 2003


Communities of Practice
Regional Collaborations as a Tool for Quality Improvements in Surgery.
A Systematic Review of the Literature
Michael Fung-Kee-Fung, MD, FRCSC et al
Annals of Surgery Volume 249, Number 4, April 2009


Outcomes
Improvements in clinical outcomes were consistently reported across the studies on the basis of the collaborative efforts.
Changes in clinical practice in-line with regional guidelines were observed in the use of chemotherapy and axillary surgery in one collaborative initiative.
Decreases in mortality rates, lower duration of postoperative intubations, and fewer surgical site infections were reported in 3 collaborative initiatives.
Quality improvement process measures improved across all of the studies.
Annals of Surgery Volume 249, Number 4, April 2009


The Trusted Provider
Who should be a trusted provider, who should filter and guide healthcare workers to good content?


The Trusted Provider
A provider that goes through a peer review accreditation process and is accountable for enforcing the norms of accreditation and evidence-based care.


Universities
University CME offices undergo cyclical peer accreditation reviews.
Universities are trusted providers


>300 Accredited events


The McGill Rural Web Series
McGill CCHPE


Delivery Worldwide

Trends

Effective healthcare will be delivered by collaborative evidence-based inter-professional teams enabled by technology.

CME

- What role does CME play in your profession?

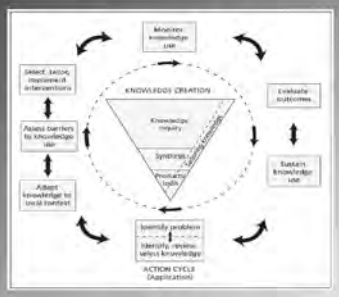
CME: Learning process

Any action designed for or performed by a physician for the purpose of acquiring, maintaining or upgrading knowledge, skills, or attitudes to improve the quality of the health care that the physician dispenses to patients.

(CMECQ, 2003 p. 5)

CME under scrutiny

- Not fulfilling the evolving professional needs of physicians
- Limited/non-existent impact on clinical practice
- Knowledge Translation: application of research & process of decision-making



The Knowledge to Action Framework (Straus et al. 2009)

CME effectiveness

- Multiple factors influence effectiveness to change professional behavior
- No definitive conclusions regarding the influence of particular factors
- Research limited due to the small number & heterogeneity of available studies.

(Davis et al. American College of Chest Physicians 2009)

Instructional model revisited

Traditional CME	Recommended CME
Teacher-centered	Student-centered
Didactic	Distributed learning
Single exposure	Multiple exposures (interactivity)
Removed from practice	Embedded in practice (point of care)
One level evaluation	Multiple levels of evaluation
Less effective in changing practice	More effective in changing practice
Needs: Medical knowledge/skills	Broader lifelong learning needs New emergency diseases Chronic disease management

Outcome-based CME evaluation

- Participation
- Satisfaction
- Learning
- Competence
- Performance
- Patient-health
- Population-health

(Moore, D. 2007)

Strategies to select CME

Principles adult educ	CME formal
• Activeness	• Interactivity
• Relevance	• Real cases
• Identify needs	• Tailored to needs
• Occasion to practice	• Simulated activities
• SDL support	• Access best eviden
• Feedback	• Peer consultation
• Reflection	• Impact practice

Learning & change model

- Learning : relevant force to influence change in physicians' practice
  - Personal, social, professional forces
  - Drive to achieve competent performance in patient care

(Fox, Mazmanian & Putnam, 1994)

Four-stage theory physicians' learning

Slotnick, & Shersheva, 2000

Learning is situated in context

Four-stage Learning Theory

- Scanning: exploring without immediate need
- Evaluation: relevance of the problem
- Learning: acquire knowledge to solve problem
- Gaining experience: apply newly acquired knowledge & skills

Slotnick, & Shersheva, 2000

Strategies : Readiness to learn

Statement	Stage
The way I diagnose AD is acceptable	Confirmation of knowledge/skills
I am dissatisfied with AD dx	Scanning
I need to learn new screening tech	Learning
I might change some aspects of AD diagnostic practice	
I plan to change the way I diagnose	
I gradually changed my practice	Gaining experience

- Educational Process (PRECEED)

Green & Kreuter, 2005

Knowledge Translation Tools

- Active educational interventions
  - Outreach visits, academic detailing
  - Quality circles of professionals
  - Active self-study materials
- Professional interventions (Prevention & test ordering)
  - Reminder systems (reduce cognitive loads)
  - Decision support

Trends

Effective healthcare will be delivered by collaborative evidence-based inter-professional teams enabled by technology.

E-learning

- Use of new multimedia & Internet technologies to enhance knowledge & performance
- North-America in 2008: 30% of CME
- Effectiveness: Equivalent to traditional methods
- Complementary traditional instruction
- Theory-driven instead of technology-driven
- Not a panacea: advantages/disadvantages
- Profile ideal learner: self-directed

Ruiz, Mintzer, Leipzig, 2006; Cook, 2009

E-learning advantages

- Flexibility: location & time (rural remote regions)
- Individualized instruction: e.g. portfolios
- Learner's control over content, pace of learning
- Opportunities for meta-cognitive strategies
- Community of practice
- Written discourse: controversial topics activates articulation and argumentation

E-learning disadvantages

- Social isolation
- Up-front costs
- Technical problems
- Loss of interest due to time-lag posting during collaborative online discussions
- Disorientation & information overload

Self-directed Learning Skills

- Live-long learning
- Defining learning needs
- Determining appropriate learning resource
- Using resource effectively
- Evaluating the accuracy & value of the info
- Recording or filing info for future reference
- Applying what has been learned to clinical practice

Self-assessment

Individualized learning strategy
Critical self-reflection: articulate knowledge gap
Difficult task for learners in general, most physicians

Context for self-assessment
Small group interaction in asynchronous forum

"The course has made me more aware of….whereas before I would have lumped them all as AD. Now, I don't put Frontal Lobe on cholinesterase inhibitors" (Ronald, PL2-570)

(Barrows, 1994; Luconi 2008)

Self-assessment tool

- METRIC: Innovative online tool to improve practice
  - Measuring
  - Evaluating
  - Translating
  - Research
  - Into
  - Care

  7 conditions (e.g. asthma, COPD, CAD, diabetes)
- American Association of Family Physicians

Conclusions Effective CME

- E-learning : Not a panacea depending on how it is has been designed and delivered
- Theory-driven design to support learning & performance
- Strategies to select CME

Take away messages

- Be a critical user of CME/CPD
- E-learning complements but does not replace traditional live methods

Online resources

- Web Portal of the Canadian Medical Schools: 17 Medical schools in Canada:
- Medscape: multiple resources
- Guidelines: Best available practice involving physician & patient ; www.agree.collaboration.org
- Guidelines: adaptation to local context
- To promote optimal drug therapy: Rx for Change

Online resources

- InfoPOEMS: (Patient Oriented Evidence that Matters) are one-page synopses of original clinical research that have been delivered as daily e-mails to physician subscribers of the service through cma.ca since 2005.
- Courriels Cochrane: consistent en synopsis des revues de littérature systématiques Cochrane les plus pertinentes pour les soins de première ligne.

CME in the US

Accreditation Council of CME

- 100, 898 accredited CME activities
- 769, 439 hours of instruction
- Registration : > 10 million of physicians & 6 million non-physicians
- Commercial support decreased (14%) (approx 200 million)

ACCME Annual Report 2008

16:00 - 17:00 Use of Diet & Excercise in Health Promotion in Teenagers

Alan Pavilanis MD, CM, CCFP, FCFP, DipEpi
Director, Family Medicine Centre, St. Mary's Hospital Centre;
Associate Professor, Family Medicine, McGill University

Notes

Notes

Tuesday, Nov. 24 – Breakfast Symposium

07:00 - 07:45 Breakfast Satellite Symposium

Chair • **Najmi Nazerali**

Preventing Cardiovascular Disease in Patients with Diabetes

Sven Wassmann MD, McGill University

Supported through an unrestricted educational grant from Boehringer-Ingelheim.

Notes

Notes

08:00 - 08:30 Pediatric Allergies

Reza Alizadehfar
Division of Allergy and Clinical Immunology, The Montreal Children's Hospital, MUHC

Research Interest: I am a pediatrician. I finished my training in clinical immunology and allergy at McGill University and I completed a fellowship in primary immunodeficiency and bone marrow transplantation at the Sick Kids Hospital in Toronto.

I am working currently at the Montreal Children's Hospital in the McGill network, where I run the primary immunodeficiency clinic as well as an allergy and asthma clinic. I also contribute to teaching and research in these 3 fields.

In addition I have recently joined the Montreal General Hospital where I see adults with primary immune disorders.

I am the assistant director of clinical immunology laboratory of the Montreal Children's Hospital and co-director of the Jeffery Modell Canadian Primary Immunodeficiency Network.

Notes

Tuesday, Nov. 24 – Morning Plenary

08:30 - 09:00 Back Pain

Mohan Radhakrishna MD, FRCPC
Assistant Professor, Division of Physical Medicine and Rehabilitation,
McGill University and Montreal General Hospital

Acute Low Back Pain Primer in 30 minutes

Mohan Radhakrishna, MD, FRCPC,
Dip. Sport Med
Physical Medicine and Rehabilitation

Objectives

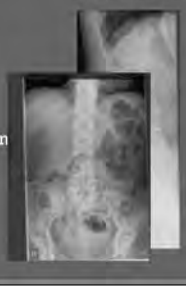
- Describe the epidemiology of low back pain
- Develop a classification system for low back pain
- Name the factors pre-disposing to chronicity
- Become familiar with the CLIP program

What we won't talk about:

- Radiculopathy
- Spinal Stenosis
- Fracture
- Osteoporosis

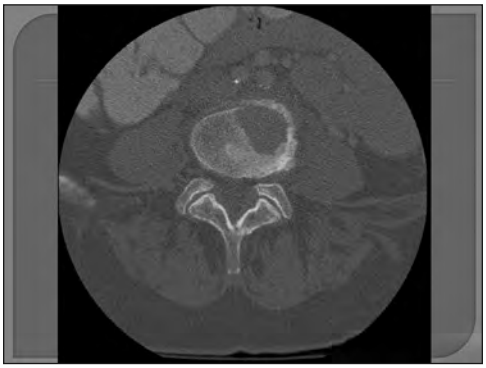
A real case

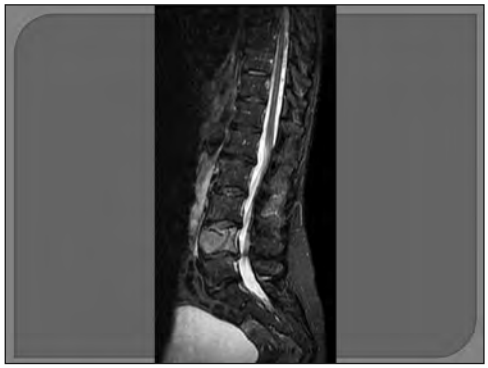
- 54 year old female
- Lifting laundry at home 4 weeks ago
- No leg irradiation
- Still wants to work
- Smoker, hypertension
- Exam normal

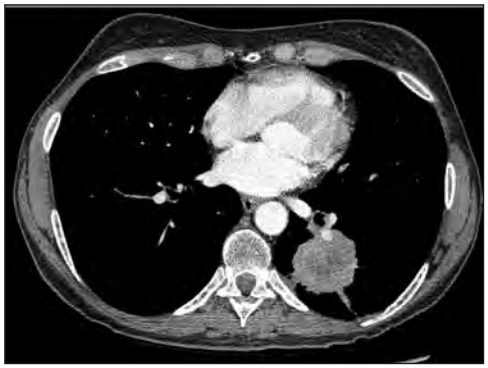


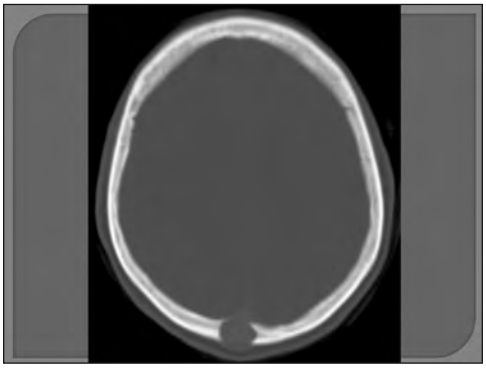
Follow-up 4 weeks later

- Cough
- Weight loss
- Back pain still present









What did the MD do right?

- Follow-up

Reassurance not dismissal

Education: Just the facts

Epidemiology

- 20% point prevalence
- 1 year prevalence of 50%
- Lifetime prevalence of 80%

- 7% chronicity rate
- 1% surgical rate

Managing the patient with low back pain

Definition: Acute low back pain

- Back pain
  - With or without leg radiation
  - Less than three months in duration
  - Limits activity

Bigos SJ et al. Acute Low Back Pain Problems in Adults
Clinical Practice Guideline No. 14.
AHCPR Publication No. 95-0642. Rockville, MD:
Agency for Health Care Policy & Research. 1994.

Mechanical Mimics

History

- Classify symptoms by category
- Detect grave pathologies
- Establish a good doctor-patient relationship
- Clarify patients expectations
- Guides the physician re: psychological and socio-economical factors
- Clarifies patient's threshold for pain and inactivity

Classification Bigos et al.

More useful in everyday practice
Incomplete ?

Bigos SJ et al. Acute Low Back Pain Problems in Adults. Clinical Practice Guideline No. 14 AHCPR Publication No. 95-0642. Rockville, MD: Agency for Health Care Policy & Research, 1994.

Modified Classification

More realistic ?
Too complicated ?

Modified Classification

More realistic ?
Too complicated ?

Practical Classification

Used by primary care physician
and spine specialists

Eliminating grave spinal pathologies

- Back pain
- Bilateral leg pain
- Bilateral leg weakness
- Saddle anaesthesia
- Fecal or urinary dysfunction
- Incidence (less than 1%)

Eliminating grave spinal pathologies

- Back pain
- Focalized pain
- Well defined severe trauma
- Minor trauma - osteoporosis
- Spondylolysis, spondylolisthesis

Eliminating grave spinal pathologies

- Back pain
- Prior surgical intervention/spine injection
- Recent bacterial infection
- IV drug use
- Fever, chills
- Severe night time pain

Eliminating grave spinal pathologies

- Back pain
- Patient > 50 years old
- Prior history of cancer
- Fever, chills
- Weight loss
- Severe night time pain (pain at rest)

Eliminating grave non-spinal pathologies

- Perforated gastric / duodenal ulcer
- Acute pancreatitis
- Appendicitis (retrocecal)
- Renal colic
- Pyelonephritis
- Ruptured ectopic pregnancy
- Endometriosis

Eliminating grave non-spinal pathologies

- Dissecting abdominal aortic aneurysm
  - Back pain
  - Male patient > 50 years
  - PMHx: HTN, vasculopathy, anti-coagulants
  - Associated abdominal pain
  - Shock
  - Syncope

Eliminating inflammatory pathologies

- Other involved joints
- Joint effusions
- Skin changes
- Morning stiffness
- Response to NSAIDS
- History of eye / intestinal infections, STIs

Projet CLIP

- Literature review and practice management suggestions
- http://www.santepub-mtl.qc.ca/Publication/pdftravail/CLIPenglish.pdf

Predicting Chronicity

- For patients with no history of LBP:
- Psychologic state: kinesiophobia, catastrophization, depression
- Clinical: pain intensity, radiation below knee, perceived health and disability
- Psychosocial factors

Predicting Chronicity

- For those with history of LBP, recurrence will occur in:
- Poor general health
- Prior episodes of LBP
- Job (in)satisfaction

Predicting Chronicity

- Less strong associations
- Professional status
- Salary
- Social contacts, sense of indemnification.

When chronicity is just around the corner..

- Use your team resources well....
- Make sure team members preach what you want them to practice

The TEAM

- PT
- OT
- Social Worker
- Psychology
- Dietician
- Vocational counsellor

Conclusion

- Acute management of low back pain focuses on 4 things
- Screen out dangerous causes
- Screen for chronicity
- Manage symptoms
- Educate the patient

Tuesday, Nov. 24 – Morning Plenary

09:00 - 09:30 CMPA

Ross Berringer MD, D(ABEM), MCFP(EM)
Physician Risk Manager, Risk Management Services,
Canadian Medical Protective Association

Research Interests: Dr. Ross Berringer graduated from the University of British Columbia in 1980. After completion of a rotating internship at St. Paul's Hospital in Vancouver, he was a general practitioner in 100 Mile House from 1981-83. During the ensuing two years, he completed a residency in emergency medicine in Jacksonville, Florida. He holds a certificate of special competency in emergency medicine and is a diplomate of the American Board of Emergency Medicine. From 1985 through 2006, he practised full time emergency medicine at St. Paul's Hospital achieving the rank of Clinical Associate Professor. In addition, he was the Medical Director for Vancouver Fire and Rescue Services and the Advanced Life Support Medical Advisor for the BC Ambulance Service. His research interests have been in pre-hospital care and out-of-hospital cardiac arrest. In May of 2006 he joined the CMPA as a Physician Risk Manager.

Notes

Tuesday, Nov. 24 – Workshop D-01

09:00 - 10:30 ER: Psychiatric Emergencies

Hani Iskandar MD

Medical Chief, Intensive Care Unit, Emergency, Brief Intervention Unit,
Electroconvulsive Therapy Unit, Douglas Institute;
Coordinator, Continuing Medical Education, Douglas Institute;
Associate Professor, Department of Psychiatry, McGill University

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Research Interests: Hani Iskandar, MD, joined the Douglas in 1982 as a psychiatry resident. As a Douglas Institute clinician and researcher, he has been involved in clinical research projects in the fields of schizophrenia, mood disorders and pharmacology (particularly in relation to antipsychotics and antidepressants). Since May 2009, he has been the Coordinator of Undergraduate Medical Education in the Department of Psychiatry at McGill University.

An active McGill professor, he is a multiple recipient of the Best Teacher Award, an award of excellence in post-graduate teaching given out by students. He also gives lectures and classes for medical students, psychiatry residents, family doctors and fellow psychiatrists.

Notes

09:30 - 10:30 GER: Behavioral Problems in Elderly

Michel Élie MD, FRCP(C)

Assistant Professor, Department of Psychiatry, McGill University;
Director, Division of Geriatric Psychiatry, St. Mary's Hospital Centre;
Associate Member, Department of Clinical Epidemiology and Community Studies, St. Mary's Hospital Centre

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Objectives

1. To help the participants to better diagnose behavioural and psychological symptoms associated with dementia.
2. To help the participants to better treat behavioral and psychological symptoms of dementia.

Behavioral and Psychological Symptoms of Dementia:

A Review

With the aging of the population, the prevalence of dementia will increase. The presence of behavioral and psychological symptoms is extremely frequent with this disorder and can be devastating for the patient and their caregivers. The goals of this lecture will be to help the participants to identify these symptoms, consider underlying different causes for these behaviours and develop a therapeutic approach to manage them

Major take home messages

- Behavioral and Psychological symptoms are frequent in dementia.
- Its management includes looking at different causes and having a holistic approach

Reference

Canadian coalition for Seniors' Mental Health (2006) National Guidelines for Seniors' Mental Health: The assessment and treatment of Mental Health issues in long term care homes (focus on mood and behavior symptoms) Toronto: Canadian Coalition for Seniors' Mental health. www.ccsmh.ca

Notes

09:30 - 10:30 PEDS: Wheezing Child

Reza Alizadehfar

Division of Allergy and Clinical Immunology,
The Montreal Children's Hospital, MUHC

Research Interest: I am a pediatrician. I finished my training in clinical immunology and allergy at McGill University and I completed a fellowship in primary immunodeficiency and bone marrow transplantation at the Sick Kids Hospital in Toronto.

I am working currently at the Montreal Children's Hospital in the McGill network, where I run the primary immunodeficiency clinic as well as an allergy and asthma clinic. I also contribute to teaching and research in these 3 fields.

In addition I have recently joined the Montreal General Hospital where I see adults with primary immune disorders.

I am the assistant director of clinical immunology laboratory of the Montreal Children's Hospital and co-director of the Jeffery Modell Canadian primary immunodeficiency Network.

Notes

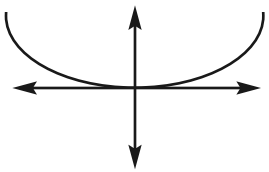
Tuesday, Nov. 24 – Workshop D-04

09:30 - 10:30 Hands On : Back Exam

Mohan Radhakrishna MD, FRCPC
Assistant Professor, Division of Physical Medicine and Rehabilitation,
McGill University and Montreal General Hospital

Inspection

- Begins from when patient is called from waiting room
- Observe for abnormal gait, sitting posture, behavior indicating level of suffering, spontaneous movement during history-taking
- Ensure patient is adequately undressed



Standing:

- Fortin finger sign for sacroiliac joint pain
- Assess curvature: kyphosis, scoliosis
- Leg length discrepancy (buttock and popliteal creases)
- Ask the patient to bend forwards: postural scoliosis resolves, a structural scoliosis does not disappear
- (Modified)Schober's test
  - When the spine flexes, the distance between each pair of vertebral spines increases.
  - In the Schober's test, a tape with a 10 cm mark is placed vertically in the midline upwards from the level of the dimples at the level of the posterior superior iliac spines). Mark the skin at 0 and at 10 cm and then ask the patient to flex as far forward as they can.
    Record where the 10 cm mark on the skin strikes the tape. The increased distance along the tape is due only to flexion of the lumbar spine and is normally about 6-7 cm (less than 5 cm should be considered as abnormal).
- Ask the patient to extend their lower back.
- Ask the patient to move laterally (sideflex to touch knee).
- Look for any other abnormalities, e.g. cafe-au-lait spots, which may suggest neurofibromatosis, a fat pad or hairy patch suggestive of spina bifida, or scarring suggestive of spinal surgery.
- Unipedal stance—look for contralateral iliac crest to drop (sign of weak ipsilateral hip abductors)
- Have patient squat to screen range of motion in hips, knees, ankles plus kinesiophobia.
- Functional overlay (Waddell's Signs)
  - Hip rotation
  - Axial loading: apply pressure to the head. Overlay is suggested if this aggravates the back pain.

Sitting

- Myotomes:
  - L2 and L3: resisted flexion of the hip
  - L3: resisted knee extension
  - L4: resisted dorsiflexion of the ankle
  - L5: resisted extension of the big toe
  - S1: resisted toe flexion

  NB: Need to test ankle plantarflexion with repeated heel raises

  NB: Giveway weakness versus true neurologic weakness

- Dermatomes: test sensation to pinprick:
  - L2: mid thigh
  - L3: medial knee
  - L4: medial aspect of the ankle
  - L5: 1st webspace
  - S1: lateral aspect of the foot, the heel and most of the sole
  - S2: posterior aspect of the knee
  - S3: ischial tuberosity
  - S4, S5 concentric rings around the anus

- Reflexes:
  - Patellar tendon L4
  - Medial hamstrings or tibialis posterior L5
  - Achille's reflex S1
  - Plantar response
- Seated straight leg raise
  - Tripod
  - Slump

Lumbar rotation

Supine

- Straight leg raising:
  - Passively flex hip with knee straight while patient is supine. Pain in leg and back between 30-70o implies

    neural involvement. If positive lower leg until symptoms better then dorsiflex ankle. Stop when the patient

    complains of back or leg pain (hamstring tightness is not relevant). The test is negative if there is no pain.

    Paresthesia or pain in root distribution is very significant, indicating nerve root irritation.
  - Repeat on other side. A positive crossed SLR is highly specific.
- Femoral stretch test:
  - With the patient prone and the anterior thigh fixed to the couch, flex each knee in turn.

    This causes pain in the appropriate distributions by stretching the femoral nerve roots in L2-4.
  - The pain produced is normally aggravated by extension of the hip.
  - The test is positive if pain is felt in the anterior compartment of thigh.
- Hip Range of Motion
- Sacroiliac joint tests: Highly non-specific.
- Abdominal exam as required
- Bridge evaluation for core muscle strength

Prone

Palpation

- Check for bone tenderness of the spine: local tenderness may indicate serious pathology such as infection, fracture or malignancy.
- Look for involuntary muscle splinting.
- A palpable step at the lumbosacral junction may indicate spondylolisthesis.
- In prone position can load the spinal column by applying pressure through both hands.

  There should be some movement. Anklyosing spondylitis when advanced has no give.
- Look for diffuse tenderness/fibromyalgia

References

David Magee: Orthopedic Physical Assessment

Stanley Hoppenfeld: Physical Examination of the Spine and Extremities

Stuart McGill: Low Back Disorders

Tuesday, Nov. 24 – Workshop D-05

09:30 - 10:30 CMPA - Obligation of Reporting, Suicide, Homicide

Ross Berringer MD, D(ABEM), MCFP(EM)

Physician Risk Manager, Risk Management Services,
Canadian Medical Protective Association

Research Interests: Dr. Ross Berringer graduated from the University of British Columbia in 1980. After completion of a rotating internship at St. Paul's Hospital in Vancouver, he was a general practitioner in 100 Mile House from 1981-83. During the ensuing two years, he completed a residency in emergency medicine in Jacksonville, Florida. He holds a certificate of special competency in emergency medicine and is a diplomate of the American Board of Emergency Medicine. From 1985 through 2006, he practised full time emergency medicine at St. Paul's Hospital achieving the rank of Clinical Associate Professor. In addition, he was the Medical Director for Vancouver Fire and Rescue Services and the Advanced Life Support Medical Advisor for the BC Ambulance Service. His research interests have been in pre-hospital care and out-of-hospital cardiac arrest. In May of 2006 he joined the CMPA as a Physician Risk Manager.

Notes

Tuesday, Nov. 24 – Workshop D-06

09:30 - 10:30 Addictions

John Sader MD, BSc, ASAM certified
Assistant Medical Director, Clinique du Nouveau Départ;
Affiliate Professor, Department of Family Medicine, McGill University

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Research interests: Dr Sader is a family physician also trained and certified by the American Society of Addiction Medicine since 1992.

He has been working with patients challenged by all sorts of dependencies from alcohol and drugs to eating disorders, gambling and sex addiction.

He is especially interested in the often present psychiatric comorbidities that accompany dependencies: anxiety disorders, depressive disorders and attention-deficit disorders to name a few.

In the past, he has worked at a private treatment centre and was Assistant Medical Director for over 12 years of the 16 spent there.

He has since moved on and recieves his patients in the context of a FMG at la Clinique 1851 as well as being the main physician for The Baca Health Group for eating disorders.

Over the years, he has developped an expanded model of Generalized Anxiety Disorders that include 15 different sub-types and which infers new alternatives for treatment both pharmacologically, psychologically and behaviorally.

Over the years, his experience has led him to develop a keen interest in tying- in principals relating to the physics of matter with those of the spiritual-immaterial-existential and how those intersections may relate to happiness.

The NiCE-Q;

Nicotine Co-Morbidity Evaluation Questionnaire,

A New Tool for Tobacco Cessation

John P. Sader MD, ASAM certified, ABAM diplomate
Clinique médicale 1851-GMF
November 2009
McGill 60th Annual Refresher Course for Family Physicians

Disclosure Form

- Active affiliations over recent years with the following companies:
- Wyeth Pharmaceuticals: 2005-2006 (conferences)
- Janssen-Ortho inc: 2007 – to present (conferences)
- Shire Biochem: 2007 – to present (conferences)
- Pfizer: 2008 – to present (conferences/Special Study Groups)
- Egothera inc.: 1999 – to present

What is todays reality?

- Many of the statistics concenring smoking are eloquent and useful (especially those concerning medical consequences) HOWEVER the statistics that relate to general or psychological characteristics of smokers are less and less pertinent to todays smoker because they were collected with a different population of smokers:
- In 1995: 39% of the adult Quebec population were smokers.
- In 2008: only 23% of this same population still smokes.
- Very important headway.
- So important in fact, that we must be very vigilant not to accord too much importance to certain statistics that may be based on another population altogether.

How so?

- In 1995: studies pertaining to psychiatric co-morbidity associated with smoking demonstrated rates of co-morbidity in the order of 40-45%.
- 39% smokers times 40% =’s 16 ψpersons / 39 smokers.
- Presently, we have approximately 23% of adults who are active smokers.
- Considering that nothing has been done to target and help the smokers with underlying psychiatric co-morbidity it should be easy to see that we can project that we now have a population of smokers with
- 16 ψpersons/23 smokers! or **75% psychiatric co-morbidity**

This should change everything

- The ‘Law of Diminishing Returns’ describes very well what clinicians are seeing every day:
- We succeeded in stopping the easy ones
- We have having less and less success with the more difficult ones AND:
  - They are getting discouraged and
  - We are getting discouraged and
- Those who are succeeding are doing so short term and are suffering from the emergence of psychiatric conditions that were contained while they continued to smoke.

What are the associated co-morbidities that we should expect to come across?

- ADD/H
- Anxiety disorders of all types
- Affective Disorders of all types including BP
- Schizophenia
- Antisocial personality
- Alcohol and drug dependency
- Pathological gambling

Tobacco and its co-morbidities

- 88 % of Schizophrenics
- 70 % of Bipolar Disorders
- 49 % of Depressives
- 47 % of Anxiety Disorders
- 46 % of Personality Disorders
- 45 % of Adjustment Disorders
- ? % of ADD/H Disorders
- SMOKE vs 30 % of controls
  - Hughes et al 1986

How can we explain this neurochemically?

- Smoking increases the levels of monoamines in the brain including Dopamine, Nor-Adrenalin, Acetylcholine and Histamine (all excitatory) as well as Serotonin (more calming).
- Some of these effects are direct and others occur through the action of Monoamine Oxydase inhibition.
  - Monoamine oxydase inhibition by smoking =’s 70% efficacy of Parnate-Nardil
    - Thus a useful effect for for Anxiety disorder patients as well as Depressed patients.
  - Dopamine – May have a positive effect for ADD/H and antisocial personality
  - Molecule ‘X’ associated with smoking appears to very helpful for schizophrenics by improving **‘gating’** thus decreasing paranoia-type symptoms. This means that the schizophrenic smoker who quits faces a double-whammy with conflicting effects: not only may he be more paranoid but because of the induction of the cytochrome system that regresses after he stops smoking, his antipsychotic medication might need lowering and/or his anticholinergique medication increasing! Close follow-up is suggested to ensure the proper adjustements are to meds are made.

The Medical Model of Dependency: A Brain Disease

- A primary and chronic disease whose development is influenced by genetic, psychosocial and environmental factors.
- Often Progressive and Fatal.
- Caracteristic elements are:
  - **1. Loss of control**
  - **2. Use despite associated problems**
  - **3. Excessive preeoccupation**
  - **4. Denial**
- Smoking fits this model

Etiology I

- Multi-factorial:
  - Genetics: studies ++++
    - The relative risk increases 3-4 fold for the children of alcohol-dependant persons
    - **Twin studies:**
      - **concordance MZ = 60%**
      - **concordance DZ = 30%**
    - **Adoption** – The risk increases with the alcohol status of the biological father **BUT** an adoptive father who is alcohol-dependant increases the risk of **intentional avoidance** of alcohol thus decreasing his risk relative to the boy adopted into a non-alcohol-dependant family.
- **Generally genetic weighting = 60%** and other = 40%
- **BUT,** we should consider this a **low estimate** because other forms of dependency (process dependencies) were not considered in these studies.

Etiology 2

- What do they inherit?
  - Often (but not always) a polymorphism at the level of the post-synaptic Dopamine receptor that results in a state of chronic hypo-stimulation of their pleasure-excitatory system that results in the individual seeking out excess stimulation to compensate their intrinsic **Hypothymia.**
  - Blum has called this:
    - **“The Reward-deficiency syndrome”**
  - Different such Dopamine-receptor alleles have been found to be associated with conduct disorders, pathological gamblers, alcohol-dependant individuals, ADD/H and smokers.

Different smokers are smoking for different reasons.

• I want to relax:	Think MAO-I effect – Dx?
• I want to be less anxious:	Think MAO-I effect – Dx?
• I smoke to concentrate:	Think ADD/H and Dopamine
• I smoke when I’m bored:	Think ADD/H and Dopamine
• It makes me feel better:	Think Grief, Depression, Primary hypothymie.
• It’s my only pleasure:	Think Dopamine-hobbies
• It accompanies me:	Think Endorphins, Attach’t

Depression

- DSM-IV-R:

Depression(5)	Withdrawal(4)
– ***Depressed affect***	***YES***
– Decreased interests and irritabiltiy	
– Frustration and anger	
– ***weight loss or gain***	***YES***
– ***insomnia or hypersomnia***	***YES***
– ***agitation or retardation***	***YES***
– fatigue	anxiety
– worthlessness or guilt	**Slowed pulse**
– ***decreased concentration***	***YES***
– thoughts of death	

- So many criteria in common.

Is he really depressed?

- Firstly;
  - The clinician should aim to completely replace the nicotine that smoker is exposing himself to.
  - So the smoker making a quit attempt WITH NRT is not yet in withdrawal.
  - Withdrawal begins only when the dose of nicotine is reduced (frequency or dosage).
  - If the smoker is not well with **adequat NRT** then it is worth thinking about the MAO-I effect that seems to be related to something other than nicotine (compulsiveness?).

Smoking as Hidden Self-Medication 1

- The psychoactive effects of smoking easily explain the relationship between psychiatric co-morbidity and the smoking behavior
- Eg. A depressed patient has much more to get from smoking than just the Nicotine Pleasure; increased monoamine activity, increased endorphin activity as well as dopamine effects on pleasure, attention and improved intellectual performance

Smoking as Hidden Self-Medication 2

- The problem as it stands:
  - The smoker with a co-morbid disorder that is attenuated by the smoking is in a sub-clinical / non-clinical state.
  - He /she and the treating physician are most probably completely unaware of the underlying co-morbidity as the patient has no complaints.
  - Once the patient tries to stop, then the symptoms are exposed but are they recognized?

Smoking as Hidden Self-Medication 3

- DSM-IV attempts to sensitize against erroneous diagnoses of dependency by insisting that:
- Criteria D:
  - *'The symptoms are not due to another general medical condition and are not better explained by another type of mental disorder'.*

Co-Morbidity

- Over the past 15 years the co-existance of psychiatric disorders and substance misuse, abuse and dependency has been largely recognized
- However the research into psychiatric co-morbidity as related to tobacco use and dependency has been slow to make its way to the bedside.

Many working hypotheses

- Self-medication: The smoker depressed or otherwise uses tobacco to improve his mood and/or his performance.
- Reward Deficiency Hypothesis of Blum
- Relative-subjective perceived hypo-stimulation compensated with tobacco use
- Probably a combination of these apply

The Reality of the Available Pharmacological Tools

- **Zyban**;
  - after 6 weeks of Tx: 58 % abstinent
  - after 52 weeks: 24 % abstinent (placebo=12 %)
- **Patch/Gum/Lozenges**;
  - after 6 weeks of Tx: 35 % abstinent
  - after 52 weeks: 20 % abstinent (placebo= 10%)
- So despite **'100% more success'** claims, the real clinical efficacy is still small -- So?

The Reality of the Available Pharmacological Tools

- **Nicotrol inhaler**:
  - efficacy about équivalent to the gum.
  - 'smoked' more like a cigar than a cigarette; that is the inhalation is kept in the mouth to be absorbed.
- **Champix**:
  - Is a nicotine receptor partial agonist that also blocks nicotine from interacting with said receptor (associated antagonist effect).
  - Efficacy is **doubled** w.r.t. other methods.
  - Adverse affects more related to stopping smoking (and uncovering co-morbidities) than the medication itself.

Pharmacological Limitations

	Dopamine	ACh	Endorphins	Other Monoamines
Substitution	Yes	Yes	?	No
Bupropion	Yes	No	No	No
Smoking	Yes	Yes	Yes	Yes
Champix	Yes	Yes	Yes	Yes

The Symptoms

	Nicotine withdrawal	Major Depression	Anxiety disorder
Dysphoria	Yes	Yes	Yes
Insomnia	Yes	Yes	Yes
Irritability	Yes	Yes	Yes
Anxiety	Yes	Yes	Yes
Bradycardia	**Yes**	**No**	**No**
Concentr	Yes	Yes	Yes
Restlessness	Yes	Yes / No	Yes
Appetite	Yes	Yes / No	Yes / No

The NiCE-Q Questionnaire

- Screening tool
- Developed to aid the clinician to be more aware and able to better identify co-morbid disorders hidden behind the smoking behavior.
- It is a work in progress.
- It still needs to be validated and perfected.
- It can be filled out before the smoker attempts to quit and then done again, after he has done so, depending on the clinical picture

Then what?

- The principle is easy:
  - Any positive response merits to be examined more closely by the physician and the appropriate psychiatric questionnaires should be completed to confirm the tentative diagnosis.
  - If there is a high suspicion of one or more underlying co-morbid conditions, the clinician may choose to wait for the full-blown disorder to come to light or decide to treat before attempting to help to smoker to quit using none or many of the available pharmacological tools once stable.

» John P. Sader MD, ASAM certified 11/2009

09:30 - 10:30 Finding Answers to Your Clinical Questions in Two Minutes (or Less)

Roland Grad MD, M.Sc, FCFP

Associate Professor, Department of Family Medicine, McGill University

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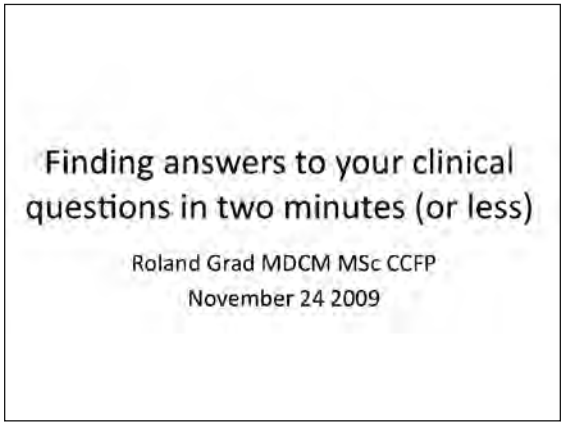
Research Interests: Roland Grad is a family doctor in Montreal who runs a clinical practice since 1986. Later, he obtained a Master of Science in Clinical Epidemiology and Biostatistics from McMaster University. Since 2003, his research to develop and validate the Information Assessment Method (IAM) is funded by the Canadian Institutes of Health Research. IAM is a promising tool for research on e-learning in a push or pull context, focused on evaluation of practice-based education and how to stimulate reflective learning in the health profession.

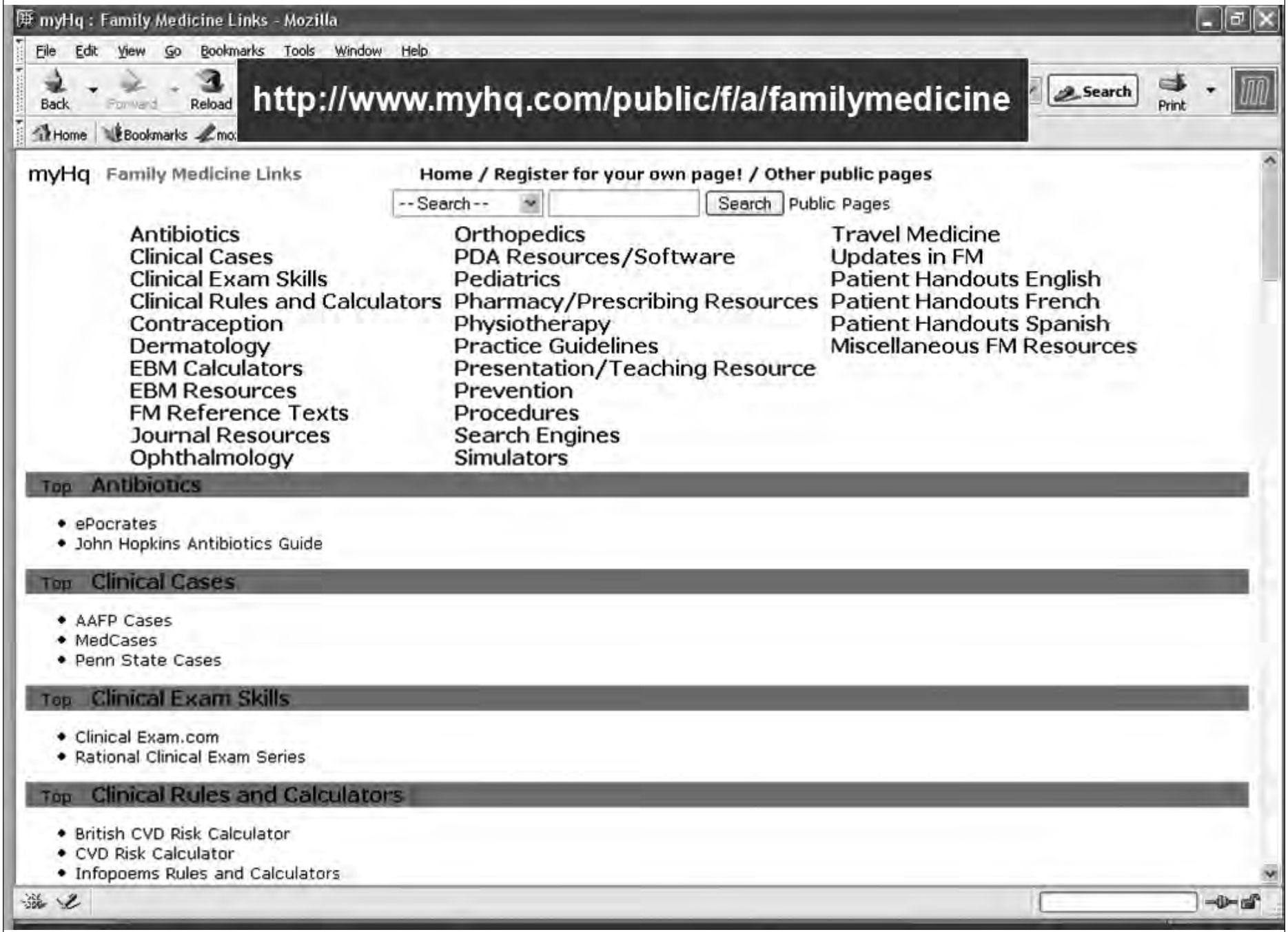
1. Impact of Technology-enabled Knowledge Translation:

My research seeks to implement and evaluate users' assessment of information hits, and thereby promote knowledge exchange between "providers" and "users" of health information technology. Since 2003, I have been funded in two CIHR Knowledge Translation competitions. From this line of work, I co-discovered a new method to assess the impact of information hits derived from electronic knowledge resources. Our impact assessment method was recognized by the McGill Office of Technology Transfer, who filed a U.S. Patent Application on our behalf in 2006. This method is comprised of an ordinal impact assessment scale that can be completed by a health professional in real-time. The impact scale is systematically deployed on the user's computer screen as a pop-up questionnaire linked to specific information hits e.g. a clinical decision rule, in either a 'push' or 'pull' context. Our new method requires further evaluation of validity in the context of daily work. As such, I am principal co-investigator (along with Pierre Pluye MD PhD) on a grant application to CIHR in 2006. Since 2001, I have co-authored seven papers and 10 abstracts with Pierre Pluye, who I helped to recruit to a new full-time PhD research position in the Faculty of Medicine at McGill in 2005. Consistent with my interest in knowledge translation, I was McGill site representative to the Canadian Cochrane Centre and Network from 2000-2006.

2. Medical informatics and the MOXXI projects:

MOXXI is a series of studies to develop electronic systems to optimize the planning and delivery of drug prescribing in primary care office practice. Since 1999, I have been a member of the MOXXI team that developed electronic prescribing software for the handheld computer. Impact: One randomized controlled trial in progress, two papers and a U.S. Patent Application.

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Clinical Cases
Clinical Exam Skills
Clinical Rules and Calculators
Contraception
Dermatology
EBM Calculators
EBM Resources
FM Reference Texts
Journal Resources
Ophthalmology
Orthopedics
PDA Resources/Software
Pediatrics
Pharmacy/Prescribing Resources
Physiotherapy
Practice Guidelines
Presentation/Teaching Resource
Prevention
Procedures
Search Engines
Simulators
Travel Medicine
Updates in FM
Patient Handouts English
Patient Handouts French
Patient Handouts Spanish
Miscellaneous FM Resources
Top Antibiotics
• ePocrates
• John Hopkins Antibiotics Guide
Top Clinical Cases
• AAFP Cases
• MedCases
• Penn State Cases
Top Clinical Exam Skills
• Clinical Exam.com
• Rational Clinical Exam Series
Top Clinical Rules and Calculators
• British CVD Risk Calculator
• CVD Risk Calculator
• Infopoems Rules and Calculators

Tuesday, Nov. 24 – Workshop D-08

09:30 - 10:30 Separation, Divorce and Family Mediation

Gerald Schoel c.o.

Director of Educational Professional Services, Ordre des Conseillers et Conseillères et des Psychoéducateurs et Psychoéducatrices du Québec

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Research interests: Dr. Schoel is Director of Educational Professional Services and has worked with separating and divorcing families since 1970. He offers training courses as well as providing supervision in Family Mediation. He has served on the Board of Directors and the executive of (OCCOPPQ) Ordre des Conseillers et Conseillères et des Psychoéducateurs et Psychoéducatrices du Québec for over 25 years. He is Immediate Past-President, member of the Board of Directors and an Officer of (FMC) Family Mediation Canada. He is a member of the Board of Directors of (AMFQ) Association des Médiateurs Familiale du Québec and its Executive as Treasurer He is a member of the (COAMF) Comité des Organismes Accréditeurs en Médiation Familiale and sit on its Advisory Committee He servers on a number of other committees related to Divorce and Mediation that meets occasionally at both the provincial and federal levels.

Lecture 1

Separation, Divorce and Family Mediation

Gerald Schoel, c.o., Director Educational Professional Services; Past President, Family Mediation Canada; Treasurer, Association de Médiation Familiale du Québec; Phone/ Fax : (514) 733-9081

The objectives of this presentation are for the GP to gain an insight into Family Mediation, to understand the processes involved and for whom it is appropriate. GPs may see patients, exhibiting high stress levels, who are thinking about or are in the process of a separation or divorce and these patients may benefit from Mediation. The presentation will be open with questions from the participants welcomed at all times.

1) **INTRODUCTION**

2) **DEFINITIONS**
   - Mediator
   - Mediation

3) **BASIC LAWS AND REGULATIONS**
   - Mediator
   - Mediation

4) **MEDIATION AND THE GP**
   - Psychological aspects
   - Medicate / Mediate

5) MEDIATION PROCESS

a. Intake
b. Readiness
c. Information re process
d. Contract
e. Information gathering
f. Solutions (brainstorming)
g. Evaluation
h. Decisions in principle
i. Review of decisions
j. Summary of Mediated Agreements

6) MEDIATION CONTENT

a. Children; best interests
b. Couple
c. Child Support
d. Spousal Support
e. Division of Assets
   Family Patrimony
   Family Assets
f. Review of :
   Health insurance
   Life insurance
   Estate planning.

7) REFERENCES

a. Family Mediation Canada
   FMC@FMC.CA
b. Association de Médiation Familiale du Québec
   Tel.: (514) 990 4011 or 1 800 667 7559
   Fax: (514) 270 4155
   Video: AN OTHER WAY TO WIN: FAMILY MEDIATION

c. Educational Professional Services
   Tel.: (514) 733 9081
   Fax: (514) 733 9081
d. Québec Minister of Justice
   www://Justice.gov.qc.ca
   Tel.: 1 800 667 4444

e. Montréal Palais de justice
   Service de médiation familiale
   10 rue Notre-Dame, est
   Bureau 1.150
   Tel.: (514) 393 2326
   Fax: (514) 873 4760

NOTE: A number of pamphlets re mediation will be available for each participant.

Tuesday, Nov. 24 – Workshop E-01

11:00 - 12:00 ER: Acute Confusional State

Eric Tremblay MD

St. Mary's Hospital Centre

Notes

Notes

11:00 - 12:00 Driving Assessment in the Geriatric Patient

Paul G. Lysy MD, FCFP

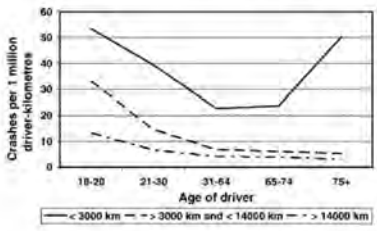
Assistant Professor of Family Medicine, McGill University

Research interests: When Dr. Lysy graduated from Medicine, Geriatrics had not yet been invented. He therefore grandfathered into the field having an interest and practice in the care of the elderly since the early 1980s. Since then he has worked as a consultant to the home care service at CLSC Metro and been the director of the Geriatric Unit at the now closed Queen Elizabeth Hospital, both in Montreal. He is currently the medical director of Chateau Westmount, a private long term care institution, and a staff member of the McGill University Health Centre Geriatric Clinic. His private practice continues to be heavily slanted toward the care of the elderly and includes a large number of home care patients.

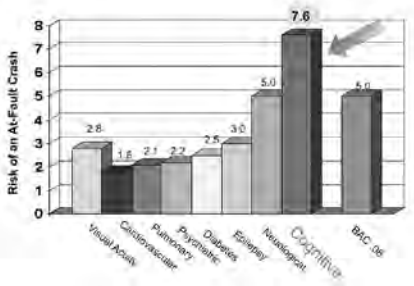
Learning objectives

- to raise awareness of the need to systematically consider an older patient's driving status
- to become more familiar with the currently available clinical tools that may help to evaluate driving ability
- to become more comfortable discussing driving cessation with patients

Crashes and driving exposure



Increased Risk of an At-Fault Crash



Why do we miss congnitively impaired unfit drivers?

1. Dementia is often missed.
2. It is perceived that interfering with driving may harm the doctor-patient relationship.
3. There are no evidence based tools for assessment.
4. The gold standard of road assessment is costly and therefore not available to all.

Physicians role

- It is the role of the physician to provide relevant medical information to the Ministry so that it can make the decision about the patient's ability to drive.

Physicians' Reporting Requirement

- Most provinces *require* that physicians report unfit drivers. In other words it is an offence not to report a patient who is unfit to drive for medical reasons. There are exceptions. So in Quebec, Nova Scotia and Alberta reporting is not mandatory: physicians are *permitted* to breach confidence and "report a patient who they believe may have a medical condition that renders the person unable to operate a motor vehicle" (CMPA).

Warning!

- There is legal precedent for doctors being sued for not reporting in provinces where reporting is mandatory. (In all Canadian jurisdictions the doctor is protected if he does report.)

How to assess fitness to Drive

- The first thing to remember is that the situation may be easy.
- The patient may be either obviously fit or obviously unfit.
- If neither is the case then proceed to more specific evaluation.

SAFEDRIVE

Factors that should be considered in the assessment of older drivers spell out the mnemonic SAFEDRIVE.*

Safety record	Is there a history of driving problems?
Attentional skills	Does patient experience lapses of consciousness or episodes of disorientation?
Family report	What are family's observations of driving ability?
Ethanol	Screen for alcohol abuse
Drugs	Review medication, especially for psychoactive drugs
Reaction time	Are neurologic and musculoskeletal disorders slowing reactions?
Intellectual impairment	Complete Mini-mental State Examination
Vision	Test for visual acuity
Executive function	Does the patient have trouble planning and sequencing tasks and self monitoring behaviour?

1. Dementia Type

- Generally unsafe:
  - Lewy Body dementia
    - fluctuations, hallucinations, visuospatial problems
  - Frontotemporal dementias
    - if associated behaviour or judgment issues

2. Functional Impact of the Dementia

- Consider ADLs and IADLs as a hierarchy with Driving being at the top as the highest level IADL (the only one where fractions of a second can result in accidental death)
  - According to CMA guidelines and Canadian Consensus Guidelines on Dementia; persons with dementia are *unsafe to drive* if
- Impairment of >1 IADL *due to cognition* (IADL mnemonic = SHAFT)
  - Shopping
  - Housework/Hobbies
  - Accounting
  - Food
  - Telephone/ Texts
- *OR* impairment of 1 or more personal ADLs *due to cognition* (ADL mnemonic = DEAT)
  - Dressing
  - Eating
  - Ambulation
  - Transfers
  - Hygiene

3. Family Concerns

- **ask in a room *separate* from the patient:**
  - If family feels the patient is safe/unsafe (make sure family has recently been in the car with the person driving).
  - *The granddaughter question*—Would you feel it was safe if a 5-year-old granddaughter was in the car alone with the person driving? (Often different response from family's answer to previous question)
  - Generally if the family feels the person is unsafe to drive, they are unsafe. If the family feels the person is safe to drive, they *may still be unsafe* as family may be unaware or may be protecting the patient.

Ask Family Specific Questions - Signs of a Potential Problem

- Collisions and/or damage to the car
- Getting lost
- Near-misses with vehicles, pedestrians
- Confusing the gas and brake
- Traffic tickets
- Missing stop signs/lights; stopping for green light
- Deferring right of way
- Not observing during lane changes/ merging
- Others honking/irritated with the driver
- Needing a co-pilot (cannot compensate for emergencies)

4. Visuospatial Issues

- Intersecting pentagons/clock-drawing test
  - if major abnormalities, likely unsafe.

5. Physical Inability to Operate a Car (Often a "physical" reason is better accepted).

- musculoskeletal problems, weakness/ multiple medical conditions affecting
  - neck turn,
  - use of steering wheel/pedals,
  - ability to move feet rapidly
  - ability to feel the gas / brake pedals,
  - level of consciousness
    - cardiac/neurological problems (episodic "spells")

6. Vision/Visual Fields

- Significant problems including visual acuity, field of vision.

7. Drugs (If associated with side effects—drowsiness, slow reaction time, lack of focus)

- **especially high doses or changing doses**

- Alcohol, benzodiazepines, narcotics, neuroleptics, sedatives, anticonvulsants
- Anticholinergics—antiparkinsonian drugs, muscle relaxants, tricyclic antidepressants, antihistamine (OTC), antiemetics, antipruritics, antispasmodics, others (next slide)

8. Trail Making A and B

- Trail Making A:
  - *Unsafe* = >2 minutes or 2 or more errors
- Trail Making B:
  - *Safe* = <2 minutes and <2 errors (0 or 1 error)
  - *Unsure* = 2–3 minutes or 2 errors (consider **qualitative dynamic information** regarding ***how*** the test was performed—slowness, hesitation, anxiety or panic attacks, impulsive or perseverative behaviour, lack of focus, multiple corrections, forgetting instructions, inability to understand test, etc.)
  - *Unsafe* = >3 minutes or 3 or more errors
    - The longer the patient takes and the more errors they make, the more certain you can be that they are unsafe

9. Ruler Drop Reaction Time Test

- The bottom end of a 12 inch (30-cm) ruler is placed between thumb and index finger (1/2 inch (1 cm) apart) → let go and person tries to catch ruler (normal = 6-9 inches (15–22 cm); abnormal = 2 failed trials out of 3 trials
  - No validated norms / cut-offs

10. Judgment/Insight

- **ask the person:**

- What would you do if you were driving and saw a ball roll out on the street ahead of you?
- With your diagnosis of dementia, do you think at some time you will need to stop driving?

Driving Cessation–Still competent

- A patient who has a progressive condition which will lead to incapacity to drive needs to be told this so that he and his family can plan ahead.
- As the patients insight and judgement deteriorate this may be less useful.
- Allows physician to gauge patient response.

If you expect serious resistance

- Meet first with the family.
- It will usually be easier for the family to understand the need for cessation.
- Go through the results of the tests and the legal obligation to report.

The Driving and Dementia Toolkit. RCPEO

Educating the family

- Explain that the aim is to prevent accidents.
- Older people do very poorly if they survive an MVA.
- Other people may be seriously injured and this will be partly their responsibility

The family in a supportive role

- While you are required to give the bad news, the family can play a supportive role to help the patient deal with the news and its consequences. (good cop/bad cop)

Family also resists

- Re-explain the legal situation
- Explain the tests used and show them the results
- If necessary re-test the patient in their presence chosing the most telling test.

Meeting with patient and family

- Clarify roles: family will be the good cop and support the patient, doctor will be the bad cop who discloses.

Disclosing

- Give the patient something positive: he has been a good, responsible driver. He would never want to hurt anyone.
- Attribute your decision to the results of the examinations and to the legal obligations
- Acknowledge patient's reaction as normal.

Talk about transportaion options

- Taxis cost less if less than 4000 km a year is driven.
- Point out how the family can help to meet transportation needs.
- Volunteer drivers and helpful taxi drivers will meet some of the needs.

Patient still refuses to stop

- Family should remove the patient's opportunity to drive: take away the keys, disable the car, remove the car.
- Provide written notice.
- Indicate that you will notify the authorities.

Useful documents

- http://www.cma.ca/index.cfm/ci_id/18223/la_id/1.htm CMA's guide to determining medical fitness to drive.
- http://www.rgpeo.com/en/resources/DementiaToolkitJune2009.pdf Driving and dementia toolkit from the Regional Geriatric Program of Eastern Ontario.
- http://www.drivingwell.net/data/media/docs/UIowa_TrailMaking.pdf Trail Making Test A and B

Notes

Notes

11:00 - 12:00 Knee Evaluation

Alan Vernec MD

Medical Director, World Anti-Doping Agency (WADA)

Evaluation of Ankle Injuries

Alan Vernec, M.D.; Dip Sport Med
Univ. de Montréal: Clinique de médecine du sport
McGill University: HFPC. Jewish General Hospital

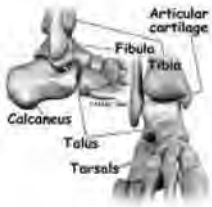
Goal of presentation

- Recognize injuries in and around the ankle
- Management and treatment of selected injuries
- Know when to refer

Evaluation of the Ankle

« Ankle injuries » are often not in the ankle joint.

Know your differential diagnoses.



Injuries in and about the Ankle

- Ankle sprains
  - Lateral ligaments,
  - Medial ligaments
  - Tibiofibular ligaments
- Fractures
  - Ankle (lower tibia, fibular, **talar dome**)
  - Lower leg (tibia, fibular)
  - Foot (**5th MT**, navicular, calcaneus, **lateral** or post. talus,…)

Injuries in and about the ankle

- Osteochondritis dissecans (talus)
- Lateral retinacular tears/peroneal subluxation
- Lisfranc injury (mid-foot sprain)
- Os trigonum
- Sever's Disease

Ankle sprains Anatomy

History Mechanisms of injury

Plantar Flexion / Inversion

Dorsiflexion / Eversion

Mechanisms of Injury

Pure Eversion

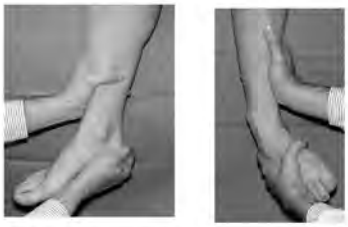
Anterior tibio-fibular sprain

- "high ankle sprain"
- May include posterior tibiofibular ligament + tibiofibular syndesmosis
- Consider further imaging

Physical Exam
Ankle ligaments

- Observation
- Palpation
- Anterior Drawer Test (ATF lig.)
- Talar Tilt (CF lig.)
- Tib-fib stress test (ant tib-fib lig.)
- Spring test (ant tib-fib lig.+ syndesmosis)

Lateral ligament tests



Anterior drawer

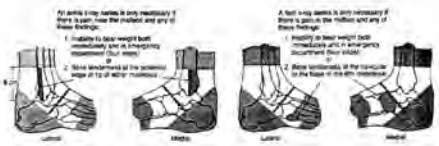
Talar Tilt

Anterior tibiofibular ligament and syndesmosis tests

Ant. tibiofibular stress test

Spring test

Imaging – X-Ray



Ottawa Ankle Rules

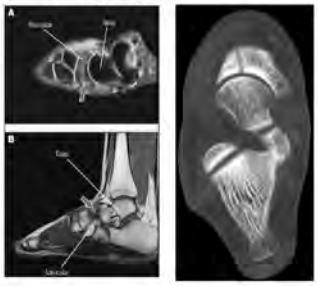
Ottawa Ankle Rules Made Easy

- Is patient unable to walk?
- Is there tenderness anywhere other than anterior ankle ligaments?
- Are there possible fractures elsewhere?

If yes - X-Ray

Imaging

Stress X-rays
Bone scan
CT
MRI



Ankle Sprain
Acute Management

PRICE
- Protect (brace)
- Rest
- Ice
- Compression
- Elevation

Ankle Sprains

When should surgery be considered?

Persistent functional or mechanical instability <u>after rehabilitation</u>

Why surgery for 3rd degree ankle sprains should rarely be seriously entertained:

- Risk of infection and arthritis
- Risk of scarring and traumatic neuromas
- Increased loss of work days
- Residual symptoms of non-operated group are usually of minimal concern
- and the number one reason against early surgery is…

<u>delayed surgery gives equally good results</u>

Ankle Sprain
Further Management

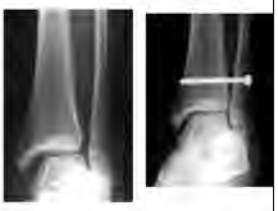
Rehabilitation exercises
- Proprioception, strengthening…
- Even a first degree sprain can benefit from proper rehab.

Ankle Sprain Management

- Bracing.
  - Lace-up or semi-rigid brace helps decrease recurrences (Cochrane review 14 studies)

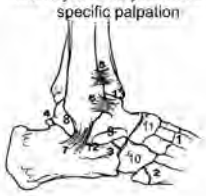
High ankle sprain management
Tib-fib and interosseus membrane injuries

- May cause long-term instability
- Should consider casting (NWB) x 6 weeks
- May need pin



Recognize other injuries in and around the ankle

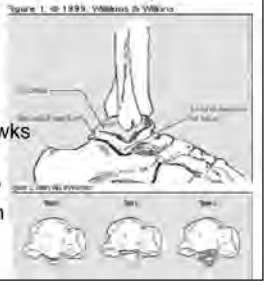
The key is history followed by specific palpation



1. **Lisfranc fracture/dislocation**
2. **Base of 5th MT**
3. Anterior calcaneus #
4. **Posterior talus or os trigonum**
5. *Anterior tibiofibular ligament*
6. *Anterior talofibular ligament*
7. *Calcaneofibular ligament*
8. *Posterior talofibular ligament*
9. **Lateral talar fracture**
10. Cuboid subluxation
11. Navicular injury
12. Sinus Tarsi Syndrome
13. **Osteochondral injury of talus**

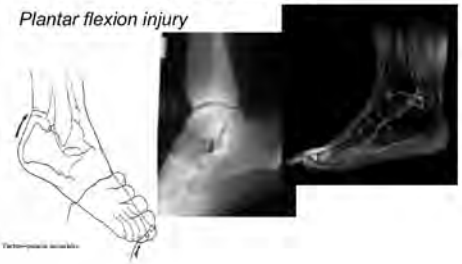
Lateral process of talus fracture (Snowboarder's ankle)

- *Dorsiflexion and eversion injury*
- May need CT to dx
- If chip – cast x 4-6 wks NWB
- Larger fracture may need pin or excision



Os trigonum or posterior process of talus fractures

Plantar flexion injury



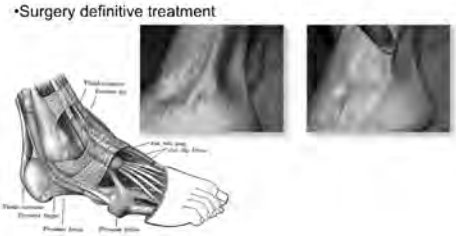
Osteochondral injuries of the talus
Osteochondritis dissecans (OCD)

- Usually primary trauma then ischemia
- Posteromedial or anterolateral talus
- Often not evident on initial imaging
- Refer to ortho

Figures: Courtesy of David B. Thordarson, MD

Peroneal tendon injury/subluxation

- *Inversion, plantar flexion injury*
- Surgery definitive treatment

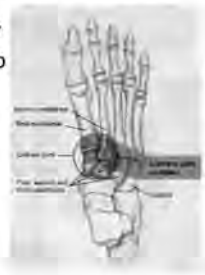


Jones fracture
(proximal 5th MT fracture)

- *Inversion injury*
- Acute fracture
- Should be casted, NWB x 6 weeks
- Consider pinning

Lisfranc injury of the midfoot

- *full plantar flexion injury*
- if missed – may develop significant pathology
- Bilateral standing XR; MRI and/or CT
- Refer to ortho

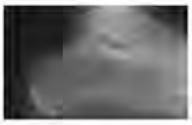


Achilles rupture

- *Sudden eccentric stop*
- Palpation defect may be missed early
- Thompson test
  - may mislead
- Standing toe raise!

Sever's Disease

- *Overuse injury*
- Cousin of Osgood Schlatter's
- Traction apophysitis
- Age 10-14
- Self-limiting



Consider referrals to Orthopedics if:

- Complex fractures with unstable ankle (disrupted mortise)
  - Bimalleolar or trimalleolar fractures (unstable)
  - Fracture of posterior malleolus of tibia (large or displaced)
  - Medial or lateral malleolar fractures more than just tip
  - Lateral talar fracture more than tip
- Anterior tib-fib. sprain with # or with suspected instability
- Osteochondral lesion
- Jones # in high level athlete
- Navicular pathology
- Lisfranc lesion (midfoot sprain)
- Achilles rupture

Case studies

- 40 y.o. plumber with ankle pain and swelling following inversion injury
- 47 year old with heel pain following pick-up basketball
- 14 year old soccer player with heel pain
- 19 year old courier and skateboarder with lateral ankle pain and swelling

Summary

Know your anatomy and differential dx.

Don't neglect sprains

X-ray appropriately and use further imaging when necessary

Refer as discussed

Notes

Notes

Tuesday, Nov. 24 – Workshop E-04

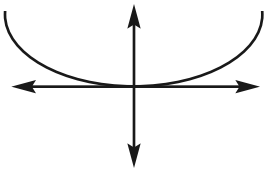
11:00 - 12:00 HANDS ON: Back Exam

Mohan Radhakrishna MD, FRCPC

Assistant Professor, Division of Physical Medicine and Rehabilitation,
McGill University and Montreal General Hospital

Inspection

- Begins from when patient is called from waiting room
- Observe for abnormal gait, sitting posture, behavior indicating level of suffering, spontaneous movement during history-taking
- Ensure patient is adequately undressed



Standing:

- Fortin finger sign for sacroiliac joint pain
- Assess curvature: kyphosis, scoliosis
- Leg length discrepancy (buttock and popliteal creases)
- Ask the patient to bend forwards: postural scoliosis resolves, a structural scoliosis does not disappear
- (Modified)Schober's test
  - When the spine flexes, the distance between each pair of vertebral spines increases.
  - In the Schober's test, a tape with a 10 cm mark is placed vertically in the midline upwards from the level of the dimples at the level of the posterior superior iliac spines). Mark the skin at 0 and at 10 cm and then ask the patient to flex as far forward as they can.
    Record where the 10 cm mark on the skin strikes the tape. The increased distance along the tape is due only to flexion of the lumbar spine and is normally about 6-7 cm (less than 5 cm should be considered as abnormal).
- Ask the patient to extend their lower back.
- Ask the patient to move laterally (sideflex to touch knee).
- Look for any other abnormalities, e.g. cafe-au-lait spots, which may suggest neurofibromatosis, a fat pad or hairy patch suggestive of spina bifida, or scarring suggestive of spinal surgery.
- Unipedal stance—look for contralateral iliac crest to drop (sign of weak ipsilateral hip abductors)
- Have patient squat to screen range of motion in hips, knees, ankles plus kinesiophobia.
- Functional overlay (Waddell's Signs)
  - Hip rotation
  - Axial loading: apply pressure to the head. Overlay is suggested if this aggravates the back pain.

Sitting

- Myotomes:
  - L2 and L3: resisted flexion of the hip
  - L3: resisted knee extension
  - L4: resisted dorsiflexion of the ankle
  - L5: resisted extension of the big toe
  - S1: resisted toe flexion

  NB: Need to test ankle plantarflexion with repeated heel raises

  NB: Giveway weakness versus true neurologic weakness

- Dermatomes: test sensation to pinprick:
  - L2: mid thigh
  - L3: medial knee
  - L4: medial aspect of the ankle
  - L5: 1st webspace
  - S1: lateral aspect of the foot, the heel and most of the sole
  - S2: posterior aspect of the knee
  - S3: ischial tuberosity
  - S4, S5 concentric rings around the anus

- Reflexes:
  - Patellar tendon L4
  - Medial hamstrings or tibialis posterior L5
  - Achille's reflex S1
  - Plantar response
- Seated straight leg raise
  - Tripod
  - Slump

Lumbar rotation

Supine

- Straight leg raising:
  - Passively flex hip with knee straight while patient is supine. Pain in leg and back between 30-70o implies neural involvement. If positive lower leg until symptoms better then dorsiflex ankle. Stop when the patient complains of back or leg pain (hamstring tightness is not relevant). The test is negative if there is no pain. Paresthesia or pain in root distribution is very significant, indicating nerve root irritation.
  - Repeat on other side. A positive crossed SLR is highly specific.
- Femoral stretch test:
  - With the patient prone and the anterior thigh fixed to the couch, flex each knee in turn.

Tuesday, Nov. 24 – Workshop E-05

11:00 - 12:00 CMPA

Ross Berringer MD, D(ABEM), MCFP(EM)

Physician Risk Manager, Risk Management Services,
Canadian Medical Protective Association

Research Interests: Dr. Ross Berringer graduated from the University of British Columbia in 1980. After completion of a rotating internship at St. Paul's Hospital in Vancouver, he was a general practitioner in 100 Mile House from 1981-83. During the ensuing two years, he completed a residency in emergency medicine in Jacksonville, Florida. He holds a certificate of special competency in emergency medicine and is a diplomate of the American Board of Emergency Medicine. From 1985 through 2006, he practised full time emergency medicine at St. Paul's Hospital achieving the rank of Clinical Associate Professor. In addition, he was the Medical Director for Vancouver Fire and Rescue Services and the Advanced Life Support Medical Advisor for the BC Ambulance Service. His research interests have been in pre-hospital care and out-of-hospital cardiac arrest. In May of 2006 he joined the CMPA as a Physician Risk Manager.

Tuesday, Nov. 24 – Workshop E-06

11:00 - 12:00 End of Life Care

Michael A. Dworkind MDCM, CCFP, FCFP
Assistant Director, Herzl Family Practice Centre,
SMBD-Jewish General Hospital
Director, Living Will Project of the Clinical Ethics Committee,
SMBD-Jewish General Hospital;
Associate Professor, Department of Family Medicine, McGill University

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This workshop is about caring for the dying. We will focus our work on the principles of palliative care and how they are woven into the vital role that family physicians play in caring for people at the end of life.

Palliative care is the active, comprehensive and compassionate care of the terminally ill at a time when their disease is no longer responsive to traditional treatment aimed at a cure or prolongation of life, and when the control of symptoms is paramount. The symptoms requiring utmost attention are multifaceted in nature and incorporate physical, emotional, social and spiritual needs, with maximization of comfort and minimization of suffering.

Palliative care is multidisciplinary in its approach and encompasses the patient, the family, and the community in its scope. The use of family meetings and various models of community/home care resources will be discussed.

As well, knowledge of the ethical decisions at the end of life and communication of hope where it is seemingly a hopeless situation, are skills that can be developed by primary care physicians who really know their patients and their families. Discussion around topics of quality of life like truth-telling, medical directives, goals of care and DNR will be explored.

This privileged position, allows family physicians to help all those suffering loss, to better be able to heal through the dying process. Case narratives will be shared and dialogue with participants will be encouraged. Dilemmas and challenges at end of life care will be explored giving practical patient centered suggestions on adding meaning and reducing some of the unnecessary suffering of the final journey.

REFERENCES

1. Oxford Textbook of Palliative Care. Doyle, Hands and McDonald.

2. Caring for the Dying. Educational Resource document. American Board of Internal Medicine.

3. Palliative Care: Easing the Pain. Elizabeth J. Latimer. Canadian Journal of Diagnosis. September 1996.

Research Interests

Quality of end of life care; Advance directives in clinical practice.

Dr. Dworkind is also a consultant in pain and palliative care at the Jewish General Hospital, with affiliations to Mount Sinai Hospital and CLSC Côte-des-Neiges. In addition, he is a Board Member of Physicians for Global Survival.

Notes

11:00 - 12:00 Treatment of Resistant Depression

Khalil Geagea MD, FRCP[C]

Director, In-Patient Services, Psychiatry, S.M.B.D. Jewish General Hospital
Assistant Professor, McGill University
Chargé de formation clinique, Université de Montréal

TREATMENT RESISTANT DEPRESSION: DIAGNOSIS & TREATMENT

By: Khalil Geagea, M.D.

Jewish General Hospital
October 2008

Depression Is . . .

- Common
- Recurrent
- Debilitating
- Physical as well as emotional
- Potentially lethal
- Treatable
- A handful

•The discovery of effective antidepressant modalities resulted in depression being among the most "potentially" treatable disorders encountered in medicine.

•60-70% of depressed patients will "respond" to antidepressant treatment.

- 60-70% who can tolerate an AD medication will "respond" to the drug of first choice.

- 70% "response" rate has been reported to CBT in a heterogeneous group of unmedicated unipolar patients.

•Approximately 20% of patients with major depression fail to respond meaningfully to presently-available antidepressant treatment.

- An additional 30% of patients achieve only a partial response to antidepressant treatment with many residual target symptoms in varying degrees of severity.

Defining TRD

- No universally accepted criteria
- Resistance can be relative or absolute
- Duration of Depression greater than 8 months
- Failure to achieve response or remission after two adequate treatment trials
- Lack of agreement on *adequacy* of dose or duration
- Anything less than *remission* is a form of resistance?

Definitions of Treatment Resistant Depression

- Clinical Definitions
  - Failure to return to premorbid self
  - Persistence of depressive syndrome
  - Need for further treatment
  - Patient and family not satisfied with outcome

Poor Outcomes To Antidepressant Treatment Has Been Associated With Several Factors

- Chronicity
- Early or late age onset
- Marked severity
- Psychosis
- High levels of psychic anxiety
- Serious personality pathology
- High levels of dysfunctional attitudes
- Lack of social support (i.e., long-term marital discord)
- Lack of economic resources

Clinical Presentations of TRD

1. Complex lack of acute response
2. Partial response
3. Relapse/recurrence during treatment

Causes of TRD

1. Compliance issues
2. Intolerance to treatment
3. Comorbid psychiatric disorder(s)
4. Comorbid medical disorder(s)
5. Inadequate treatment trial
6. Inadequate assessment of response
7. Partial response to treatment
8. Psychosocial factors
9. Failure to respond

Comorbid Psychiatric Disorder(s) Contributing to TRD

- Common psychiatric disorders comorbid with depression
  - Anxiety disorders
  - Dysthymia
  - Substance use disorders
  - Eating disorders
  - Personality disorders
- Assess whether treatment strategies are appropriate for comorbid disorder(s)
- More intensive treatment of non-affective disorder might improve prognosis of depression (*Keller et al, 1984*)

Anxiety Disorders in Major Depression Temporal relationship

Anxiety disorders usually predate depression
- Social anxiety disorder precedes MDD in 65% of cases
- Generalized Anxiety Disorder precedes MDD in 63% of cases
- Panic disorder precedes MDD in 22% of cases

Except...
OCD follows onset of MDD in 63% of cases

Fava et al. Comprehensive Psychiatry, 2000;160: 17-30

Consequences of Anxiety and Depression

- As many as 90% of depressed patients suffer from anxiety symptoms [1-3]
- More severe illness at baseline
- More psychosocial impairment
- Greater likelihood of chronic illness
- Poorer, slower response to treatment
- Greater likelihood of committing suicide

1. Richou H. et al. Human Psychopharmacol 1995; 10:265-71 2. Coplan JD et al. J Clin Psych 190, 51 (Suppl 10): 9-13 3. Kasper S. et al. Primary Care Psych 1997; 3:7-16

Co-Morbidity
Major Depression – Substance Abuse

- One third of mood disorder patients have a lifetime history of substance abuse
- 20% of persons with alcohol problems have a lifetime history of a mood disorder
- Co-morbidity of mood and anxiety disorders in substance abuse population range from 30% - 60%

Kessler et al. Arch Gen Psychiatry. 1994, Regier et al. JAMA 1990, Merikangas et al. Addict Beh 1998, Ems et al. Gen J Psychiatry 2001

Comorbid Medical Disorders Contributing to TRD

- Depressions complicated by intercurrent medical-neurological conditions tend to have more chronic or protracted courses
- Unrecognized medical illness can either cause or significantly contribute to TRD

Medical Conditions Associated With TRD

- Iatrogenic effects of medications
- HPA axis dysfunction
  - i.e., thyroid disorders, Cushing's syndrome, Addison's disease
- Neoplasma
  - i.e., CNS tumors, paraneoplastic syndromes
- Neurological disorders
  - i.e., Parkinson's disease seizure disorders, cerebrovascular disease dementias, autoimmune disorders (i.e., multiple sclerosis, lupus)
- Infectious disease
  - i.e., tuberculosis, post-viral fatigue syndrome, HIV

Unexplained Physical Symptoms

	% Unexplained
Headache	48
Stomach pain	46
Dizziness	39
Chest pain	36
Back pain	30
Joint pain	26
Dyspnea	25

Kroenke K, Price RK. Arch Intern Med. 1993; 153 (21):2474-2480

Unexplained Physical Symptoms and Psychiatric Disorders

	% Depression	% Anxiety
Headache	53	44
Stomach pain	66	50
Dizziness	66	44
Chest pain	66	66
Back pain	53	40
Joint pain	58	48
Dyspnea	64	44

Kroenke K, Price RK. Arch Intern Med. 1993; 153(21):2474-2480

Possible Markers for Depression in the Medically Ill

- Physical symptoms disproportionate to findings
- Excess functional disability
- High utilization of medical care
- Poor self-care
- Decreased adherence to medical and/or lifestyle-changing regimens

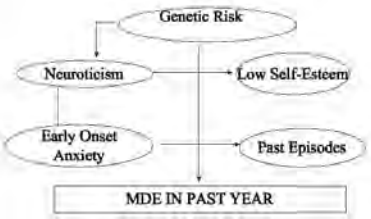
Katon W. Sullivan MD. J Clin Psychiatry. 1990;51(suppl): 3-11

Keys to Case-Finding in Depression
Physical Symptoms
The Common Calling Card in Primary Care

- But don't forget the key depression questions
  1. Are you depressed?
  2. Are you less interested in things?
- Question 1 detects 85-90% of major depression
- Question 2 increases sensitivity to 95%

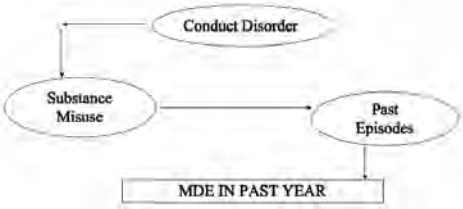
Williams JW, et al. Am J Med. 1999; 106(1):36-43. Whooley MA, et al. J Gen Intern Med. 1997; 12(7):439-445.

Internalizing Pathways to Major Depressive Episode



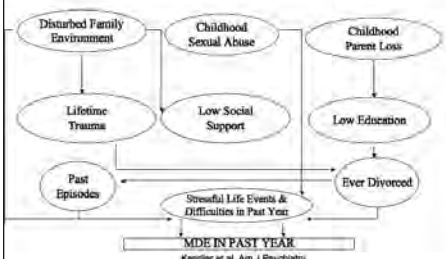
Kendler et al. Am J Psychiatry 2002; 159:1133-1145

2. Externalizing Pathways Major Depressive Episode



Kendler et al. Am J Psychiatry 2002; 159:1133-1145

3. Adversity/Interpersonal Difficulties Pathways to Major Depressive Episode



Kendler et al. Am J Psychiatry. 2002; 159:1133-1145

Life Event Dimensions of Loss, Humiliation, Entrapmen and Danger as Predictors of Onset of MDD

- "In addition to loss, humiliation events were strongly linked to risk of depressive episodes".
- "Environmental experiences that involve loss of status and elicit psychobiological programs of defeat and submission are more depressogenic than those involving solely loss".

Kendler et al. Arch Gen Psychiatry. 2003;60:789-796

Clinical Targets for Antidepressants

1. Major Depressive Disorder
   - Homogeneity vs. Heterogeneity
2. Anxiety
   - Co-symptomicity vs. Co-morbidity
3. Substance Abuse
   - Frequent & Frequently Ignored
4. Physical Symptoms & Depression
   Rapidly re-emerging concept
5. Energy, Motivation & Cognition
   - Slowly Emerging Interest
6. Bipolar Depression
   Discrete Therapeutic Target

SSRIs Have Become De Facto Standard Antidepressants And Displaced TCAs

- Effective
- Convenient
- Favorable side effect profile
- Safe in overdose

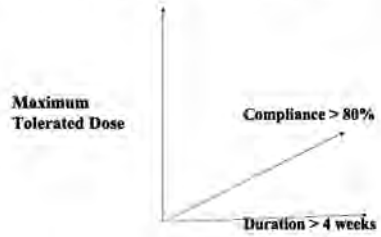
SSRIs Have Had Profound Media Transformations

- Miracle drug
- Killer drug
- Verbal drug
- Designer personality drug

The Approach To Nonresponse To The SSRIs Is Extrapolated From The Literature On TCAs

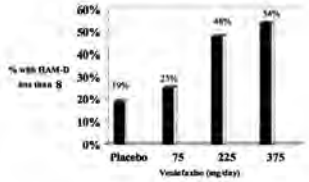
- Optimization
- Augmentation/Combination
- Substitution

Criteria for an Adequate Trial



Optimization: Adequate Doses

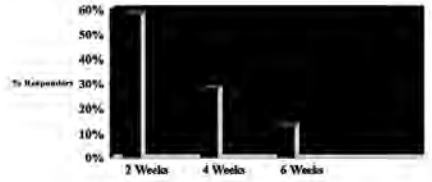
Dose-Response Curve at Week 8
Venlafaxine



11/16/09 Rudolph et al, 1998

OPTIMIZATION: Adequate Duration

Timing of Onset of Antidepressant Response: Patients Who Responded to Fluoxetine



Nierenberg et al, 2000

Proposed Staging Based on Levels of Treatment Resistance

- Treatment-resistance can be conceptualized as a continuum, beginning with the first failure to respond to an adequate trial of an antidepressant

Thase et al. J Clin Psychiatry 1997; 58(Suppl 13):23-9

"The true polypharmacy is the skillful combination of remedies."

– William Osler

Augmentation Strategies in TRD

Therapeutic Choice	*Recommendation*	*Dose*
First	. Lithium	600-900 mg; Or to therapeutic serum levels
Second	. Novel antipsychotics Olanzapine Risperidone	 5-15 mg 1-2 mg
Third	. Triiodothronine (T3)	25-50 mcg
Fourth	Buspirone Lamotrigine	Usual doses

Atypical Antipsychotics

- Risperidone
  - Ostroff & Nelson (1999) – case series of 8 patients. Remission after one week with addition of Risperidone 0.5-1 mg/day to Fluoxetine or Paroxetine.
- Olanzapine
  - Shelton et al (2001) – randomized, double-blinded, placebo controlled trial in 28 patients with Fluoxetine and Olanzapine.
  - 60% response in patients unresponsive to Fluoxetine alone.

Other Novel Antipsychotics

- Ziprasidone
  - Two open label trials
  - Effective augmentation of sertraline in SSRI naïve group
- Quetiapine
  - Open label comparison of quetiapine and haloperidol augmentation of citalopram
  - Quetiapine better tolerated

Kennedy & Lam, Bipolar Disorders 2003;5(suppl2):36-47

Time to Remission During Risperidone Augmentation

	Ostroff	O'Connor	Stroll	Schar (Delusional Depression)
Number of patients	8	4	5	13
Patients in remission	8	4	3	7 full 1 part
Time	1 week	1 week	1 year	4 weeks
Dose (mg/d)	0.5 - 1	0.5 - 2	0.5 – 1.5	3-4

Risperidone add-on

- Depression and severe, intractable anxiety and obsessional, ruminational thinking

Olanzapine/Fluoxetine Combination

"One trial in 28 patients showed that this combination was an effective treatment, compared to the individual components with unipolar depressed patients who had not responded to two antidepressants of different chemical classes.

Two subsequent large-scale attempts at replication have resulted in failed trials....

A recent study showed that monotherapy with olanzapine produced a greater effect than placebo in bipolar depression and the combination of olanzapine and fluoxetine yielded an even more robust response".

Shelton Expert Opin Pharmacother 2001;2:1175-1182

Other Novel Antipsychotics

- Ziprasidone
  - Two open label trials
  - Effective augmentation of sertraline in SSRI naïve group
- Quetiapine
  - Open label comparison of quetiapine and haloperidol augmentation of citalopram
  - Quetiapine better tolerated

Kennedy & Lam, Bipolar Disorders 2003;5(suppl2):36-47

Combination Strategies in TRD

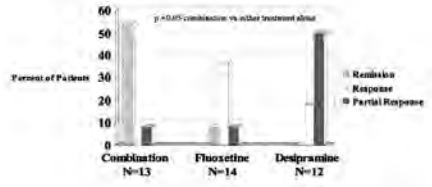
Therapeutic Choice	Recommendation
First	▪ SSRI + mirtazapine/mianserin
Second	▪ SSRI/SNRI + bupropion-SR
Third	▪SRI + TCA (caution for increased serum TCA levels with some SSRIs) ▪SRI + RIMA (caution for serotonin syndrome)

Clinical Response After Bupropion Combination Therapy

	Week 0 (Baseline)	Week 8
Non-Response (HAMD-D ≥ 16)	10 (47.6%)	3 (14.3%)
Partial Response (8 ≤ HAM-D ≤ 15)	8 (38.1%)	10 (47.6%)
Full Response (HAM-D ≤ 7)	3 (14.3%)	8 (38.1%)
X^2=6.26, df=2, p = 0.04		

Kennedy et al., 2001 J Clin Psychiatry. 2002;63:181-186

Fluoxetine and Desipramine Combination Treatment of MDD



Nelson et al., Biol Psychiatry 2004; 55:296-300

Switching
Summary of Response Rates

Switch	Response Rate
SSRI to TCA	46-73%
SSRI to other SSRI	50-60%
SSRI to Venlafaxine	33-70%
SSRI to Bupropion	56%
SSRI to Mirtazapine	67%

Howland. Thesc. J Pract Psychiatry Behav Health 1999;5: 216-23

Switching from SSRI to TCA

From	To	Reason	Outcome
Fluvoxamine[1] (n=31)	Oxaprotiline 100-300mg	Non-response	39% response
Paroxetine[2] (n=25)	Imipramine 65-275mg	Non-response	73% response
Sertraline[3] (n=117)	Imipramine 50-300mg	Non-response	44% response 25% dropout

1. Nolen et al., Acta Psychiatry Scan, 1995 2. Peselow et al., Psychopharmacol Bull, 1989 3. Thase et al. Arch Gen Psychiatry, 2002

Switching SSRIs

From	To	Reason	Outcome
Fluoxetine[1] (n=112)	Sertraline 50-200mg Open trial	Intolerance to SEs	72% response 21% dropout
Fluoxetine[2] (n=57)	Citalopram 20-60mg	Non-response	63% response 18% dropout
Sertraline[3] (n=106)	Fluoxetine 20-60mg	Non-response or intolerance	Intolerance: 70% response NR: 58% response Overall: 63% response 5% dropout
Paroxetine[4] (n=61)	Citalopram 20-40mg	Intolerant	56% response 10% dropout

1. Brown & Harrison, J Clin Psychiatry, 1995 2. Thase et al., J Clin Psychiatry, 1997 3. Thase et al. J Clin Psychiatry, 2001 4. Zarate et al., J Clin Psychiatry, 1996

Indications for ECT

- Acute suicidal ideation
- Psychotic features (delusional depression)
- Treatment resistant depression
- Repeated medication intolerance
- Rapidly deteriorating physical status
- Prior favorable response

Kennedy, Lam, Nutt & Thase 2004

Sequential Combination Treatment Strategies in Partial Responders

Measurement	Li Mean (SD)	CBT Mean (SD)
Ham-D 17		
Baseline	23.1 (3.9)	24.4 (5.2)
Randomization	11.6 (1.9)	12.1 (2.2)
End of	12.8 (7.2)	15.8 (7.1)
Randomization	9.2 (6.7)	14.8 (9.9)
Follow-up		

Kennedey et al., J Clin Psychiatry. 2003;64: 439-444

Summary

- TRD remains a significant clinical problem
- Depression is highly comorbid with anxiety, substance abuse and personality disorder; which correlate with treatment failure
- Interventions should be employed early in treatment to convert non and partial response to remission

Notes

How And When To Discontinue Antidepressants

By: Dr. Khalil Geagea

November 2009

Depressive Illness

- Major Health problem
- By 2020, second only to heart disease as leading cause of morbidity
- Life time risk: 12-26% for women, 4-12% for men
- Chronic and Recurrent (75-80% of treated patients have recurrences)
- Aim of treatment is full remission

Antidepressant Classes

1. Selective Serotonin Reuptake Inhibitor (SSRI)
   - Sertraline (Zoloft)
   - Fluoxetine (Prozac)
   - Paroxetine (Paxil)
   - Citalopram (Celexa)
   - Escitalopram (Lexapro)
1. Tricyclic Antidepressant (TCA)
   - Amitriptyline (Elavil)
   - Nortriptyline (Pamelor)
   - Imipramine (Tofranil)
   - Desipramine (Norpramin)
   - Doxepine (Sinequan)
   - Trimipramine (Surmontil)
   - Protriptyline (Vivactil)
   - Maprotiline (Ludiomil)
   - Amoxapine (Ascendin)
   - Clomipramine (Anafranil)

Antidepressant Classes

1. Serotonin-Norepinephrine Reuptake Inhibitors (SNRI)
   - Venlafaxine (Effexor)
   - **Desvenlafaxin (Pristiq)**
   - Duloxetine (Cymbalta)
2. MAO Inhibitors
   - Phenelzine (Nardil)
   - Tranylcypromine (Parnate)
1. Atypical Antidepressants
   - Bupropion (Wellbutrin)
   - Trazodone (Desyrel)
   - Mirtazepine (Remeron)

Antidepressants Treatment Principles

- Begin with a modest dose
- For partial response or nonresponse:
  1. **Optimize dose or duration of therapy**
     - Minimum of six (6) weeks. If a patient exhibits a significant partial response during this initial period, another 4-6 weeks of treatment should be added (Total: 10-12 weeks)
     - Some may benefit from antidepressant dosages that are higher than recommendations
  1. **Drug Substitution**
     - If no (or inadequate) response – switch to another antidepressant class
       - Exception: SSRI
  1. **Combination Therapy** – add another antidepressant (another class)

Antidepressant Treatment Principles

- Partial response and Nonresponse
  1. Augmentation – add a second agent (not an antidepressant)
     - Lithium
     - Thyroid hormone
     - Pindolol (Viskin)
     - Buspirone (Buspar)
  1. Electroconvulsive Therapy
     - For psychotic depression and severe refractory depression

Antidepressant Treatment Principles: Follow-up

- Every 1-2 weeks for six to eight weeks during the initiation phase of medication treatment – office visits for supportive care, access to provider by phone, and/or proactive phone calls to check on therapeutic response, side effects, and adherence to treatment
- First episode of depression – medication for at least 6-9 months after **remission**
- Two or more episodes of depression – two years (or more) of medication
- Taper medication over 2-4 weeks to avoid withdrawal

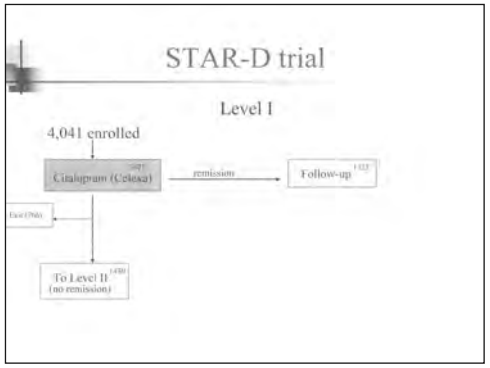
STAR-D trial

- **Sequenced Treatment Alternatives to Relieve Depression**
- Funded by National Institute of Mental Health and published 11/2006
- The largest and longest study to evaluate depression treatment
- Overall objective: define preferred treatments for depression – in a way that mirror methods that clinicians use in practice:
  - Determine best "next-step" treatments for depressions not responding satisfactorily to one or more prior treatment attempts
  - Compare relative efficacy of different treatment approaches
- Participants:
  - 18-75 years old (64% female)
  - Met DSM-IV criteria for Major Depressive Disorder
  - Not pregnant or breast feeding
  - 4,041 enrolled at 41 clinical sites (18 primary care + 23 psychiatric care)

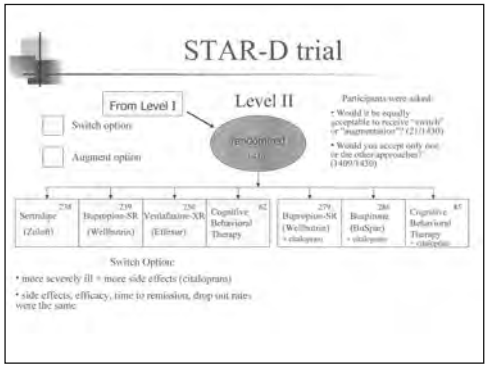
STAR-D trial

- All patients were treated for 12 weeks at each level
- All patients who achieved remission of depression could enter a 12-month follow-up phase (continue with effective medication + any psychotherapy, medication, or medication dosage change could be made)
- All patients who did not achieve remission (or were unable to tolerate their medication) were strongly encouraged to proceed to the next treatment phase (level)

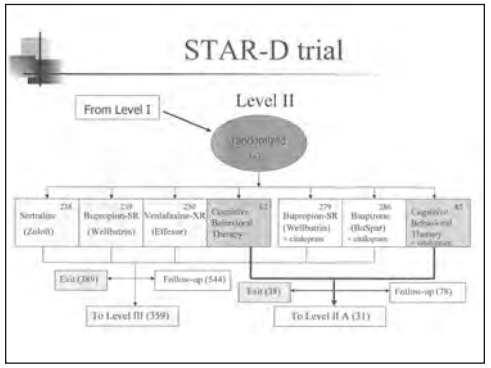
STAR-D trial



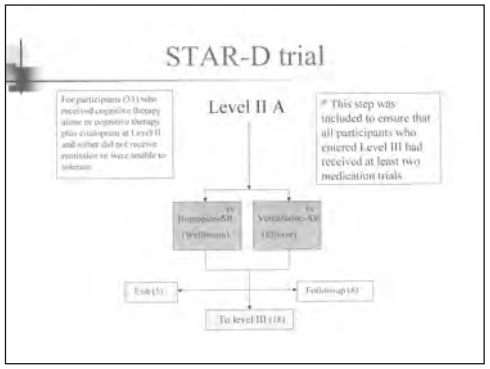
STAR-D trial



STAR-D trial



STAR-D trial



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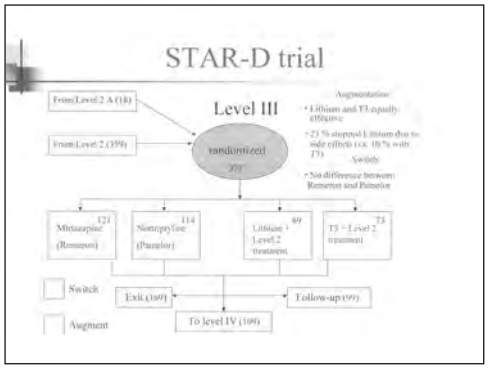
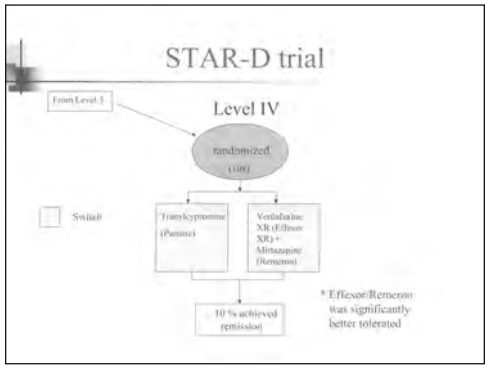
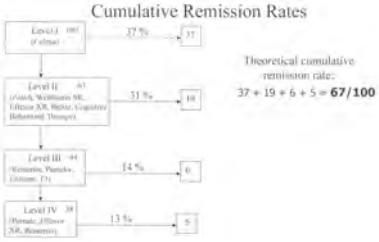


STAR-D trial



STAR-D trial

Cumulative Remission Rates



Theoretical cumulative remission rate:
37 + 19 + 6 + 5 = **67/100**

When To Stop Antidepressants

- Assess patient's current mood state
- Assess likelihood of relapse
  - Number and severity of previous episodes
  - Success of treatment of earlier episodes
  - Risk of suicide in case of relapse
  - Risk of disruption to the lives of patient and family
- **Discussion of these risk factors essential in coming to a decision about discontinuing therapy**

How to Stop Antidepressants

- Gradual tapering unless medical indications for abrupt tapering i.e.: pregnancy, severe adverse reactions, inability to take oral medications

Antidepressant Discontinuation Syndrome

- 20 % of patients after abrupt discontinuation
- More likely with longer duration of treatment and
- Shorter half-life of antidepressant
- Education of patient essential about potential problems with abrupt discontinuation

TABLE 1
Pharmacologic Properties of Selected Antidepressants

Drug	Dosage range (mg per day)	Half-life (hours)	Active metabolite?
Selective serotonin reuptake inhibitor			
Citalopram (Celexa)	10 to 60	35	No
Escitalopram (Lexapro)	10 to 30	27 to 32	No
Fluoxetine (Prozac)	20 to 80	84 to 144	Yes
Paroxetine (Paxil)	10 to 60	21	No
Paroxetine CR (Paxil CR)	12.5 to 62.5	15 to 20	No
Sertraline (Zoloft)	50 to 200	26	Yes
Atypical antidepressant			
Bupropion (Wellbutrin)	75 to 450	12 to 30	Yes
Bupropion SR (Wellbutrin SR)	100 to 400	12 to 30	Yes
Bupropion XL (Wellbutrin XL)	150 to 450	12 to 30	Yes
Duloxetine (Cymbalta)*	40 to 60	15 to 19	Yes
Mirtazapine (Remeron)	15 to 45	20 to 40	No
Trazodone (Desyrel)	50 to 400	7.1	Yes
Venlafaxine (Effexor)	75 to 450	3 to 13	Yes
Venlafaxine XR (Effexor XR)	75 to 450	3 to 13	Yes
Monoamine oxidase inhibitors			
Phenelzine (Nardil)	15 to 90	1.2	Yes
Tricyclic antidepressant*			
Amitriptyline	25 to 300*	9 to 25	Yes
Clomipramine (Anafranil)	25 to 250	22 to 64	No
Desipramine (Norpramin)	25 to 300	14.3 to 24.1	No
Doxepin (Sinequan)	25 to 300	11 to 23	No
Imipramine (Tofranil)	25 to 300	13 to 16	Yes
Nortriptyline (Pamelor)	25 to 150	18.2 to 36	No

*Dosages listed are for treatment of depression.
†Discontinued from nefazodone 20 and 21.

Clinical Manifestions

TABLE 2
Signs and Symptoms of Antidepressant Discontinuation Syndrome

	SSRI	Atypical antidepressant	Tricyclic antidepressant	MAOI
General				
Flu-like symptoms	-	-	-	-
Headache	+	-	+	-
Lethargy	+	-	+	-
Gastrointestinal				
Abdominal cramping	-	-	+	-
Abdominal pain	-	-	+	-
Appetite disturbance	-	-	+	-
Diarrhea	-	-	+	-
Nausea/vomiting	-	-	+	-
Sleep				
Insomnia	+	-	+	-
Nightmares	+	-	+	-
Balance				
Ataxia	-	-	+	-
Dizziness	+	-	+	-
Lightheadedness	+	-	+	-
Vertigo	+	-	+	-
Sensory				
Blurred vision	-	-	-	-
"Electric shock" sensations	+	-	-	-
Numbness	+	-	-	-
Paresthesia	+	-	-	-
Movement				
Akathisia	-	-	+	-
Myoclonic jerks	-	-	-	-
Parkinsonism	-	-	+	-
Tremor	-	-	+	-
Affective				
Aggression/irritability	+	-	-	+
Agitation	+	-	+	+
Anxiety	+	-	+	-
Insomnia	+	-	+	-
Psychosis				
Delusions	-	-	-	+

SSRI/SNRI Discontinuation Syndrome in **Adults**

F.I.N.I.S.H.

- **F**lu-like symptoms: fatigue, muscle aches, headache, diarrhea
- **I**nsomnia: vivid or disturbing dreams
- **N**ausea
- **I**mbalance: gait instability, dizziness, lightheadedness, vertigo
- **S**ensory disturbance: paresthesia, "electric shock" sensation, visual disturbance
- **H**yperarousal: anxiety, agitation
- Onset: 24-72 hours + Resolution: 1-14 days
- Incidence: Approximately 20-40% (who have been treated at least 6 weeks)

Maintain a High Index Suspicion

- Close questioning of for: missed doses, unreported download adjustments in doses, medication discontinuation

Differentiation From Relapse

- Shared features with Major Depression:
  - Dysphoria, appetite changes, sleep problems
  - Cognitive problems, fatigue
  - Distinguishing symptoms: dizziness, "electric shock" sensations, "rushing" sensations in the head, headache, nausea
  - Rapid reversal of symptoms after restarting AD's
  - Depression relapses: gradual worsening of depression, insomnia and psychomotor symptoms

Differentiation From Other Conditions

- Irritability, sleeplessness and anxiety raise suspicion of antidepressant induced bipolar manic episode
- A.D.S. misdiagnosed as stroke, other neuroleptic conditions, infectious diseases, adverse effects of other medications

Use of Anti-Depressants

- Psychosocial interventions recommended along with or alternatives to pharmacological therapies
- "Short-term" treatment for off-label non-mental health reasons (e.g.: irritable bowel, weight loss, insomnia, headaches) associated with early anti-depressant discontinuation

Discontinuing Medication

- Gradual tapering recommended whenever possible
- Patients forewarned of the possibility of A.D.S.
- Supervised tapering over 6-8 weeks may be required; up to 3 months after maintenance therapy

TABLE 5
Gradual Taper Rates for Antidepressants

Drug	Recommended taper rate
Monoamine oxidase inhibitor	
Phenelzine (Nardil)	Reduction of 15 mg per day every two weeks or 10 percent per week
Tricyclic antidepressant	Gradually, up to three months
Selective serotonin reuptake inhibitor	
Fluoxetine (Prozac)	Gradual taper generally unnecessary
Paroxetine (Paxil)	Reduction of 10 mg per day every five to seven days with a final dosage of 5 to 10 mg per day before discontinuation*
Sertraline (Zoloft)	Reduction of 50 mg per day every five to seven days with a final dosage of 25 to 50 mg per day before discontinuation
Atypical antidepressant	
Venlafaxine (Effexor)	Reduction of 25 mg day every five to seven days with a final dosage of 25 to 50 mg per day before discontinuation*
Venlafaxine XR (Effexor XR)	Reduction of 37.5 to 75 mg per day every week with a final dosage of 37.5 mg per day before discontinuation*

NOTE: These recommendations are expert opinion only.
*Slower taper may be needed.
Adapted with permission from Shelton RC. Steps following attainment of remission: discontinuation of antidepressant therapy. Primary Care Companion J Clin Psychiatry 2001;3:172.

Management

- Provide reassurance that condition is reversible, not serious or life threatening, will run its course in 1-2 weeks
- Consider restarting anti-depressants with slow tapering; severe symptoms should resolve within 5 days and often 24 hours
- If slow tapering poorly tolerated, an agent for longer half-life (fluoxetine) may be substituted

Tuesday, Nov. 24 – Workshop E-08

11:00 - 12:00 Occupational Medicine - Returning Patient to Work

Avi Whiteman MD, MPH, FCBOM, FACOEM
Assistant Professor, Family Medicine, McGill University
Director, Occupational Health Department, Merck Frosst Canada

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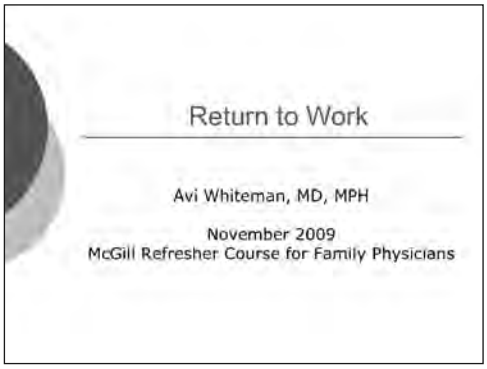
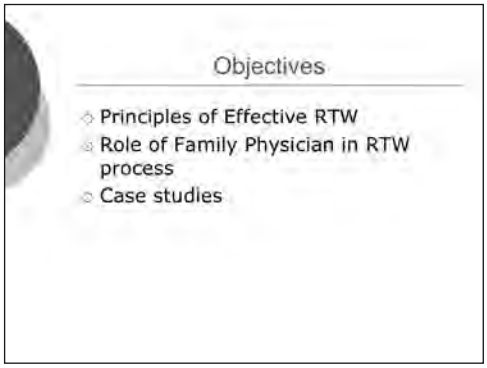
Research interests: Dr. Whiteman is a graduate of Laval University and completed his residency in Family Medicine at McGill University. He also completed a Master of Public Health at the Medical College of Wisconsin, and is a Fellow of both the Canadian and American Board of Occupational Medicine. He is the President of the Canadian Board of Occupational Medicine for the term 2009-2011. On staff at the McGill University Hospital Centre (MUHC), and Maimonides Hospital Geriatric Centre, he divides his time between hospital and office based family medicine. As director of the Occupational Health Department at Merck Frosst Canada Ltd, he oversees a team of health care professionals in the prevention of work-related disease and injury. He is also responsible for their disability program and in that capacity, interfaces with the employer, the treating physician and the patient in returning the employee to work in the most effective manner.





Outline

The Problem

Definitions

Concepts

Examples

Solutions

Family Physician perspective

RTW is not really my problem – why should I care?

Advocate for patient

Concerned about Dx, Tx

Insurance company, employer – not my concern

Ivory tower syndrome

One third of lives spent at work

The Problem

Trained to diagnose, treat

Minimal training re:

functional interface with workplace

dealing with insurance companies

Increasing demands for completing forms

Disease process demands for healing – rest?

Patient demands for time off work – pain/ fatigue/entitlement?

Assumptions

Majority of patients:

Well intentioned

Want to regain function

Want to RTW as soon as possible

Malingerers, fraudsters – the exception

80/20 "rule"

Definitions

Impairment: (*an organ-based concept*) is any loss or abnormality of psychological, physiological, or anatomical structure or function.

Disability: (*a task-based concept*) is any restriction or lack of ability to perform an activity in the manner or within the range considered normal

Disease → impairment

Job → Disability

Definitions

Limitation:

Can do, but not at the usual force, pace, duration

Restriction:

Cannot or should not do

Undue risk to self or others

What is Disability Management ?

A process in the workplace designed to facilitate the employment of persons with a disability through a coordinated effort addressing:

- Individual needs
- Work environment
- Employer needs
- Legal responsibilities

Official Disability Guidelines™

- **Most up to date evidence-based medical treatment and disability duration guidelines** to improve as well as benchmark outcomes in workers' compensation and non-occupational disability.
- Authoritative – **Based on an aggregate of over 10 million disability cases and a decade of research, including a systematic medical literature review.**

http://www.disabilitydurations.com/

Concepts

Longer time off work, the harder it is to return to work

After 6 months off work:

Only a 10% chance of return to original job

Planning for RTW begins at the first appointment

Figure 1: A temporary partial disability:

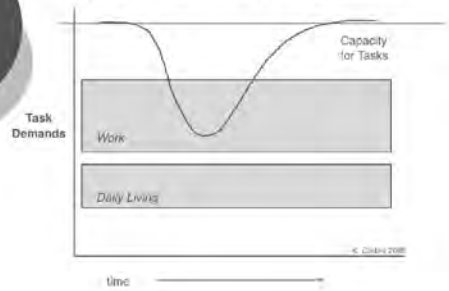


Figure 2: A temporary total disability:

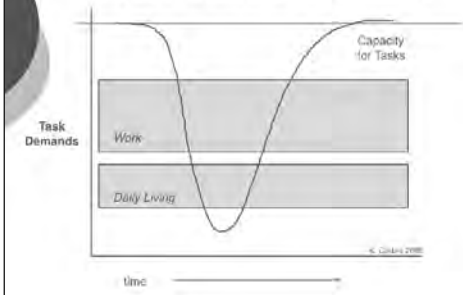
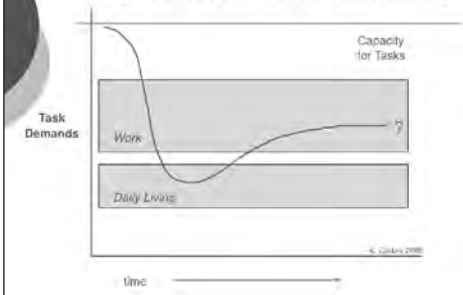


Figure 3: A permanent partial disability:



Position statement (CMA)

"The sooner a patient returns to work the more likely it is that he or she will fully regain health and productivity"

"Return to work is primarily the responsibility of the employer and employee, and that the role of the physician is to provide medical advice and support"

CMAJ, March 1, 1997 ; (156) 5

Types of Disability

1) Physical
2) Psychiatric* (highest cost)
3) Both

Need to understand what job demands are in order to translate "impairment" into "disability"

Physical demands analysis

Ask questions about job function

Don't be afraid to ask:

"Why can't you do this work ?"

About their insurance plan

% replacement, when does it kick in?

→ May drive patient requests

Assessment of patient's fitness to RTW following an injury

- describe the the mechanism of the injury, the body region affected, and the tissues/structures involved
- obtain a history of how the injury interferes with usual activities of daily living, recreation & sports, and work.
- obtain a history of the patient's job demands that are relevant to the injury
- undertake a physical examination to assess function of the affected body region

Assessment continued

- identify if the injury poses a risk to the patient or others at the workplace
- recommend limitations and restrictions that may allow for a safe and sustainable return to work
- recommend specialist, rehabilitative, psychological or vocational assessments when appropriate
- communicate medical and return to work opinions effectively, with respect for the patient's privacy and the information needs of the employer, workers' compensation board, or disability insurer.
- appreciate the boundaries and limitations of medical practice when assessing fitness to work.

When to consider psychological services

Medical indicators:

- Minimal functional gains by 8-12 weeks post injury
- Somatic symptoms or pain with few or no objective findings
- Poor compliance with prescribed treatment
- Excessive use of prescribed or non-Rx Medication

Psychological indicators

- Depressed mood, negative outlook
- Anxiety about RTW
- Anger or passivity
- Alcohol or substance abuse
- Significant disturbance in ADL's

Social Indicators

- Conflict in the workplace
- History of poor job performance
- Recent life stresses
- Prior history of prolonged disability
- Significant family conflicts

Main sources of Disability

Back pain <2000

Depression > 2000

By 2020, depression will be the leading source of work years lost from disability and premature death (WHO)

Depression: a psychiatric emergency ?

25% never RTW from a leave of absence due to depression

Early and optimal intervention is critical

Merck Frosst experience

Psychiatric Disability

Bio-Psycho-Social Model

Bio:

accurate diagnosis? Depression? Bi-Polar?

care gap

stigma: delay in seeking care

sub-optimal treatment → chronicity

Psycho-Social:

Work issues: downsizing ? Difficult boss?

financial/marital/family

percentage of salary replacement, insurance plans – STD (100%), LTD (60%), Workers compensation(90%)

Workplace stress

Psychosocial risk factors in the workplace:

Lack of fairness

Insufficient control

Few opportunities

Lack of support

Free web tool for employers:

Guardingminds@work

Documentation

- Subjective symptoms
- Objective findings*

* ...and how they impair work

Management of a short term absence

Is the recommended absence period too long?

Is there a specific plan for what should happen during this absence?

Risk of further demoralization, loss of confidence

Most depression is compatible with work (modified, part time or FT)

Absence as treatment?

Benefits of work absence

- Removed from occupational stressors
- More time to engage in activities conducive to recovery
- Less risk of workplace safety incidents

Costs of work absence

- Inactivity/withdrawal
- Social isolation
- Secondary anxiety re workplace
- Prolonged absence is negative prognostic factor
- Medicalisation of a non-medical problem?

Other options

Modified work – - Part time work

Intervention of human resources

Reintegration specialist

Case management to mediate triggering workplace interactions

Occupational Rehab programs

.....Collaborative approach

If time off: keep it as short as is medically possible

What does an employer need to know?

Why?

For work planning purposes

For insurance

What?

Limitations (task, time, work environment), aids, protective devices

But not the diagnosis, investigation or Tx plan

Elements of a good Insurance note

If consent in place:

Diagnosis

Restrictions –

Functional + anatomical restrictions, not job restrictions

Treatment plan

Still subject to standards of confidentiality (→ MD, RN; not supervisor!)

Case Studies

1. Company perspective
2. Physician perspective

Case # 1

- Employee changed department as result of restructure
- Same grade, title
- New supervisor with different management style
- Different job expectations

- Employee avoids contact with supervisor

Case # 1 Continued

Contributing Factors & Outcome
- Employee perceived friction with supervisor
- Employee afraid to approach supervisor to discuss roles & responsibilities
- Different communication styles
- Cultural differences
- Applied for STD; 72 lost work days

Lessons Learned
- Communicate: talk to your employee
- Manage expectations
- Minimize social/work withdrawal (even if off on "sick leave")
- Proactive discussions
- Discuss differences in management style (two-way discussion)
- Be ready to listen to "employee's perspective"

Case # 2

- Friday afternoon year end review with new Manager: Employee did not deliver on objectives
- Previously rated as "top performer"
- Employee shocked by comments in Year End Review

- Employee calls OHS Monday morning "tell my supervisor I will not be coming into work"

Case # 2 Continued

Contributing Factors & Outcome
- Performance expectations have changed
- Employee reaction: anger & avoidance
- Manager evaluation was based on facts
- Psychiatric expertise indicated employee was "fit for work"
- Return to work plan was established
- Employee did not return to work on the agreed upon date
- Employee terminated

Lesson Learned
- Discuss performance expectations well in advance & several times per year
- Ongoing communication – ongoing evaluation
- Avoid delivering negative messages in Friday afternoon meetings
- Focus on positive behaviours while dealing with one negative behaviour at a time

Case # 3

- Employee accepts non-comparable position
- Mid year review: new supervisor not satisfied with employee's performance
- Employee sent for training
- Frequent casual absences
- PIP initiated
- During PIP feedback session:
  "performance not improving"

Case # 3 Continued

Contributing Factors & Outcome
- Learning curve
- Expectations unclear to employee
- Perceived lack of control
- Employee felt pressured by Manager
- 59 lost work days

Lessons Learned
- Consider poor fit with job instead of going down road to PIP (non-comparable accepted instead of package?)
- Employee in wrong job?

Case # 4

- Position abolished & employee accepted comparable position with larger territory
- Employee having difficulty managing increased travel demands of new position

- Avoided areas of territory that required overnight travel

Case # 4 Continued

Contributing Factors & Outcome
- Difficulty balancing family and work obligations issues
- Afraid to discuss her needs with new manager
- Medical expertise indicated employee was "fit for work"
- Return to work plan was established
- Applied for new position
- Employee applied for STD: 32 lost days

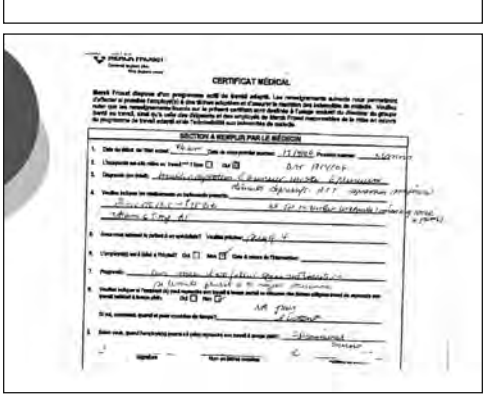
Lessons Learned
- Keep lines of communication open
- Work-Life balance considerations
- Flexible work arrangements?
- Frequent feedback -hallmark of a good manager

Physician perspective


MERCK FROSST

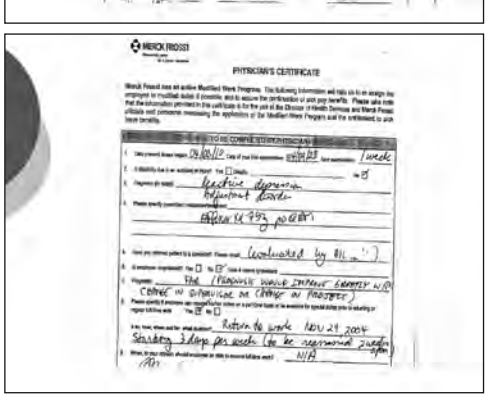
CERTIFICAT MÉDICAL


CERTIFICAT MÉDICAL


CERTIFICAT MÉDICAL


MERCK FROSST

Medical Certificate


MERCK FROSST

PHYSICIAN'S CERTIFICATE

Summary
Lessons Learned

Communicate with employees
- Proactive discussions
- Two-way discussions
- Focus on the positive
- **Avoid social/work withdrawal**
- Be ready to listen
- Timing is everything

Manage your employees
- Be clear about performance expectations (discuss them early and often)
- Provide frequent feedback
- Is it the right fit?
- Work-Life balance (consider flexible work arrangements)

Be aware of perceptions

Summary

- MD opinion highly respected
- But you are one part of insurance process
- Stick to the facts and document objective evidence
- Lose credibility if depart from medical role
- Consider planning RTW from the first visit

→ *Early RTW improves medical outcomes*

A Physician's Guide to Return to Work

ISBN: 978-1-57947-628-7

American Medical Association
List Price: $59.95

Work-related Musculoskeletal Disorders - Guide and Tools for Modified Work
ISBN: 2-89494-430-6

IRSST publications
List Price: $20.00

References

Alberta Medical Association Position Statement:
- "Early Return to Work after Illness or Injury" (1995)
- "The Physician's Role in Helping Patients Return to Work after an Illness or Injury" *CMAJ* 156(5) 1997.
- "Injury/Illness and Return to Work/Function" Physician Education Project in Workplace Health (PEPWH) 2000
- Corbet, K. "The Principles and Practice of Return to Work Assessments" *The Canadian Journal of CME* pp 197-210 (September 2000).

Notes

Tuesday, Nov. 24 – Lunch Symposium

12:00 - 12:45 Lunch Satellite Symposium

Chair • **David Dannebaum**

Dislipidemia: Prevention of Cardiovascular Disease

Morris Schweitzer MD

Pathways to Optimal Clinical Outcomes

Supported through an unrestricted educational grant from Merck-Frosst.

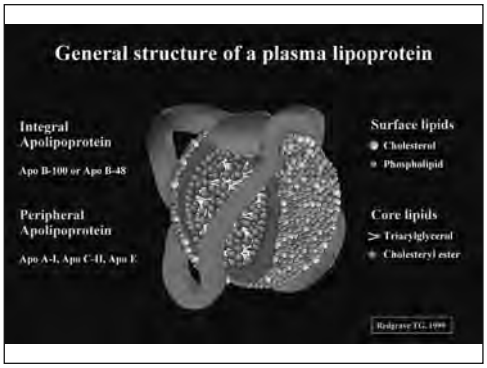
Notes

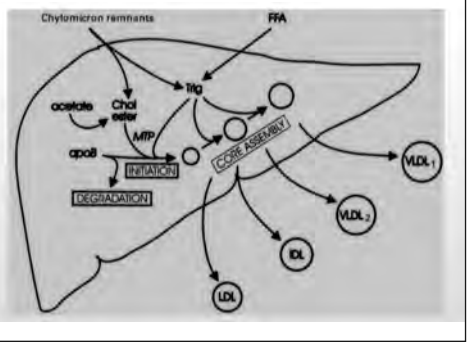
Dyslipidemia: Prevention of Cardiovascular Disease

Morris Schweitzer Ph.D. , M.D. F.R.C.P. (C)

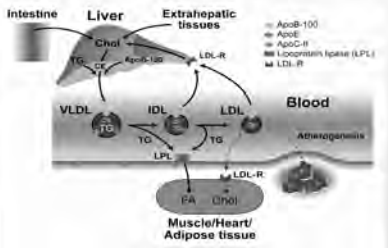
November 24, 2009

General structure of a plasma lipoprotein

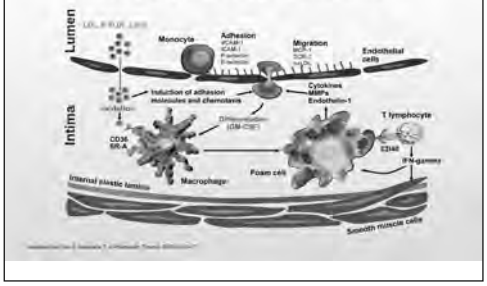


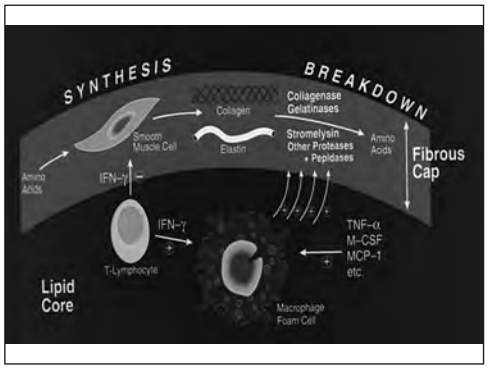


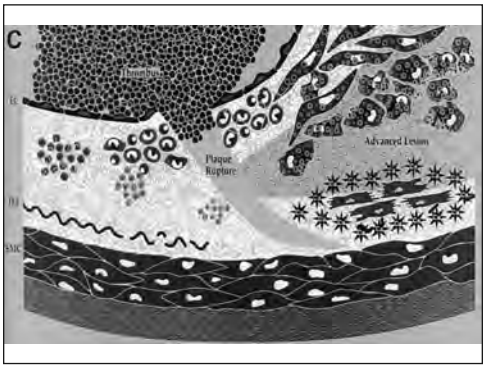
VLDL and LDL Cholesterol Transport



Development of an Atheroma

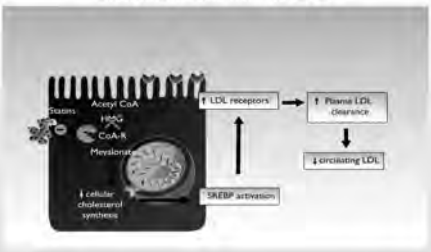


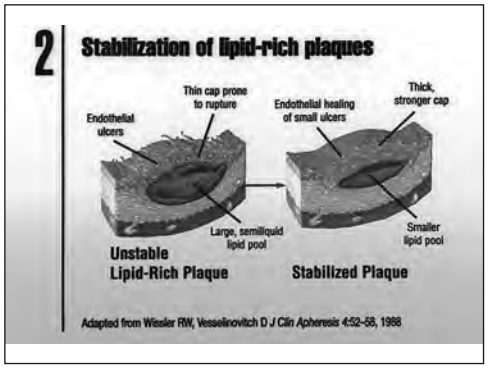


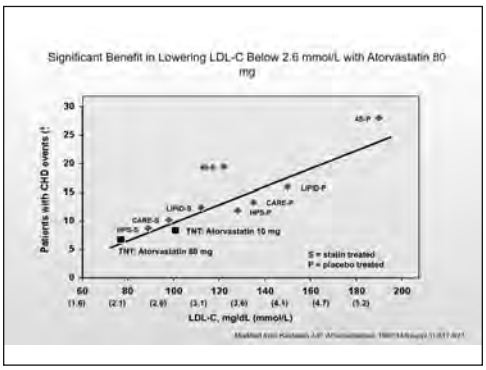


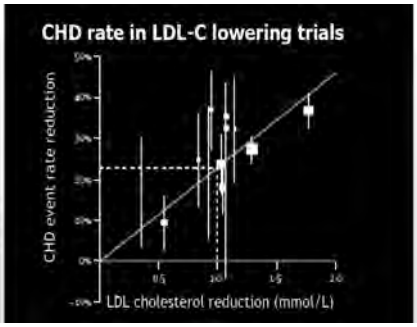
Statins

Molecular mechanisms of action





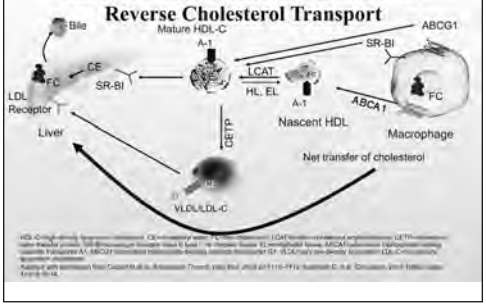




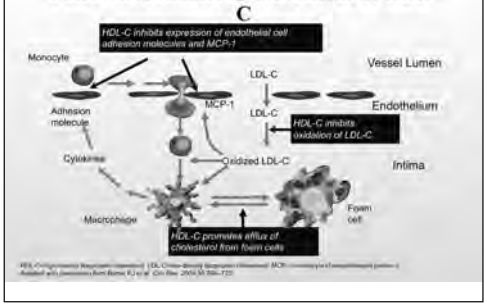
Residual risk of CHD events remaining

Trial (N)	Statin treatment	Clinical events[a] Risk reduction vs placebo
WOSCOPS (6595)	Pravastatin 40 mg	31%
AFCAPS/TexCAPS (6605)	Lovastatin 20/40 mg	37%
ASCOT-LLA (10,305)	Atorvastatin 10 mg	36%
4S (4444)	Simvastatin 20 mg	28%
CARE (4159)	Pravastatin 40 mg	24%
LIPID (9014)	Pravastatin 40 mg	24%
HPS (20,536)	Simvastatin 40 mg	27%
PROSPER (5804)	Pravastatin 40 mg	19%

Current Model of HDL-C Metabolism: Reverse Cholesterol Transport

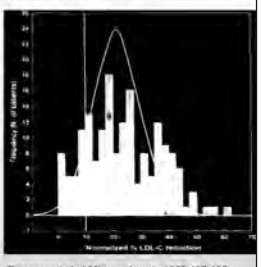


Potential Antiatherogenic Actions of HDL-C



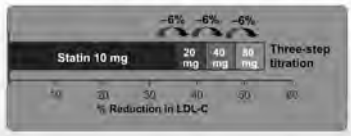
Wide Variation of Response to Statins

- % LDL reduction to simvastatin or pravastatin up to 60mg/d for 3 months
- Mean reduction 20%
- 12% had LDLC ↓ < 10%
- 5% no ↓ LDLC
- Reduction LDLC not related to
  - Baseline LDLC
  - BMI



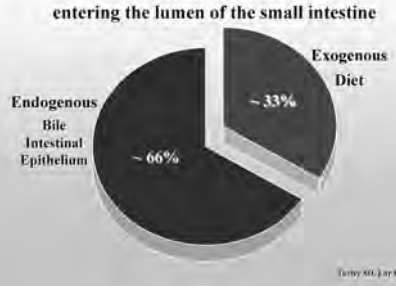
Pazzucconi et al Atherosclerosis 1995;117:189

Effect of statin therapy on LDL-C levels: "The Rule of 6"



Stein E. Eur Heart J 2001; 3(Suppl E):E11-E15

Sources of cholesterol entering the lumen of the small intestine



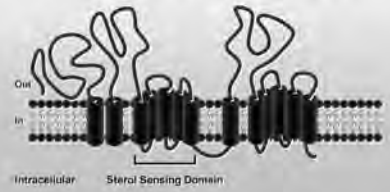
Turley KR. J Cardiovasc Pharmacol 1999

Pharmacology of ezetimibe intestinal localization

- Ezetimibe localizes to the brush border membrane of intestinal epithelial cells, primarily as glucuronidated derivative
- Rapid onset of action (< 90 min.)
- Ezetimibe and its glucuronidated derivative circulate enterohepatically
  - Repeated delivery to the site of action
  - Limited systemic exposure
  - Long biologic half-life (22 hrs.)

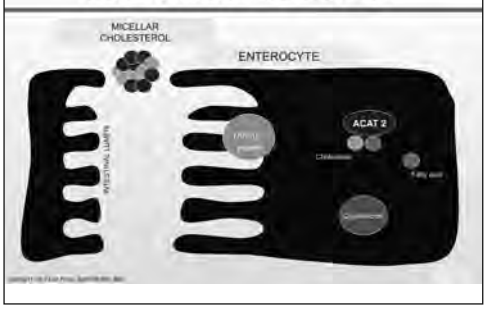
Van Heek M in al. Br J Pharmacol 2000

Niemann-Pick C1 like 1 protein is critical for intestinal cholesterol absorption

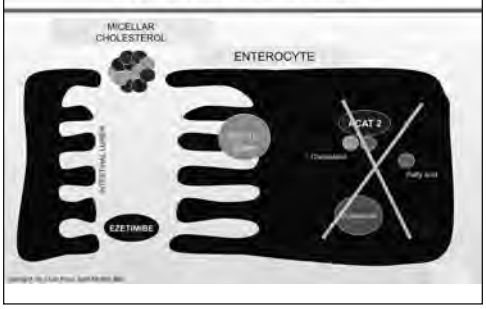


Altmann SW et al. Science 2004

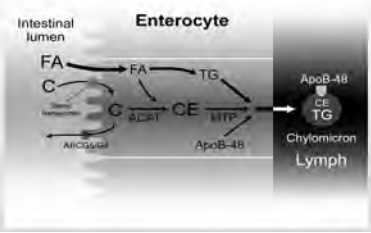
SELECTIVE CHOLESTEROL ABSORPTION



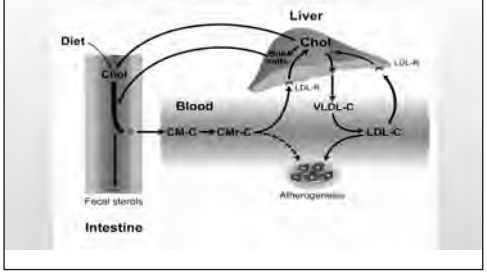
MODE OF ACTION OF EZETIMIBE



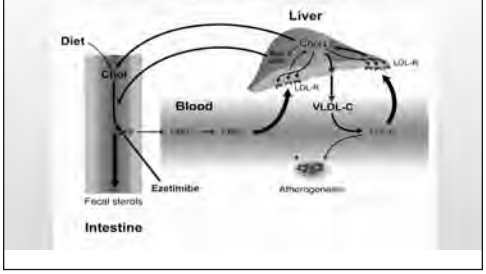
Transport of Intestinal Cholesterol

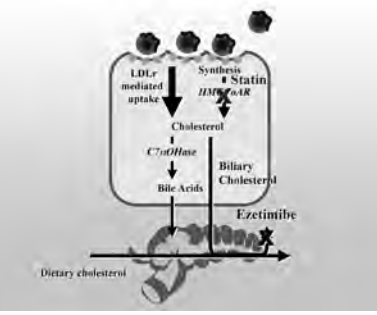


Ezetimibe: Reduction of Cholesterol Content in Atherogenic Lipoproteins



Ezetimibe: Reduction of Cholesterol Content in Atherogenic Lipoproteins

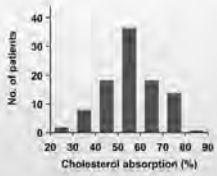




Characteristics of Cholesterol Absorption

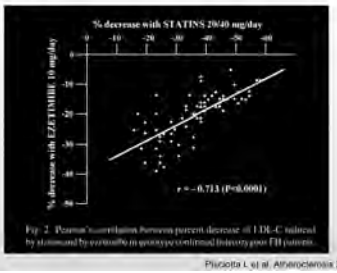
1. Efficiency

The efficiency of cholesterol absorption varies greatly within and between species.



Bosner MS et al. J Lipid Res. 1999;40:302

Strong Negative Correlation Between the LDL-C Response to Statins and the Subsequent Response to Ezetimibe



Pirillo A et al. Atherosclerosis 2008

Triple Therapy

- Fibrates
  - VA-HIT Positive
  - FIELD Negative
- Niaspan
  - ? AIM-HIGH
- I use triple therapy in patients with very high triglyceride levels where there is a significantly increased risk for pancreatitis

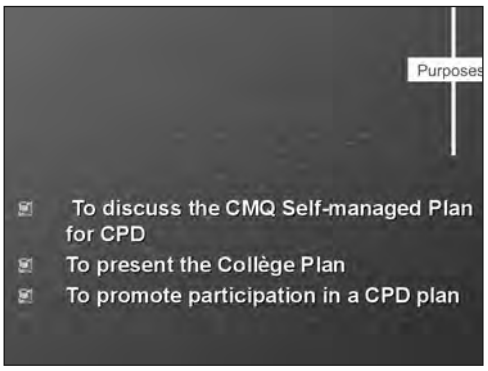
What's new in lipids?

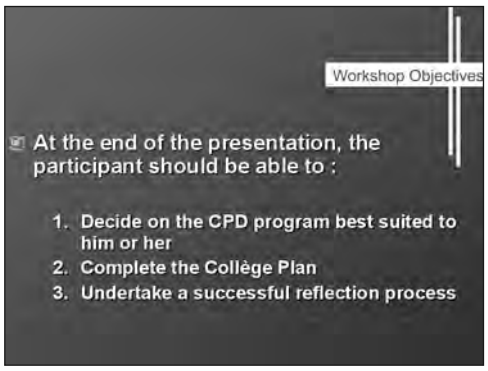
- MTP Inhibitors
- Squalene synthesis inhibitors
- Antisense Apolipoprotein B oligonucleotides
  - Hybridize to a complementary mRNA
  - Results in selective degradation of Apo-B mRNA
- Add on to statin
  - Weekly subcutaneous injection
  - Decreases LDL 30-50%

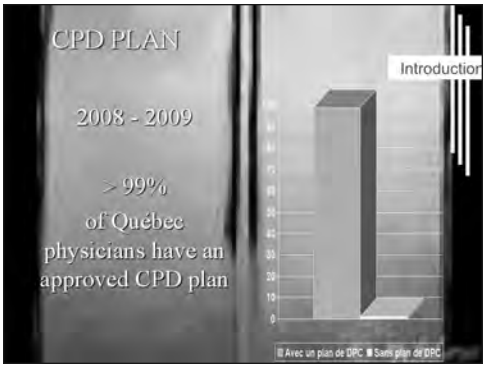
Notes

13:30 - 14:00 CPD/CME Requirements by the Collège des médecins du Québec

Roger Ladouceur MD, MSC, CCMF, FCMF
Physician in charge of the Self-managed Plan for Continuing Professional Development, Practice Enhancement Division, Collège des médecins du Québec
Professeur agrégé, Department of Family Medicine, Université de Montréal
Associate Editor, Canadian Family Physician
Family Practioner, Verdun Hospital Centre

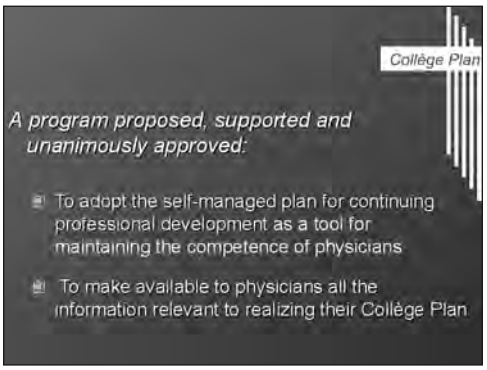


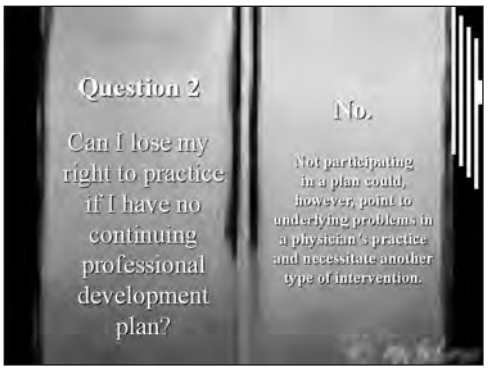


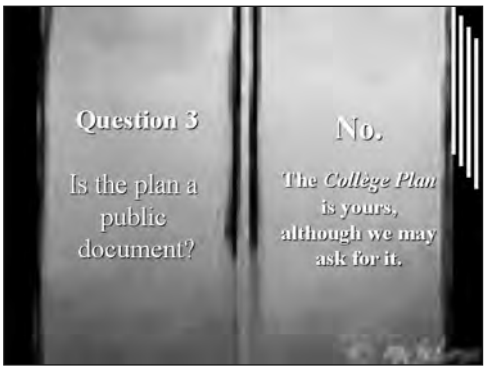

CPD PLAN
2008 - 2009
> 99%
of Québec
physicians have an
approved CPD plan
Introduction
Avec un plan de DPC
Sans plan de DPC

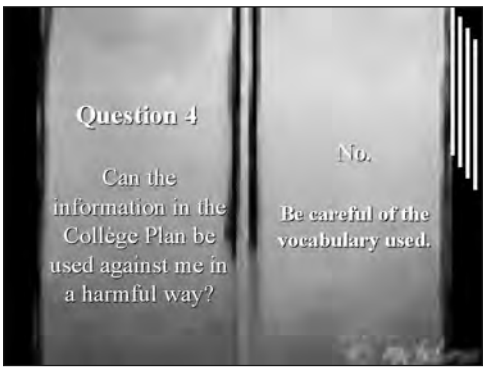

The 10
most frequently
asked questions
on the
Collège Plan
Introduction

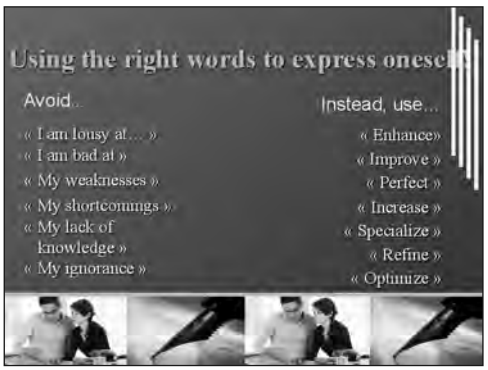

Question 1
Is the Plan
mandatory?
Reply to question
Answer :
No.
Only maintaining
one's professional
competence is
mandatory
(moral and ethical
responsibility)


Collège Plan
A program proposed, supported and
unanimously approved:
To adopt the self-managed plan for continuing
professional development as a tool for
maintaining the competence of physicians
To make available to physicians all the
information relevant to realizing their Collège Plan

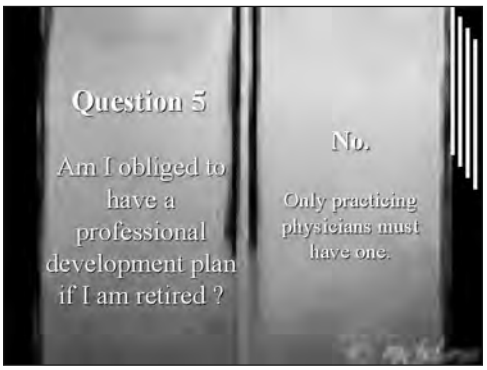

Question 2
Can I lose my
right to practice
if I have no
continuing
professional
development
plan?
No.
Not participating
in a plan could,
however, point to
underlying problems in
a physician's practice
and necessitate another
type of intervention.

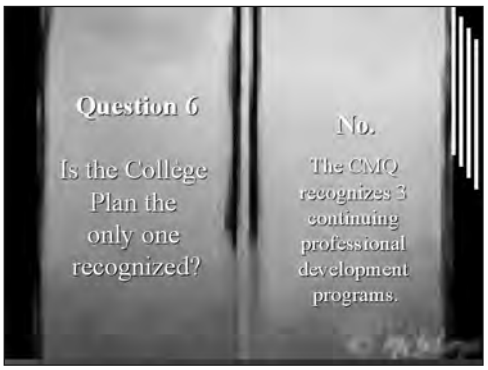

Question 3
Is the plan a
public
document?
No.
The Collège Plan
is yours,
although we may
ask for it.


Question 4
Can the
information in the
Collège Plan be
used against me in
a harmful way?
No.
Be careful of the
vocabulary used.

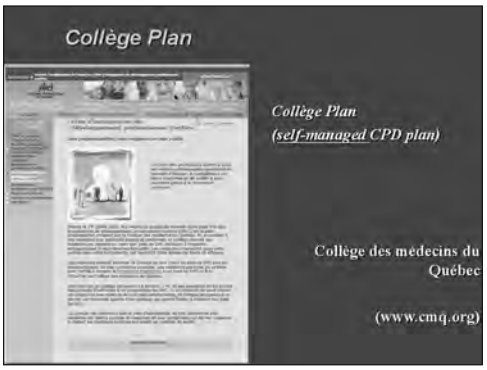

Using the right words to express oneself
Avoid...
« I am lousy at... »
« I am bad at »
« My weaknesses »
« My shortcomings »
« My lack of
knowledge »
« My ignorance »
Instead, use...
« Enhance»
« Improve »
« Perfect »
« Increase »
« Specialize »
« Refine »
« Optimize »

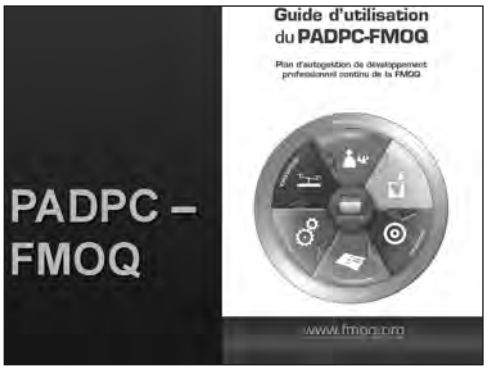

...and the legal aspects???


Question 5
Am I obliged to
have a
professional
development plan
if I am retired ?
No.
Only practicing
physicians must
have one.

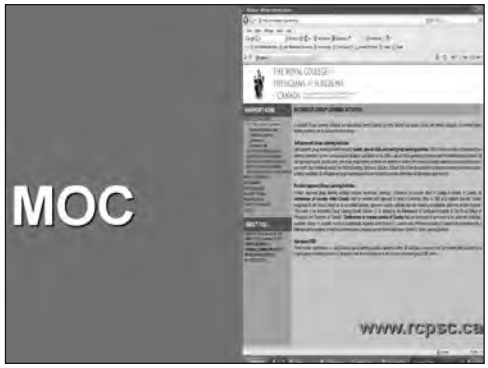

Question 6
Is the Collège
Plan the
only one
recognized?
No.
The CMQ
recognizes 3
continuing
professional
development
programs.

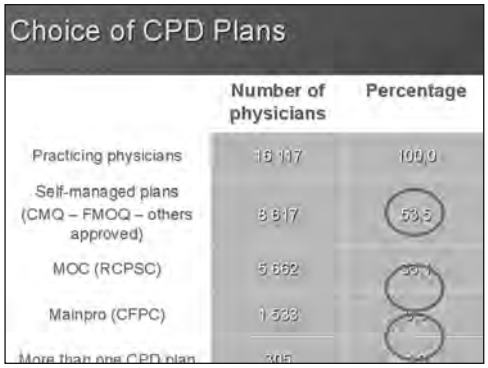

The 3
recognized
plans
(CMQ-PADPC-FMOQ,
[others approved])
MOC of RCPSC
Mainpro of CFPC
The physician chooses the one he or
she prefers.

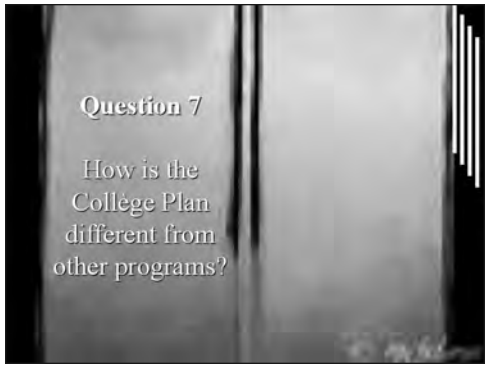

Collège Plan
Collège Plan
(self-managed CPD plan)
Collège des médecins du
Québec
(www.cmq.org)


PADPC –
FMOQ
Guide d'utilisation
du PADPC-FMOQ
Plan d'autogestion de développement
professionnel continu de la FMOQ
www.fmoq.org


Mainpro
www.cfpc.ca



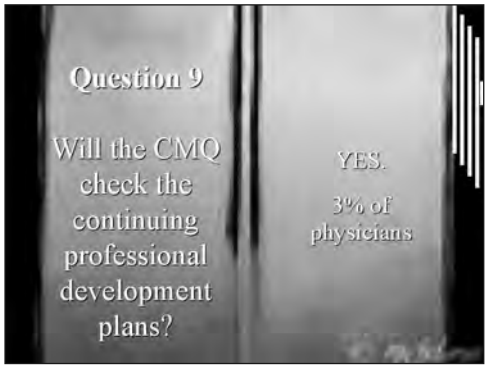


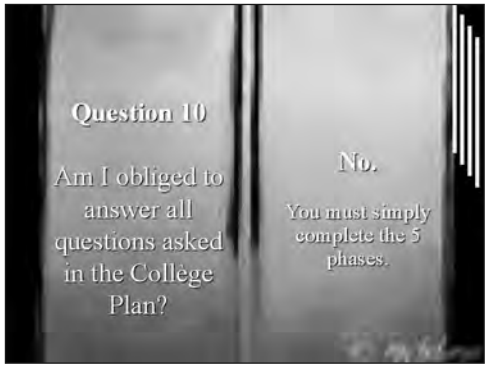


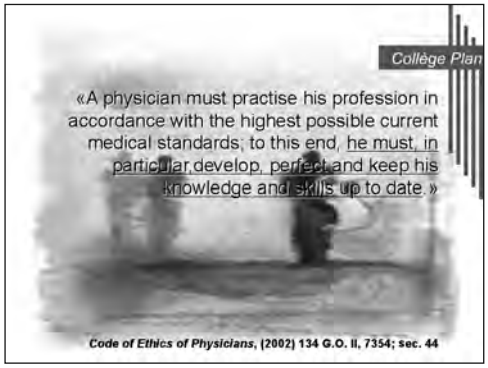
	Self-managed plans (CMQ / PADPC–FMOQ / Other approved)	CFPC	RCPSC
Evaluation of practice	√	Optional	Optional
Deciding on objectives	√	Optional	Optional
Search for information	√	Optional	Optional
Evaluation of learning	√	Optional	Optional
Summary of activities	√	√	√

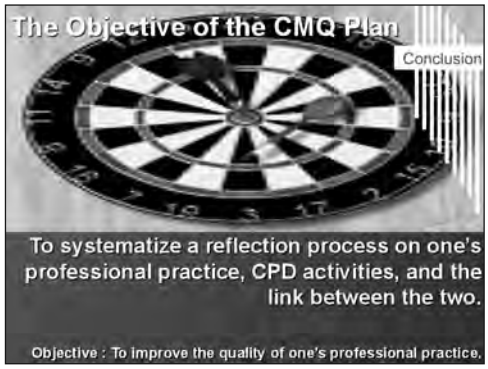
	Self-managed plans (CMQ / PADPC–FMOQ / Others)	CFPC	RCPSC
Requirements	Reflection process (analysis of practice) 5 phases Number of credits (N/A)	250 credits / 5 years (min. 125 Mainpro M-1 ou C)	400 credits / 5 years (max. 100 Section-2)
Cost	No cost	Member : No cost (If not : 355$/year)	Member : No cost (If not : 355$/year)
Required annual listing of activities	Yes	Yes (CQMF)	Yes
On-line registration	Yes	Yes	Yes

Self-managed plans (CMQ / PADPC–FMOQ / Others approved)	CFPC	RCPSC
No minimum required	250 credits / 5 years (min. 125 M-1 / C)	400 credits / 5 years (max. 100 Section-2) (min. 40 cr / year)









Notes

COLLÈGE DES MÉDECINS DU QUÉBEC

Une médecine de qualité au service du public

SELF-MANAGED PLAN FOR CONTINUING PROFESSIONAL DEVELOPMENT

IDENTITY

Permit No. _____ Year: 20____ – 20 _____

Name: _____ First name: _____

Family practitioner ❒ Specialist ❒ (specify) _____

Form to be completed annually (See User's Guide)

PHASE 1 – MY PRESENT PROFESSIONAL PRACTICE

For example:

- Field of practice (emergency, hospital, outpatient clinic, private practice, house calls, clinical research, etc.)
- Type of practice (admitted patients, walk-in clinic, youth clinic, occupational health, etc.)
- Age groups Diagnoses frequently made
- Practice profile Prescription profile

	COMMENTS

Cross-disciplinary skills[1]	COMPETENCE LEVEL*	INTEREST LEVEL*	PRIORITY LEVEL*	COMMENTS
1. Medical expert (clinical care)				
2. Communicator				
3. Collaborator				
4. Manager				
5. Health Advocate				
6. Scholar				
7. Professional				

* **Assess the level on a scale of 1- (low level) to 5 (very high level)**

[1] Adapted from Frank, JR. (Ed). 2005. The CanMEDS 2005 physician competency framework. Better standards. Better physicians. Better care. Ottawa: The Royal College of Physicians and Surgeons of Canada.

PHASE 2 – MY GOALS

2.1. Where am I at?

In my various fields of practice

What are my strong points?	*What points do I need to improve?*

2.2 What are my needs in terms of education?

What aspects of my practice could be improved over the coming year?

2.3 What learning activities will I take part in during the coming year?

PHASE 3 – MY CPD ACTIVITIES AND THEIR IMPACT ON MY PRACTICE

TITLE OF CPD PROGRAM OR ACTIVITY	DATE	DURATION (HOURS)	EFFECT ON MY PRACTICE*	ACTIONS TAK EN

* :
0 *The CPD activity did not apply to my practice.*
1 *The CPD activity confirmed that my practice was appropriate.*
2 *The CPD activity resulted in a change in my practice.*
3 *As a result of the CPD activity, I must seek additional information or acquire other skills.*

PHASE 4 – MY REFLECTION PROCESS AND ADJUSTMENTS

At the end of the year (in June, for example), what actions must I still take? How soon?

_____

_____

_____

_____

_____

_____

_____

After completing this stage, you may return to stage 1 of the CPD self-managed plan for the next year, thus beginning the cycle again.

PHASE 5 – MY ANNUAL SUMMARY OF CPD ACTIVITIES

NUMBER OF HOURS/YEAR

5.1 Individualized structured learning projects _____

- ❑ CMQ self-learning project
- ❑ Personal project (section IV of the RCPSC)
- ❑ Mainpro-C (CFPC), training period, tutorial CMQ self-learning project
- ❑ Master's or doctorate program of studies

5.2 Practice reviews _____

- ❑ Evaluation of my personal practice by the CMQ or other organization
- ❑ Participation in a committee evaluating medical acts related to my field of practice
- ❑ Participation in a risk management committee related to my field of practice

5.3 Accredited or unaccredited individual activities _____

- ❑ Lectures
- ❑ Internet
- ❑ Self-training modules

5.4 Teaching and research _____

- ❑ Publications, presentations, courses
- ❑ Writing of protocols/grant applications

5.5 Accredited group activities _____

- ❑ Conventions, conferences, workshops
- ❑ Accredited educational activities in an institution

5.6 Unaccredited group activities _____

- ❑ Conferences, other
- ❑ Non-certified educational activities

DATE _____
(DD-MM-YYYY)

14:00 - 14:30 Antibiotic Prophylaxis

Michael D. Libman MD
Department of Medical Microbiology and Division of Infectious Disease, MUHC;
Associate Professor, Faculty of Medicine, McGill University

Research interests: Dr. Libman is currently Director of the Division of Infectious Diseases at McGill University and the McGill University Health Centre, and interim director of the J.D. MacLean Centre for Tropical Diseases at McGill University. He is also affiliated with St. Mary's Hospital, and consultant in microbiology and infectious diseases for the Quebec arctic region known as Nunavik. His primary interest is in tropical and travel medicine, as well as laboratory parasitology.

Learning objectives:

- To recognize the importance of malaria prophylaxis, and the situations where it is indicated
- To become familiar with the available resources for obtaining information on malaria
- To be aware of the risk in the most popular tourist destinations

- To understand the rationale and indications for antibiotic prophylaxis of bite wounds
- To be able to assess the risk for rabies infection after a bite injury
- To know the procedures for rabies prophylaxis

Prophylaxis

- Malaria
- Bites
  - Wound prophylaxis
  - Rabies

November 2009 Michael Libman M.D. 1

Malaria Prevention

Malaria Chemoprophylaxis

November 2009 Michael Libman M.D. Infectious Disease/Microbiology MUHC 2

The Pyramid of Prevention

D Diagnosis: prompt Dx and early Rx

C Compliance with Chemoprophylaxis

B Bites: Personal Protection Measures

A Awareness: know the risk

3

The Pyramid of Prevention Level A: Awareness

- Patient and physician
- Prevention strategies
- Pre-travel, during travel & post-travel
- Prevent death and complications
- Verbal, written, video

Awareness & Education

4

The Pyramid of Prevention Level B: Bites

Bites = Personal Protection Measures

Awareness = Education

5

What is the best way to prevent infection?

6

The mercurial malariologist, Sir Ronald Ross:

"I myself have been infected with malaria only once in spite of nineteen years service in India and thirteen subsequent malaria expeditions to warm climates; I attribute this good fortune to my scrupulous use of the bed net"

Ross R. Memoirs with a full account of the great malaria problem and its solution. London: Murray, 1923.

7

The Pyramid of Prevention Level C: Chemoprophylaxis

Compliance with Chemoprophylaxis

Bites: Personal Protection Measures

Awareness: know the risk

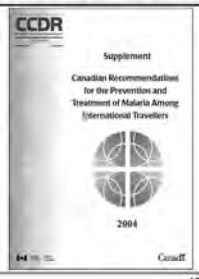
8

Chemoprophylaxis !!!

- The bitter taste of quinine was masked by gin
- Tonic water today still has quinine!

9

How do we choose chemoprophylaxis Drugs?



10

CQ sensitive *P. falciparum*

11

http://www.cdc.gov/malaria/map/

26 May 13

Available Drugs

- Chloroquine sensitive:
  - Chloroquine (Aralen)
- Chloroquine resistant
  - Mefloquine (Lariam)
  - Atovaquone/proguanil (Malarone)
  - Doxycyline
  - Primaquine

14

Value of Chemoprophylaxis

- > 90% of cases of all malaria associated with:
  - inappropriate chemoprophylaxis
  - no chemoprophylaxis
- Almost all deaths and ICU cases associated with:
  - no chemoprophylaxis

15

Prophylaxis for Bites (Human and Animal)

Probability of infection

- Presenting <8 hrs. After injury:
  - Present for "cosmetic" treatment
  - among those treated: 2-30%
- Presenting >8 hrs:
  - Most already infected
- ↑risk: puncture
  - arthritis, osteomyelitis, tenosynovitis, abcess
  - Edema, lymphatic obstruction, immunosuppressed

Prophylactic antibiotics

- Early culture does not predict infection
- Most wounds contain potential pathogens
  - Pasteurella, streptococcus, Staph aureus, anaerobes, Staph intermedius
  - Pasteurella infection typically <36hrs
- For some wounds, antiobiotic for 3-5d probably prudent

Indications for prophylaxis (<8 hrs)

- ++ edema, or crush
- Bone or joint penetration possible
- Hand injury (tenosynovitis)
- Other "critical site" eg: genital, face
- Immunocompromised, steroids, liver failure
- Lymphatic obstruction

Choice of antibiotic

- NOT ACCEPTABLE: clox, oral ceph, macrolide
  - Pasteurella, Eikenella, anaerobes
- Clavulin 500 /125 tid or 875/125 bid
- Pen or Amox acceptable early (Staph aureus less likely)
- ?Moxi (in theory). Clinda/cipro?, doxy (early)
- Serpents: gram neg rods

Rabies prophylaxis

- Type of exposure
- Type of animal
- Availability of animial
- Information on geographic area
- Health and behaviour of animal

Animal Rabies 2008
Quebec

- Entirely Eastern Townships, Richelieu, Beauce
  - No domestic animals
  - 26 racoons, 6 skunks, 4 bats, 1 fox
  - Previous years occ. bat Montreal and north
- 2 human cases since 1960 in Québec
- 5 human cases since 1970 in Canada
  - 4/5 = bats

Interventions

- Domestic Animal: observation 10d.
- Squirrel: none (maybe unprovoked attack by furious aggressive animal)
- Wild animal, bat: evaluation, prudence
  - Plausible exposure (bite or scra =significant
    - History difficult for children etc

Post exposure prophylaxis

- Careful lavage of wound, antiseptics
- HRIG 20 UI/kg around wound + IM
- Rabies vaccine: 5 doses
  - Upto 1 year later?
- Questions:
  - direction de la santé publique
  - Canadian Food Inspection Agency 450-476-1223

Vaccination: side effects

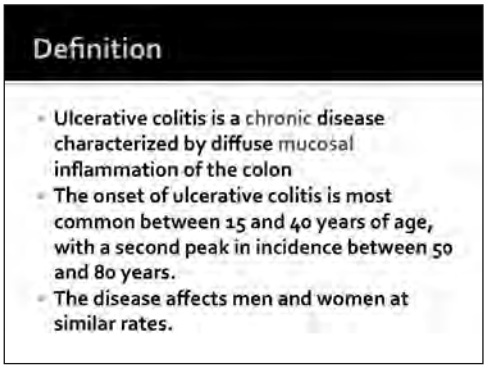
- Local reactions 30-70%
  - More intradermal than IM
- Systemic reactions 20%
  - N/V/D, abdo pain, myalgia, arthralgia, fever, rash
- Montréal 2000: 1051 doses
  - 12 reported reactions
  - allergic: 3
  - Stevens-Johnson: 1

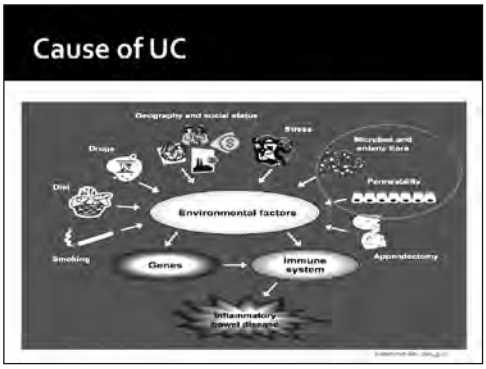
Notes

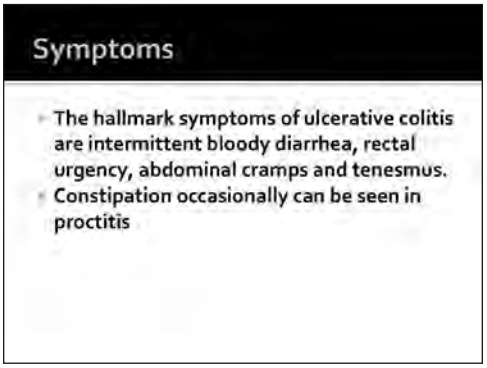
14:30 - 15:00 Managament of Ulcerative Colitis

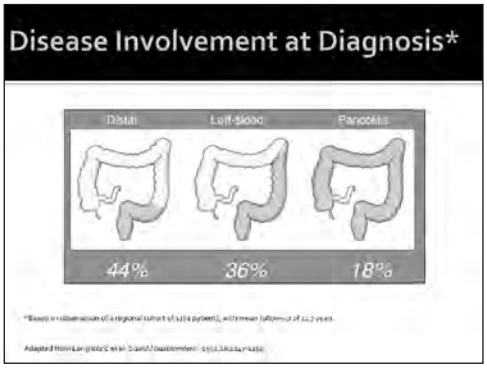
Gad Friedman MDCM, FRCP
Division of Gastroenterology, McGill University & MUHC;
Assistant Professor, School of Medicine, McGill University

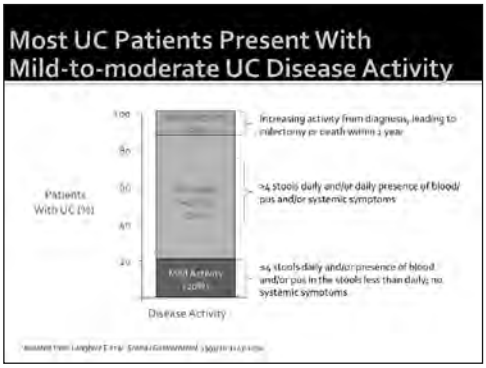
Research Interests: I have been a member of the Division of Gastroenterology of the Jewish General Hospital for over 11 years. After finishing medical school at McGill University, I completed m,y Internal Medicine residency at the Jewish General Hospital followed by a Gastroenterology fellowship at McGill. I subsequently did a year fellowship in interventional endoscopy with a focus on ERCP at the Montreal General Hospital. Although my primary interest is endoscopy with a focus on pancreaticobiliary disease, I enjoy a varied practice with large segment devoted to patients with inflammatory bowel disease. In the past two years, I have become involved in capsule endoscopy and hopfully will be starting capsule endoscopy at the Jewish General in the near future.

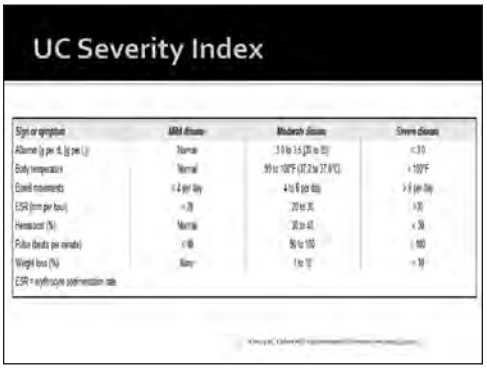


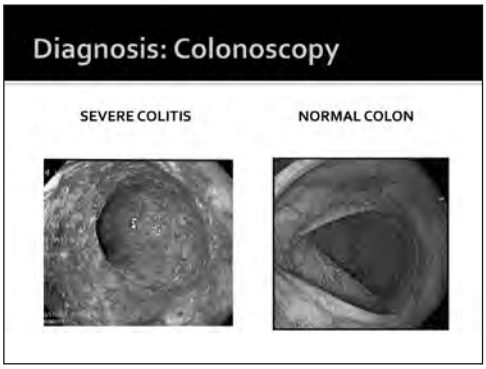












Other tests

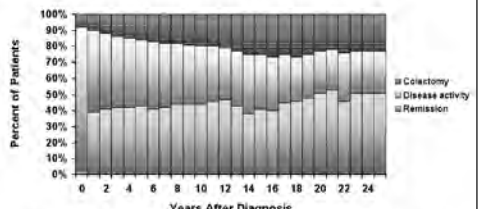
BLOOD OR STOOL

- CBC, iron studies
- Liver and renal function
- CRP (or ESR)
- Albumin
- Stool for culture, O/P and C. difficile

RADIOLOGY

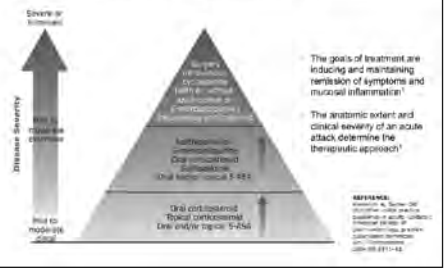
- CT can help show extent
- Bone density
- SBFT to rule out Crohn's disease

Natural History of Ulcerative Colitis*



*Percent of patients with disease activity, in remission, or having colectomy performed each year after diagnosis

UC treatment strategies



5-Aminosalicylate (Mesalamine)

ORAL 5-ASA

- Asacol 400 or 800 mg
- Pentasa 500 mg
- Salofalk 500 mg
- Mezavant 1200 mg

TOPICAL 5-ASA

- Suppositories
  - Salofalk 250, 500, 1000 mgs
- Enemas
  - Salofalk 1 or 4 gms
  - Pentasa 1, 2, or 4 gms

Treatment of Active Distal UC

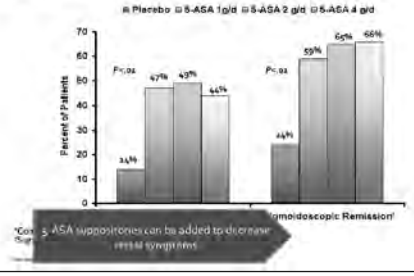
- suppositories reach the upper rectum
- enemas reach splenic flexure and the distal transverse colon

Proximal distribution of topical preparations

Adapted with permission from: Marshall JK, Irvine EJ. Am J Gastroenterol 2000; 95: 1628-1638

Oral 5-ASA Release Sites

Rectal 5-ASA Enema: Acute Proctosigmoiditis

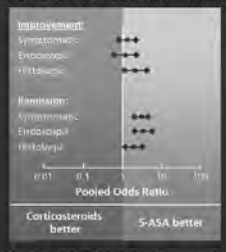


Topical Steroids

- Foam
  - Cortifoam (10% HC acetate)
- Enemas
  - Cortenema (HC 100 mg)
  - Betnesol (Betamethasone 5 mg)
  - Entecort enemas (Budesonide)

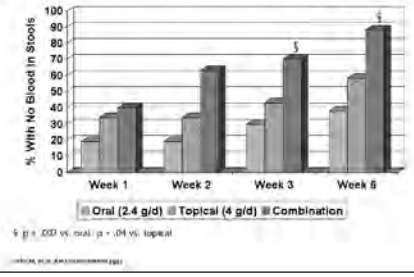
Meta-Analyses of Rectal 5-ASA in UC

Rectal 5-ASA vs Rectal Corticosteroids

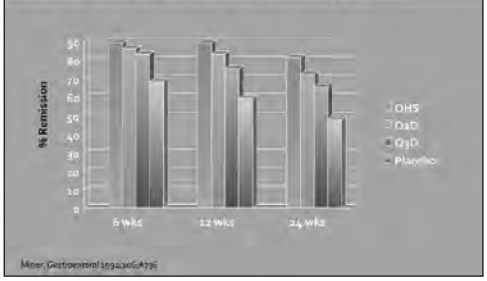


Adapted from Marshall JK, Irvine EJ. Gut. 1997;40:775-781 with permission from the BMJ Publishing Group

Treatment of Active Distal UC: Oral, Topical or Combination 5-ASA?

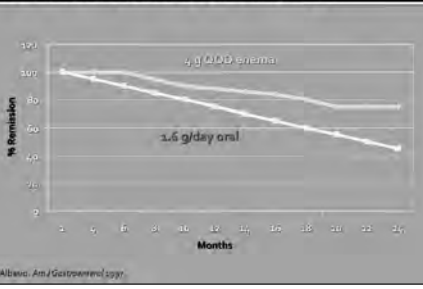


Frequency of Topical Mesalamine for Maintenance of Distal UC



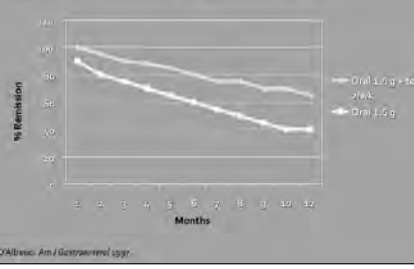
Miner. Gastroenterol 1994;106:A736

Oral vs. Topical Mesalamine for Maintenance of Distal UC



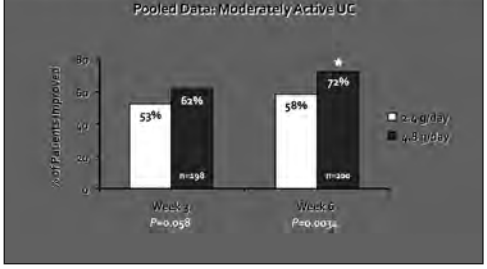
D'Albasio. Am J Gastroenterol 1997

Combined Oral + Topical Mesalamine for Maintenance of Distal UC



D'Albasio. Am J Gastroenterol 1997

ASCEND I & II: Treatment Success at Weeks 3 & 6

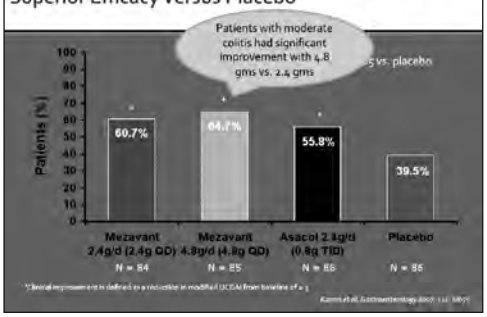


Compliance Issues

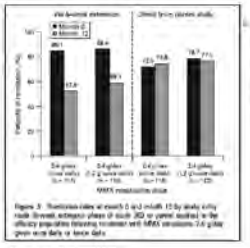
- 60% of patients do not take their medications as prescribed.
- Patients who are non-adherent, most commonly single young men or those taking multiple concomitant medications, have a fivefold increased risk of disease relapse compared with patients who take at least 80% of their prescribed dose.

Mezavant: How the Multi Matrix System works

Clinical Improvement at Week 8[1] : Statistically Superior Efficacy Versus Placebo

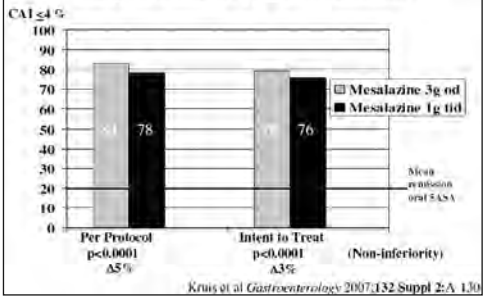


Randomized trial of once-or twice daily MMX mesalazine for maintenance of remission in Ulcerative colitis



- Patients in remission on MMX mesalazine were enrolled in a maintenance trial

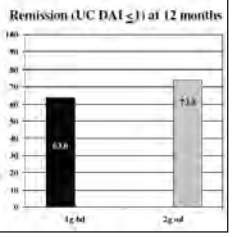
Salofalk® 3g once daily vs 1g three times daily at 8wk: CAI ≤4 'remission'



Kruis et al *Gastroenterology* 2007;**132 Suppl 2**:A 130

PODIUM Results

- n = 362
- Treatment difference 11.9% (p = 0.024)
- Better compliance (% sachets used) with once daily
- Patient acceptability better with once daily (p<0.001)



Dignass et al. UEGW 2007 (late breaking abstract)

Adverse Effects of 5-ASA

- Pericarditis, myocarditis
- Pneumonitis, pleuritis
- Pancreatitis
- Hepatitis
- Cytopenia
- Lupus-like syndrome
- Exacerbation of colitis
- Diarrhea
- Skin rash
- arthritis

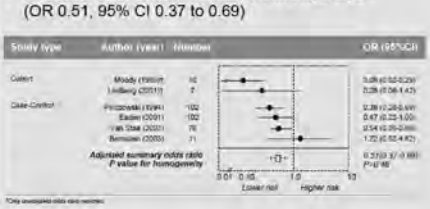
Interstitial nephritis has been reported with 5-ASA, but is likely related to the underlying disease

Risk of Colon Cancer in UC

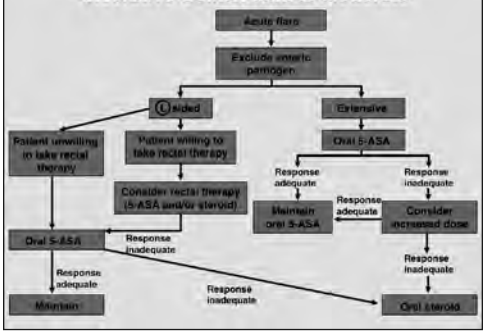
- A meta-analysis conducted in 2001 calculated the risk of CRC for an individual with UC (across severity levels) as
  - 2% at 10 years
  - 8% at 20 years
  - 18% at 30 years

5-aminosalicylic acid protective against colorectal cancer

- Protective association between 5-ASA and CRC (OR 0.51, 95% CI 0.37 to 0.69)



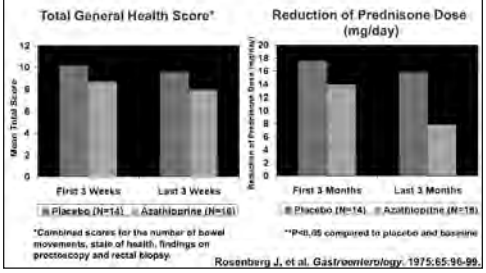
Ulcerative Colitis: Mild to Moderate



Conclusions

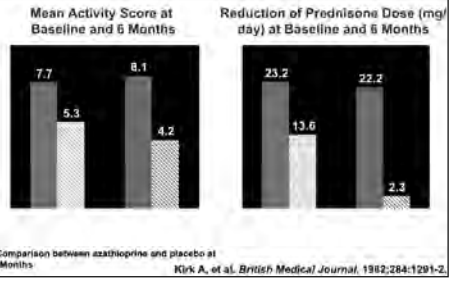
- Most patients with UC have distal disease of mild to moderate activity and can be easily managed with oral and topical 5-ASA
- Once patients need oral steroids, they should be referred to consider immunomodulating or biological therapy

Controlled Trial of AZA in Management of Chronic UC - Results



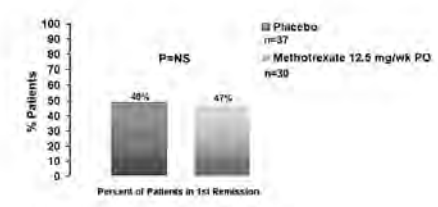
Rosenberg J, et al. *Gastroenterology*. 1975;65:96-99.

Controlled Trial of AZA in Management of Chronic UC - Results



Kirk A, et al. *British Medical Journal*. 1982;284:1291-2.

Methotrexate for Active UC and Induction of Remission



Time to first remission for MTX was 4.1 months vs. placebo (3.4 months)

Oren R, et al. *Gastroenterology*. 1996;110:1818

Complications of Surgery: Ileal Pouch-Anal Anastomosis (IPAA)

- Pelvic sepsis
- Leakage

Potential short-term complications

- Incontinence
- Intestinal obstruction
- Anastomotic strictures
- Sexual dysfunction
- Pouchitis
- Female infertility

Potential long-term complications

Lichtenstein G, *The Clinician's Guide to Inflammatory Bowel Disease*, SLACK;2003:127–129.

Complications of UC Surgery

- Mortality (<0.5%)[1]
- 3-10 stools/24 hrs so bowel pattern not normal[1]
- Impotence (1.5%)[2]
- Pouchitis (10-60%)[1]
- Small bowel obstruction (20%)[3]
- Decrease in female fertility (56-98%)[3-5]
- Pouch-vaginal fistula (4%)[1]

[1]Sagar PM, Pemberton JH. In Satsangi J, Sutherland L, et al, eds. Inflammatory Bowel Diseases. Spain: Elsevier Limited; 2003:481-511.
[2]Pemberton JH, et al. Ann. Surg. 1987;206(4):504-513.
[3]Olsen, KO, et al. Gastroenterology. 2002;122:15-19.
[4]Jamison R, et al. Dis Colon Rectum. 2004;47:1118-1126.
[5]Gorgun E, et al. Surgery. 2004;136(4):795-803.

Ileal Pouch –Functional Outcome

	Age in Years			
10 year postoperative	<45	46-55	56-65	>65
# of BM / 24 Hours	5.5	5.7	6.2	4.6
Never Incontinent (%)	56	46	42	33
Nocturnal Seepage (%)	39	48	39	60

Majority of patients had UC after diseases included Crohn's disease, indeterminate colitis, familial polyposis, and cancer

Delaney CP, et al. *Ann Surg*. 2003;238:221-228.

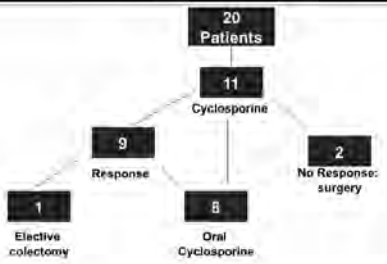
Ileal Pouch: Cumulative Incidences Pregnancy

Months	Controls (n=914)	Before Colectomy (n=84)	After IPAA (n=149)
12	75%	78%	18%*
24	82%	85%	27%*
60	88%	90%	36%*

*P<0.001 vs. Controls

Olsen KO, et al. *Gastroenterology*. 2002;122:15-19.

Cyclosporine in Patients with Severe Ulcerative Colitis



IV Cyclosporine: Major Toxicity

Renal insufficiency	23%
Infection	20%
Seizures	3%
Deaths	2%
Anaphylaxis	1%

Stenthal J et al. *Gastroenterol* 1990

ACT 1

Study Design

- Multicenter, randomized, double-blind, placebo-controlled, parallel-treatment group trial
- Conducted globally at 62 sites
- 364 subjects with moderately to severely active ulcerative colitis were randomized and treated:
  - 121 in the placebo treatment group
  - 121 in the REMICADE® (infliximab) 5 mg/kg treatment group
  - 122 in the REMICADE 10 mg/kg treatment group

Data on File, Centocor, Inc.

ACT 1

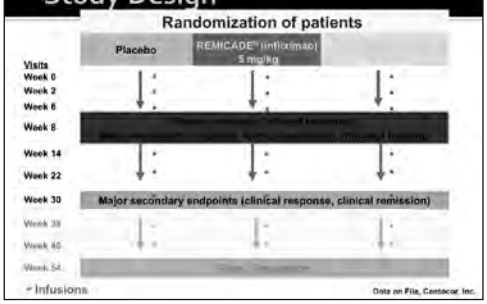
Patient Population

- Subjects with:
  - Moderately to severely active ulcerative colitis (UC):
    - Mayo score ≥ 6 points (on 12 point scale)
    - Endoscopy subscore ≥ 2 points
- Subjects must meet at least 1 of the following criteria:
  - Current treatment with ≥ 1 of the following:
    - Oral corticosteroids, 6-mercaptopurine (6-MP), or azathioprine (AZA)
  - Have failed to successfully taper, tolerate, or respond to corticosteroids within the past 18 months
  - Have failed to tolerate or respond to 6-MP or AZA within the previous 5 years

Data on File, Centocor, Inc.

ACT 1

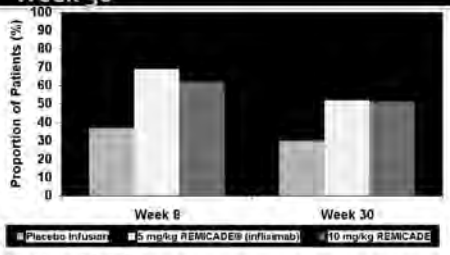
Study Design



Data on File, Centocor, Inc.

ACT 1

Clinical Response at Week 8 and Week 30

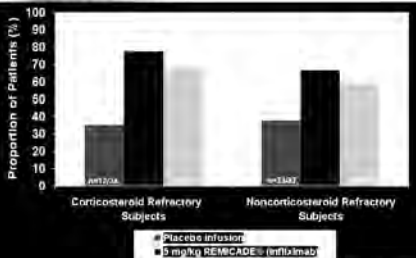


Intent-to-treat Analysis
Patients in all groups with baseline medication were continued on stable doses

REMICADE US Package Insert.

ACT 1

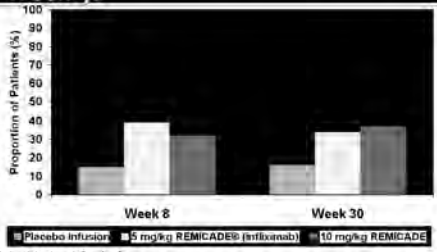
Clinical Response at Week 8 by Corticosteroid Refractory Status



Data on File, Centocor, Inc.

ACT 1

Clinical Remission at Week 8 and Week 30

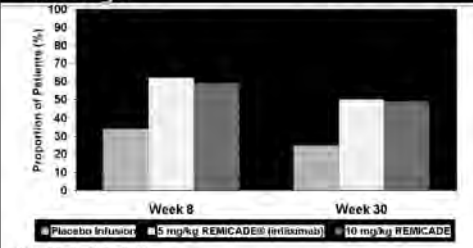


Intent-to-treat Analysis
Patients in all groups with baseline medication were continued on stable doses

REMICADE US Package Insert.

ACT 1

Mucosal Healing at Week 8 and Week 30

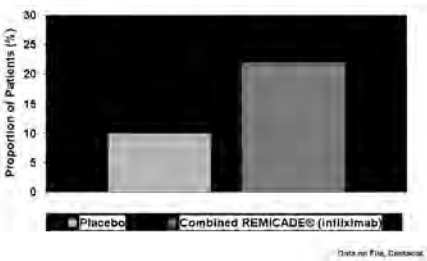


Intent-to-treat Analysis
Patients in all groups with baseline medication were continued on stable doses

REMICADE US Package Insert.

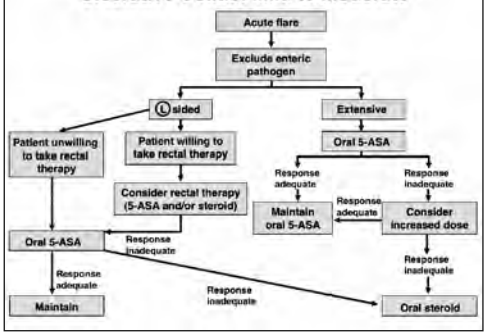
ACT 1

Clinical Remission Without Corticosteroids at Week 30

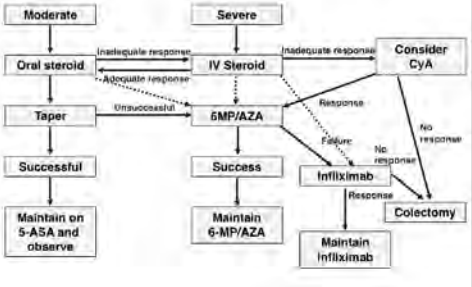


Data on File, Centocor, Inc.

Ulcerative Colitis: Mild to Moderate



Ulcerative Colitis: Moderate to Severe



Causes

- **Cigarette smokers have a 40 percent lower risk of developing ulcerative colitis than do nonsmokers; however, compared with those who have never smoked, former smokers are approximately 1.7 times more likely to develop the disease[1]**
- **No consistent link between diet and the development of ulcerative colitis has been found.**

ASCEND I & II:

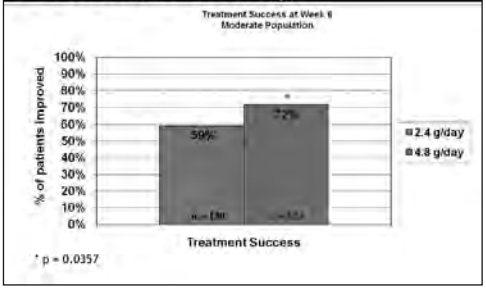
- Two Phase III, multi-center, randomized, double-blind controlled studies
- 423 patients with moderately active UC randomized to oral mesalamine (Asacol)
  - 4.8 g/day (800 mg tablets) or 2.4 g/day (400 mg tablets) x 6 weeks
- Treatment with 4.8 g/day provided a statistically significant efficacy benefit over 2.4 g/day in moderately active disease
- Both doses of mesalamine had similar safety profiles, and both were well tolerated

Hanauer et al. DDW 2005

Causes

- **The precise etiology of ulcerative colitis is not well understood.**
- **A current hypothesis suggests that primary dysregulation of the mucosal immune system leads to an excessive immunologic response to normal microflora[1]**

Treatment Success ASCEND II (Asacol study)



Peroxisome Proliferator-activated Receptors-γ (PPAR-γ)

- Recent data suggest that 5-ASA acts at least in large part through the activation of the PPAR-γ nuclear receptors.
- These receptors are expressed at high levels in colonic epithelial cells.
- PPAR-γ is involved in the control of inflammation, cell proliferation, apoptosis and metabolic functions.

CRC risk in UC

- The cumulative incidence of CRC among the patients with UC following diagnosis was
  - 0% at 5 years
  - 0.4% (95% CI: 0.0%-1.5%) at 15 years
  - 2% (95% CI: 0.0%-4.9%) at 25 years

5-ASA therapy in the prevention of colorectal cancer in ulcerative colitis

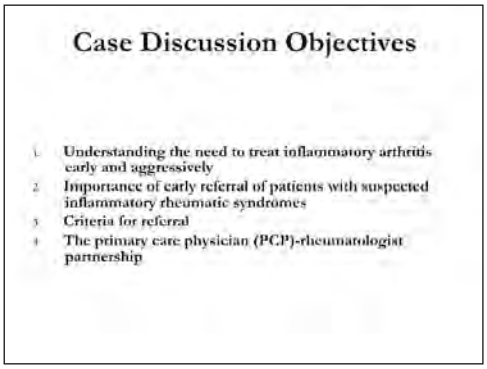
- Aminosalicylate use of 1.2 g/day or more was associated with a 72% reduction in the odds of dysplasia/CRC (odds ratio, 0.28; 95% confidence interval, 0.09-0.85).
- As the total dose of aminosalicylates increased, the odds of dysplasia/CRC decreased (P = .056).

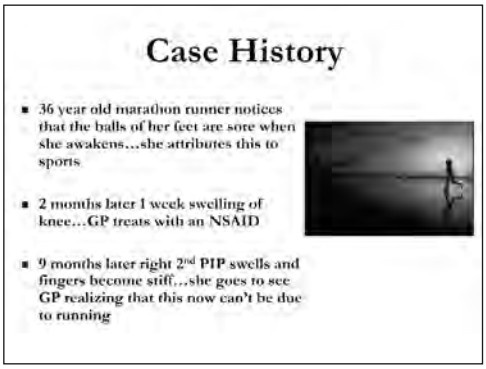
Notes

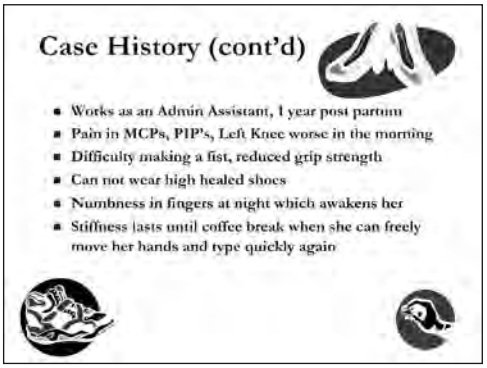
15:00-15:30 Red Flags for Early Rheumatology Referral

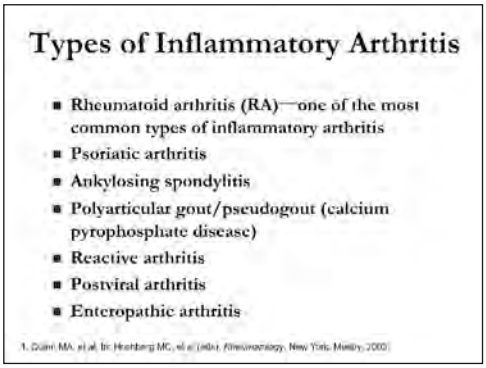
Michael R. Starr MD, FRCPC
Associate Professor, Faculty of Medicine, McGill University;
Division of Rheumatology, McGill University Health Centre

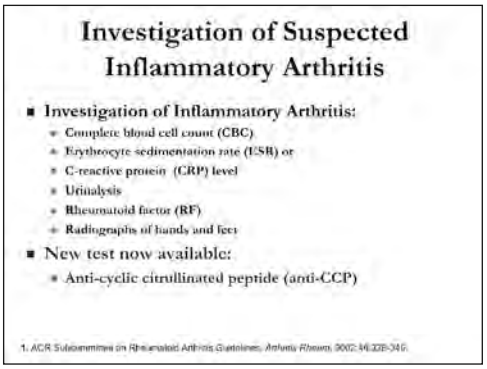
Research interests: Rheumatologist at Montreal General Hospital, was program director for 8 years and so has been involved in education and teaching at McGill for many years. Editor of "Experts on CAU' section of the Canadian journal of diagnosis. Interests in inflammatory arthritis, and has been involved in many clinical trials in the area of new therapeutics for inflammatory Rheumatic disorders.

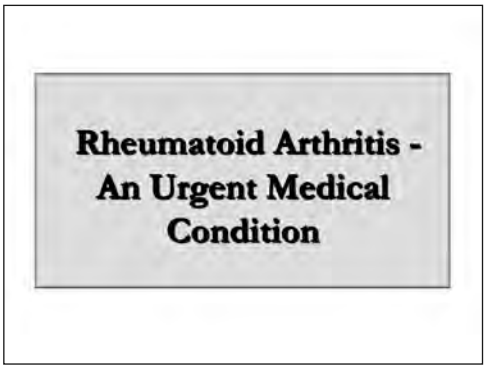




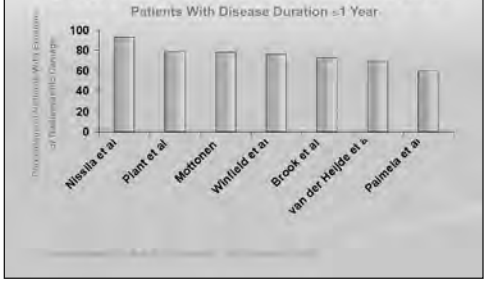




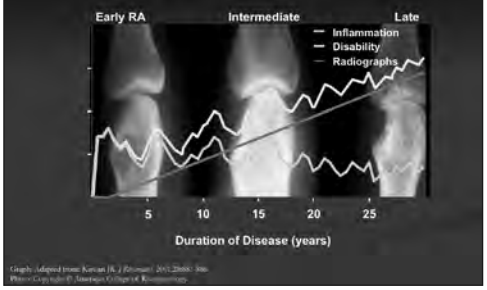




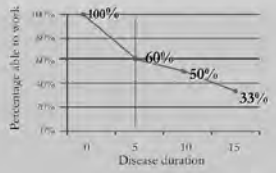
A Majority of Patients Develop Bone Erosions Within 1 Year



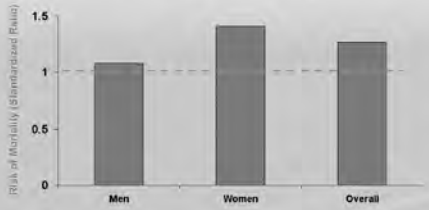
RA Linear Progression



Early Intervention In RA Is Extremely Important As Disability Is Progressive



Patients With RA Have Increased Risk of Mortality

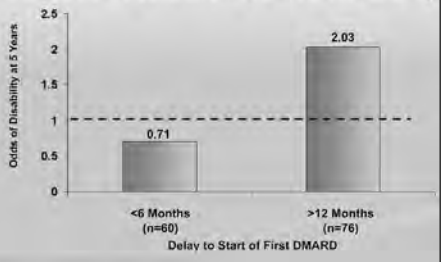


Goals of RA Treatment

- "Satisfactory treatment for RA should achieve all 3 of the following goals:
  - Relief of signs and symptoms (ie, joint swelling and tenderness)
  - Improvement in physical functioning and quality of life
  - Inhibition of the progression of joint damage"

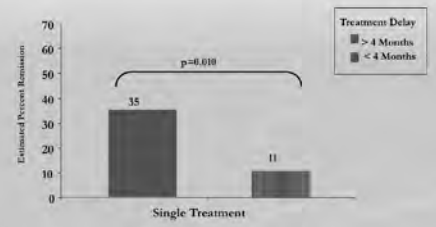
Canadian Rheumatology Association Position on the Use of Biologic Agents for the treatment of Rheumatoid Arthritis

Treatment Within the First 6 Months of Disease Onset Reduces Risk of Disability



Fin-RA Co Study

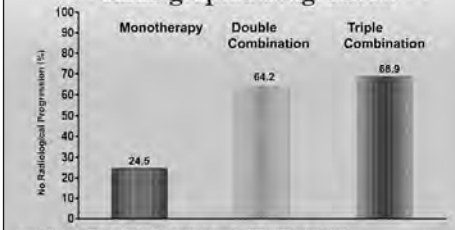
Brief Delay of Therapy Predicts Remission @ 2 Years



Early Use of Conventional DMARD Combinations Better Than Monotherapy

Calguneri et. al.

Combination Therapy in Early RA - Radiographic Progression * -



Rationale for Use of Combination DMARDs in Early RA - Improved Efficacy -

- Early use of conventional DMARDs more effective
  - brief, DMARD delay – detrimental long term
  - brief, aggressive Rx - beneficial long term
- Early use of DMARD combinations more effective than mono Rx

BUT!!!

- Many patients don't respond fully or tolerate these regimens
- Newer Biologic agents have revolutionized the treatment of Rheumatoid Arthritis

Biologic Therapy: TNF Antagonists

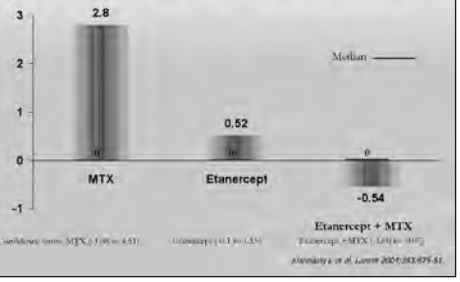
Characteristic	Infliximab	Etanercept	Adalimumab	Golimumab
Approved	Yes	Yes	Yes	Yes
Class	TNF mAb	sTNFR	TNF mAb	TNF mAb
Half-life	8-10 days[1]	4 days[2]	10-20 days[3]	14 days
Dosing	IV q8wk	SC biw	SC eow	SC monthly

mAB = monoclonal antibody; sTNFR = soluble TNF Receptor
IV=intravenous; SC=subcutaneous; biw=twice per week; and eow=every other week

1. Remicade product monograph. 2. Enbrel product monograph. 3. Humira product monograph.

Better Efficacy of TNF Antagonists in Combination Therapy With MTX by Measuring Signs and Symptoms (ACR Response) and X-ray Changes

TEMPO: Mean Change in Total Sharp Score(X-Ray progression) @ Week 52



RA Treatment: Then and Now

Before 2000:

- Emphasis on treating symptoms of the disease
- Less aggressive treatment in early stages
- NSAIDs considered least toxic: MTX and corticosteroids considered most toxic

2000 + Approach:

- Emphasis on limiting destruction of joints
- Earlier use of aggressive treatment
- MTX is considered first-choice DMARD
- Combination therapy and Biologic Therapies

Emphasis on Early Optimal Therapy

MTX = methotrexate; SSZ = sulfasalazine

Key Messages

- Disability and joint damage occur early in RA
- Early intervention limits joint damage and disability and improves quality of life and longevity of the RA patient
- There is an emergent need to treat RA early and aggressively

Importance of Prompt Referral to a Rheumatologist Upon Suspicion of RA

- Diagnosis of RA is challenging
  - Negative lab tests and x-rays do not preclude early disease
  - Accurate diagnosis is aided by rheumatology input

1. Pincus T, et al. Arthritis Rheum 1988;31:1551-1554

Removing Barriers to Rheumatologic Care

"Then"

PCPs may have been reluctant to refer patients to a rheumatologist because of the long lag time from referral to scheduled appointment.

"Now"

Rheumatologists are making substantial changes in their practices so that referred patients can be seen within a few weeks.

IF THEY ARE MADE AWARE OF THE URGENCY !
AND, EARLY RA IS CONSIDERED AN URGENT PROBLEM

1. Pincus T, et al. Arthritis Rheum 1988;31:1551-1554

REFERRAL: To Local Rheumatologist

Patient Name:
Address:
City: Postal Code:
Telephone: H) W)
Date of Birth: Health Card Number:

Reason for Referral: Suspected Inflammatory Arthritis

When did symptoms start?
How many swollen joints?
Which joints?
Other information:

Laboratory and X-ray Results: (Please attach pertinent results)

Signature of Referring Physician:
Physician Number:

Key Messages -1

Treatment should aim for clinical remission
DMARDs can alter the natural course of RA if started early **
Combination biologic plus methotrexate is more effective than either agent alone

Refer patients with early suspected inflammatory arthritis to a rheumatologist for intervention

It is never too late to treat RA aggressively!!

Key Messages - 2

- Clinical suspicion of inflammatory arthritis may be supported by the presence of any of the following:
  - ≥3 swollen joints
  - MTP/MCP involvement
  - Morning stiffness of ≥30 minutes
- The PCP-rheumatologist partnership results in optimal patient care

Case Study

- 71 y.o. male
- F.U.O. 4 months
- Work-up negative for infection or malignancy
- ESR 46, CRP 62

Case Study

- 72 y.o. woman
- Woke up one day with myalgias, stiffness shoulders then thighs over 1 month
- Fatigue,malaise
- Pain wakes her up
- a.m. stiffness profound
- ESR 65, CRP 47

Case Study

- 67 y.o. woman
- T.I.A. resolved, put on ASA
- Malaise, weight loss, low grade fever few months
- Left arm feels weak ,achy with use
- Left subclavian bruit on exam
- ESR 86

Clinical Features of PMR

- Pain in the muscles of the shoulder girdle, pelvic girdle, and neck (commonly bilateral and symmetrical, of at least 4 weeks in duration
- Stiffness after rest
- Elevation of the erythrocyte sedimentation rate (greater than or equal to 40 mm/hour)
- Frequent constitutional features including anemia, weight loss, fever, and general malaise
- Prompt clinical response to corticosteroid treatment

PMR/RA Overlap in the Elderly

- RA may present as PMR in the "elderly onset" subgroup of RA
- Difficult to tell apart initially, often seronegative
- Watch for peripheral, small joint involvement as taper steroids- this may be the clue

Treatment of PMR

- Prednisone 10-20 mg per day
- i.m. Depomedrol 120 mg q 3 weeks
  - Dasgupta, Br. J. Rheum. 1998; 37: 189-195
- Decrease 2.5 mg q 2 weeks until 10 mg/day, then decrease to 1 mg/month
- Follow the ESR and CRP, but treat the patient!
- Treat 1-2 years

Do you need to perform Temporal Artery Biopsy in PMR?

About 10% biopsy positive in PMR without symptoms of arteritis,so perform only in patients who have cranial symptoms or signs

Differential Diagnosis of Polymyalgia Rheumatica

- Arthropathies
  - Rheumatoid arthritis
  - Other inflammatory joint diseases in the elderly
  - Degenerative joint disease
  - Shoulder disorders
- Inflammatory muscle disease
- Malignant diseases
- Infection
- Hypothyroidism
- Parkinson's disease
- Functional myalgias

Treatment of PMR

- Prednisone 10-20 mg per day
- i.m. Depomedrol 120 mg q 3 weeks
  - Dasgupta, Br. J. Rheum. 1998; 37: 189-195
- Decrease 2.5 mg q 2 weeks until 10 mg/day, then decrease to 1 mg/month
- Follow the ESR and CRP, but treat the patient!
- Treat 1-2 years

GCA: Clinical Features & Epidemiology

- Mean age is 70
- 75% females
- Onset often abrupt
- Wide spectrum of symptoms
- PMR in 40 - 60%

Clinical Features of GCA

Symptoms directly related to vascular involvement:

Frequent:
- Headaches
- Abnormalities of temporal arteritis

Common:
- Ocular symptoms
- Jaw claudication

Infrequent:
- Tongue claudication
- Respiratory symptoms
- Vision loss
- Limb circulation
- Circulatory insufficiency of central nervous system
- Peripheral neuropathic syndromes
- Aortic arch syndrome

Clinical Features of GCA

Symptoms related to systemic illness

Frequent:
- Laboratory evidence for acute-phase response (elevated erythrocyte sedimentation rate, anemia, elevated C-reactive protein, elevated interleukin-6)

Common:
- Malaise, fever, anorexia, weight loss, night sweats, polymyalgia rheumatica

Infrequent:
- Arthralgias / arthritis

Treatment of GCA

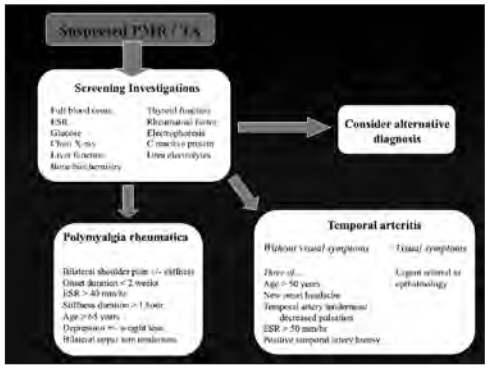
- Prednisone 40-60 mg/day
- i.v. pulse methylprednisolone for patients with visual symptoms
- Treat full dose 4-6 weeks, then reduce by 10% every 2 weeks; more slowly once 20 mg/day has been reached
- Alternate day therapy NOT recommended

Length of treatment in GCA

- Up to 50% off steroids by 2 years
  - Machado, Arthritis Rheum., 1988; 31: 745-749.
- Continue to monitor carefully for the next year post-discontinuation of therapy

Side Effects: Corticosteroids

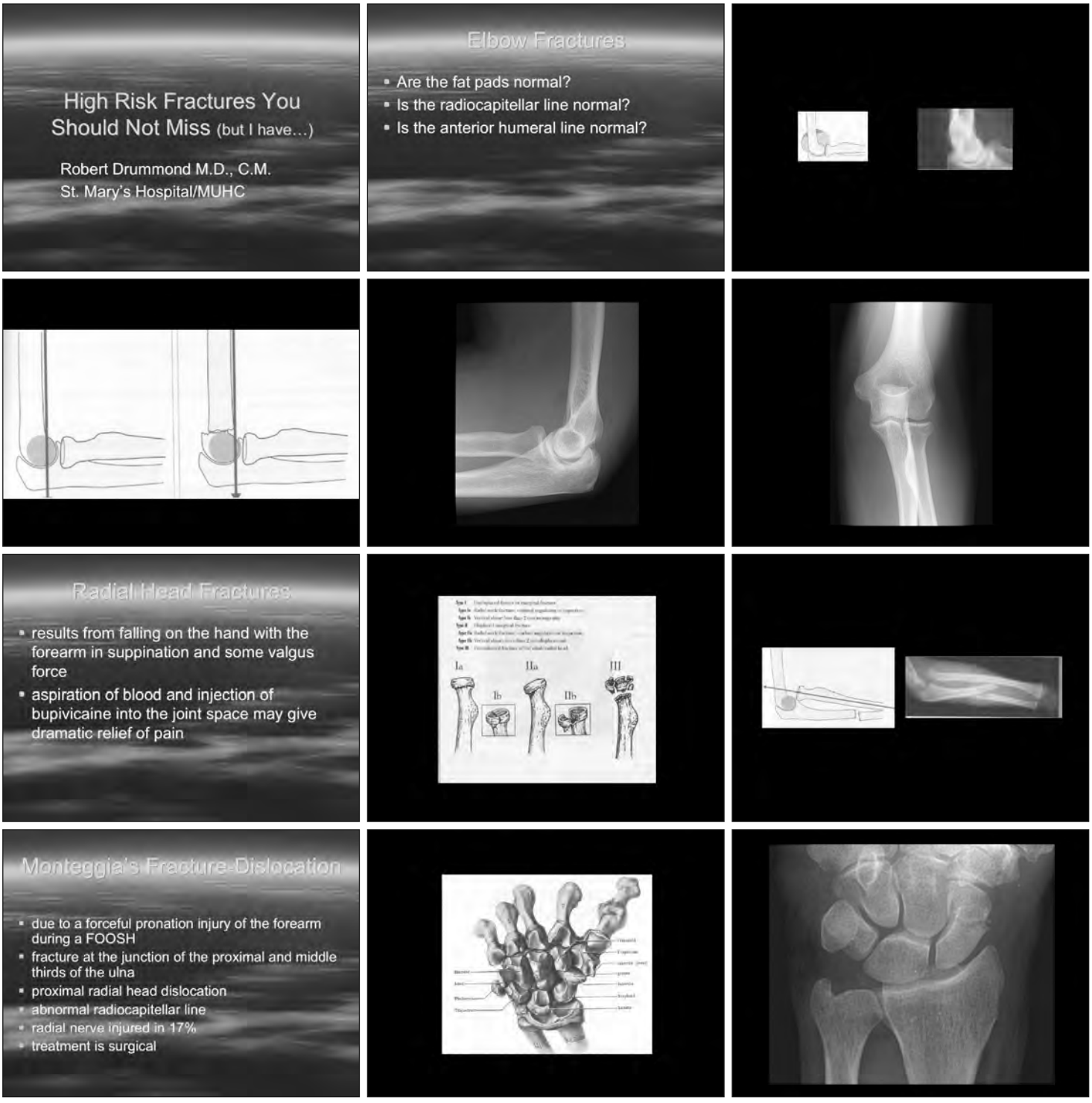
Osteoporosis:
- Osteoporosis Society of Canada (OSC) recommends Bisphosphonate therapy for all patients who take >7.5 mg/day of Prednisone for >3 months
- Calcium 1500 mg/day and Vitamin D 800-1000 u/day



16:00 - 17:00 ER: Common Fractures

Robert Drummond MD, CM

Department of Emergency Medicine, St. Mary's Hospital Centre

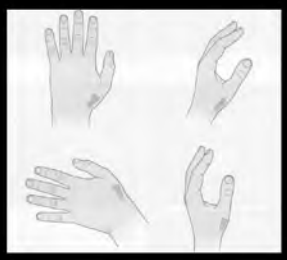


Scaphoid Fracture

- most occur with the wrist in hyperextension
- 70% occur at the waist
- risk of AVN
- 10-20% not apparent on initial radiographs

Scaphoid Fracture

- limited thumb ROM
- snuffbox tenderness sensitive but not specific
- pain may be elicited by palpation of the scaphoid tubercle, longitudinal "loading," pronation and ulnar deviation and by suppination against resistance



Treatment

- tailored to the type of fracture
- type and duration of immobilization are consultant dependant

Scaphoid Fracture – High Risk

- proximal
- oblique
- fracture gap > than 1 mm on any projection
- treat with long arm cast with the elbow flexed at 90° and the wrist in slight extension

Bohler et al. 1954

- prospective
- randomized
- more than 700 fractures
- inclusion of thumb had no effect on outcomes (time to union and incidence of nonunion)

Clay et al. *J Bone Joint Surg Br* 1991

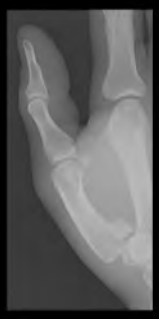
- prospective
- 392 fractures
- 6 month follow-up
- no difference in treatment outcome with treatment in a scaphoid cast or a Colle's cast

Possible Scaphoid Fracture

- CT (89% sensitivity - 91% specificity)
- MRI (95-100% sensitivity)
- radionuclide bone scanning
- splint with follow up

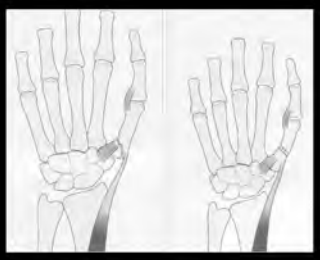
Lunate Fracture

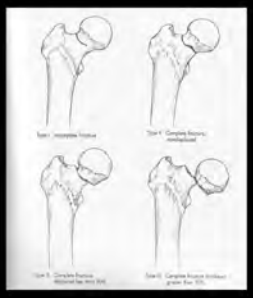
- results from a FOOSH mechanism
- pain over the dorsum of the wrist
- pain exacerbated by loading of the long-finger metacarpal
- tender on palpation distal to Lister's tubercle
- difficult to see on plain radiographs
- risk of AVN



Bennett's fracture

- intra-articular base fracture combined with dislocation or subluxation at the carpometacarpal joint
- main portion subluxes radially, although ulnar portion remains in place
- must be anatomically reduced
- thumb spica is the initial management



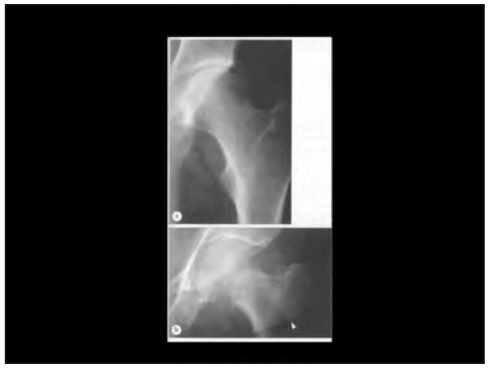


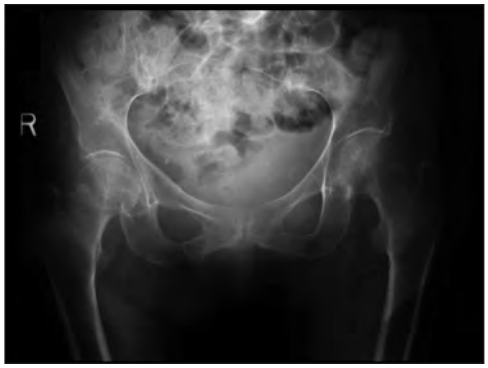
Femoral Neck Fractures

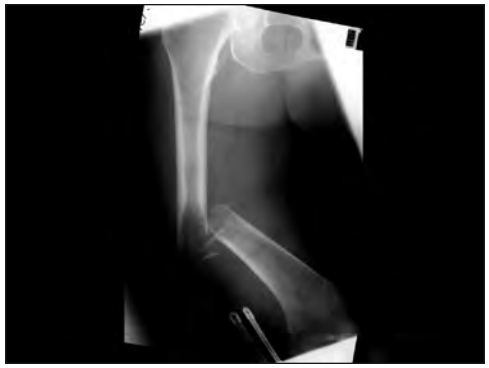
- Garden II fractures can be subtle (radiographically occult fractures in 2-9%)
- cortical disruption or impacted hyperlucency
- loss of smooth cortical transition from femoral neck to head
- trabecular disruption

Femoral Neck Fractures

- femoral neck angle (135°)
- iliopsoas shadow bulging
- Shenton's line
- consider MRI or bone scan if clinically suspicious

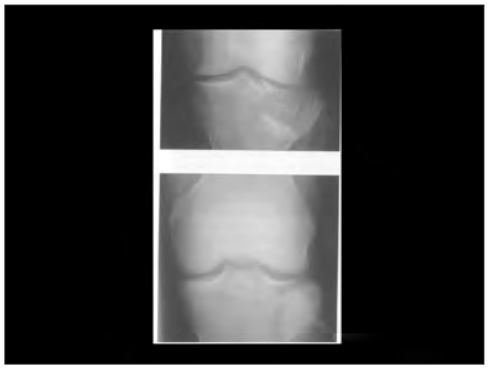


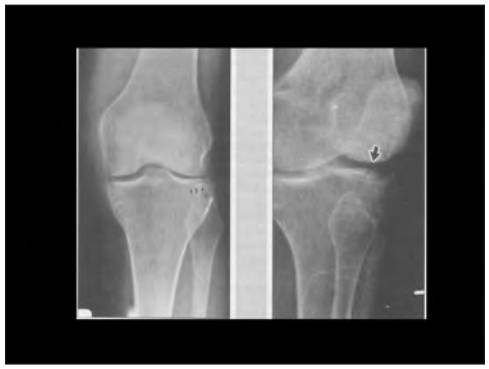


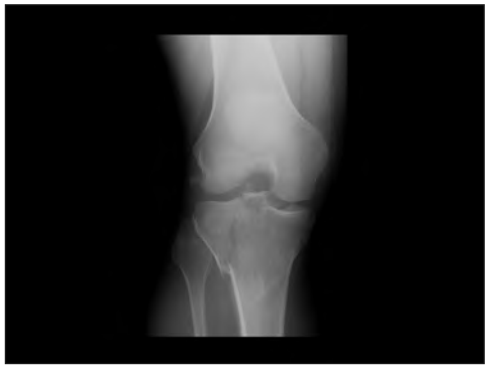


Ottawa Knee Rules

- age > 55 years old
- inability to weight bear
- tenderness of the fibular head
- isolated patellar tenderness
- inability to flex the knees to 90°

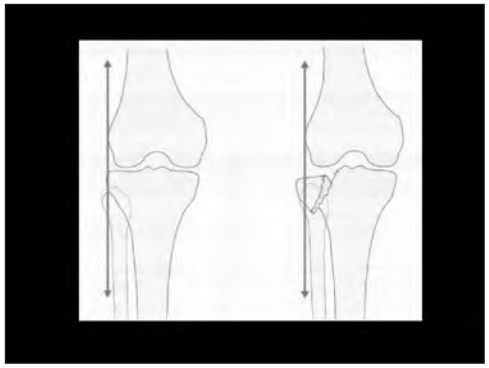


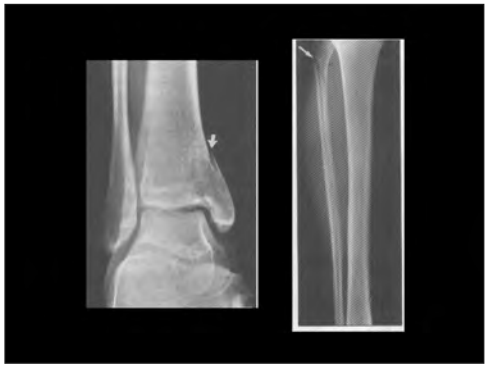




Tibial Plateau Fractures

- "bumper" or "fender" fractures
- up to 75% involve the lateral tibial condyle
- 56% have associated soft tissue injuries (mostly ACL,MCL and menisci)
- can be difficult to diagnose on plain films (79% sensitivity for 2-view/85% for 4-view)
- lateral view should be obtained as a cross table projection
- tibial plateau view is helpful



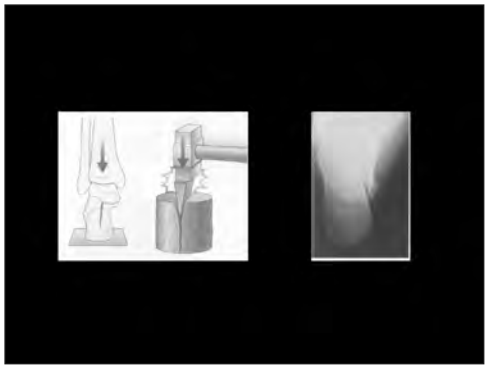


Maisonneuve's Fracture

- high oblique fibular fracture associated with ankle trauma (disruption of the interosseous membrane and talofibular ligament distally and a medial malleolar fracture or deltoid tear)
- usually results from internal rotation of the leg over a planted foot
- peroneal nerve subject to injury (foot dorsiflexion and sensation to the first web space)
- may complain only of ankle pain

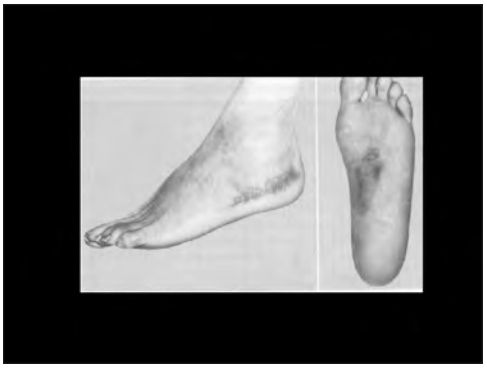
Calcaneal Fractures

- most the result of sudden, forceful, axial loading
- intra-articular fractures are more common (70-75% of os calcis fractures) and are associated with significant sequelae
- associated with other lower extremity injuries in 70% of cases and spine fractures in 10%
- open fractures are common



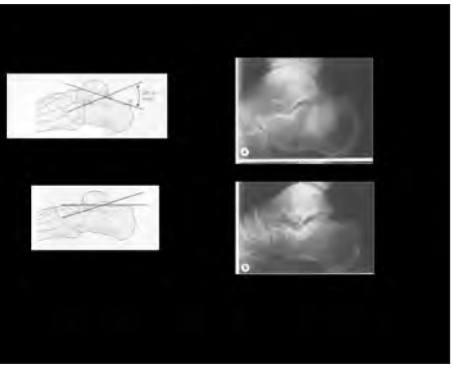
Calcaneal Fractures

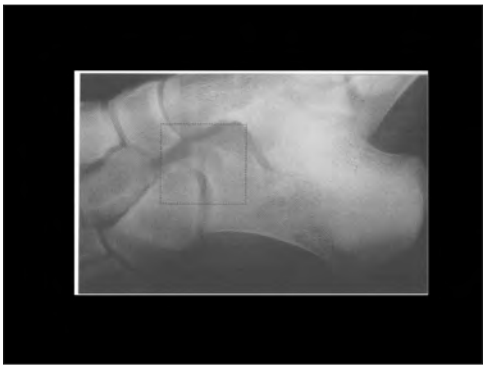
- normal contour of the heel is lost and the heel appears widened and shortened
- ecchymosis extending onto the arch of the foot is felt to be pathognomonic

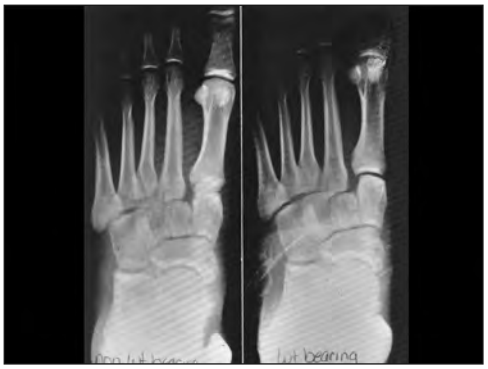


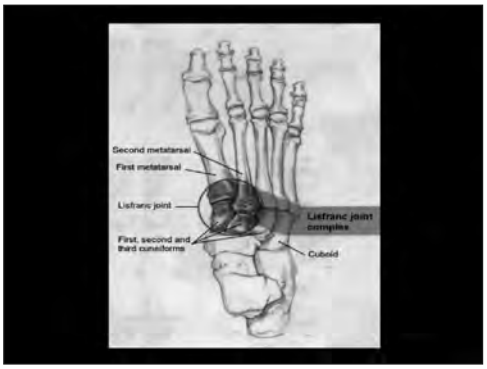
Calcaneal Fractures

- radiographic examination should include 3 views: AP, lateral and an axial calcaneal tuberosity projection which best demonstrates the tuberosity, body and sustenaculotalar joint
- a Bohler's angle < 20° implies an occult fracture







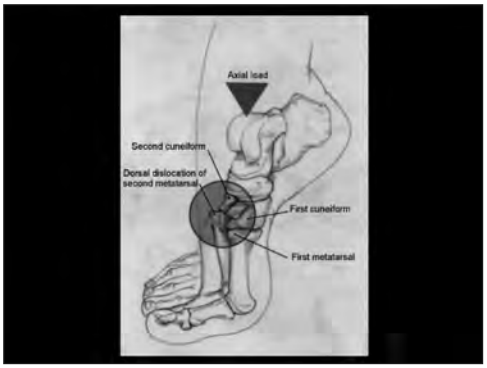


Lisfranc Injury

- injuries involving the tarsometatarsal junction
- injuries range from mild subluxations to obvious fracture/dislocations
- secondary to falls, motor vehicle or industrial accidents and athletic injuries
- high risk of chronic pain and functional disability if untreated
- 20% missed on initial presentation

Lisfranc Fracture Dislocation

- caused by either direct or indirect trauma
- indirect forces constitute the majority of injuries resulting from either rotational force applied to the forefoot with a fixed hindfoot or axial loading on a plantar flexed, fixed foot
- patients complain of pain, swelling and difficulty with weight bearing



Lisfranc Fracture Dislocation

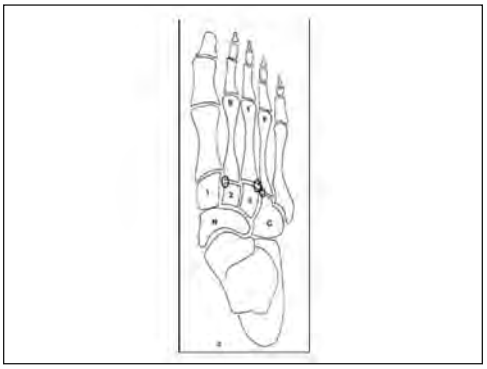
- tenderness along the Lisfranc joint is common
- plantar ecchymosis is suspicious
- patients are unable to stand on tiptoe
- serial vascular examinations are important

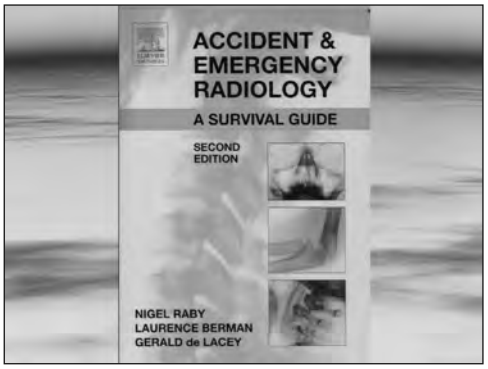
Lisfranc Fracture Dislocation

- separation between the base of the first and second metatarsal and between the medial and middle cuneiforms strongly suggests subluxation
- described as homolateral or divergent
- weight bearing films are useful

Lisfranc Fracture Dislocation

- medial border of the second metatarsal base and the middle cuneiform, the lateral border of the third metatarsal shaft and the lateral border of the lateral cuneiform, and the medial border of the fourth metatarsal base and cuboid should form straight unbroken lines
- metatarsal shaft should never be more dorsal than its respective tarsal bone
- fracture of the cuboid, cuneiforms, navicular or metatarsal shafts is suggestive





16:00 - 17:00 GER: Osteoporosis in Elderly

Suzanne Morin MD MSc FRCP FACP
Director, Internal Medicine Clinic, Montreal General Hospital
Associate Professor, Department of Medicine, McGill University

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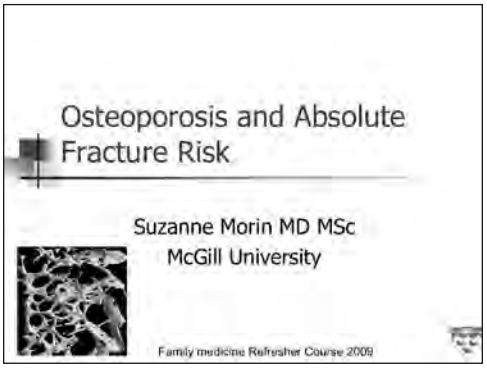
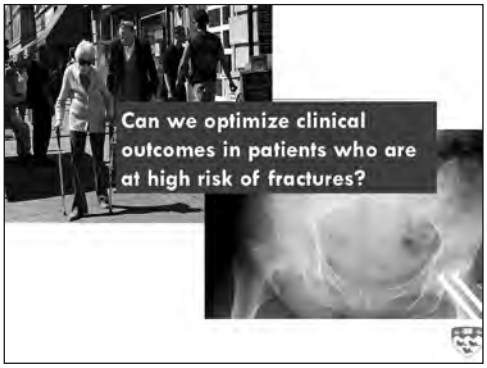
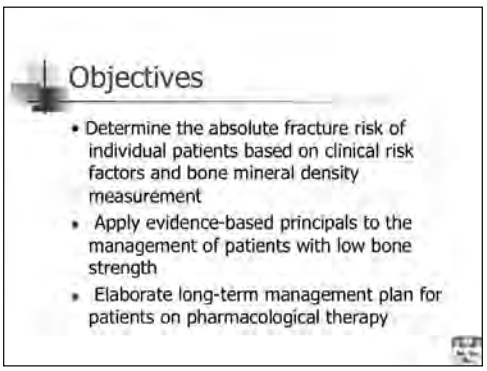
Research interests: Dr Suzanne Morin graduated from Université Laval de Québec with a Medical degree and did her Internal Medicine specialty training at McGill University where she is now Associate Professor in the Department of Medicine. In 2007, she obtained a Master's degree in epidemiology and biostatistics from McGill University. She is the Director of the Internal Medicine Clinic at the Montreal General Hospital. She is a member of the MUHC Bone Center and is in charge of the transition clinic for patients with Osteogenesis Imperfecta at the Montreal General Hospital.

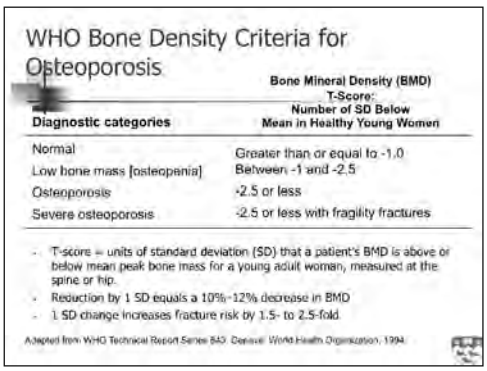
Dr. Morin's research interests include health-related outcomes for osteoporosis, particularly following hip fractures and in the population from Nunavik. She has published in peer-reviewed journals, sits on the editorial board of the Canadian Journal of General Internal Medicine.

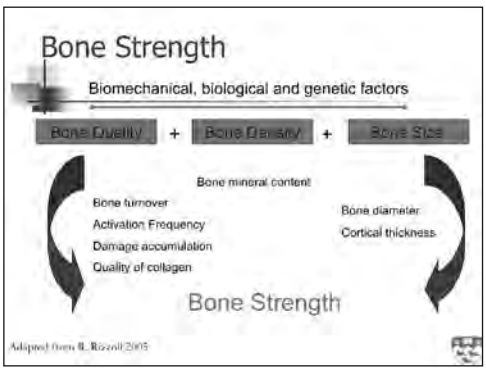
She is a member of the executive committee of the Scientific Advisory Council of Osteoporosis Canada, Chair of the Clinical Guideline committee of Osteoporosis Canada and is a member of the Research and Development committee of the McGill Bone and Periodontal Research Centre. She is also a member of numerous scientific organizations.







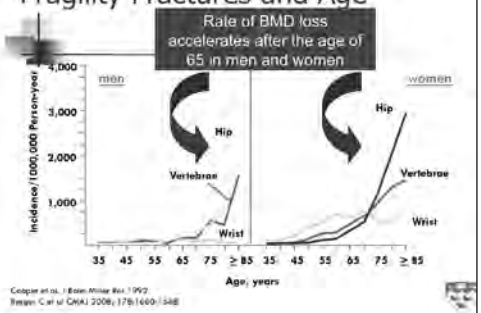




Consequences of Osteoporosis

- Fractures (hip, spine, wrist)
- Morbidity
- Loss of autonomy
- Loss of productivity
- Decreased quality of life
- Mortality
- Health care and personal costs

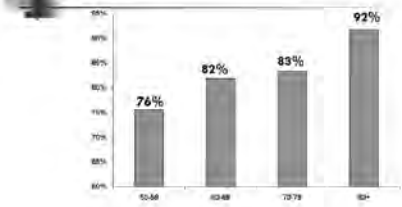
Fragility Fractures and Age



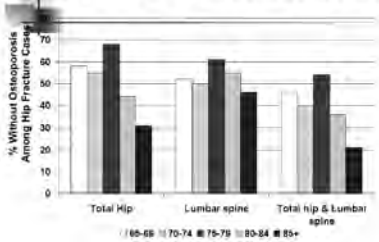
Osteoporotic Fractures ("Fragility")

- Incidence higher in women than in men
- Incidence increases with age
- Associated with low BMD
- Occur spontaneously or after a minor trauma:
  - After a fall from standing height
  - After a fall from sitting or laying from < 1m
  - After a movement outside the typical plane of motion
  - After coughing

ROCQ: Proportion of Fragility Fracture by Age Group in Women over age 50



New Hip Fractures in Patients Without BMD Defined Osteoporosis



Mrs S

68 year old woman
HBP on ramipril
Post menopausal x 2 years
Non smoker, ETOH occ
Family history of hip fracture
BMD: T-score: - 2.6 at L2-L4
-1.5 at Femoral Neck

Fracture Risk Assessment

- Some risk factors are more important than others:
  - Older age
  - Previous fractures
  - Use of corticosteroids
  - Low BMD
- In general, young individuals are at low risk for fractures
- We should target the patients at highest risk for fractures with pharmacological therapies

Fracture Prediction Models

To predict 10 year absolute risk of fractures:

- We combine:
  - The presence of specific risk factors:
    - Age
    - Prevalent fragility fracture
    - Corticosteroid use
  - With the results of the BMD

Canadian System: USING LOWEST T-SCORE TO FIND 10-YEAR FRACTURE RISK*

WOMEN			
AGE	10-YEAR RISK		
	LOW <10%	MODERATE 10 to 20%	HIGH >20%
50	>2.3	-2.2 to -3.9	<-3.9
55	>1.9	-1.9 to -3.4	<-3.4
60	>1.4	-1.4 to -3.0	<-3.0
65	>1.0	-1.0 to -2.6	<-2.6
70	>0.8	-0.8 to -2.2	<-2.2
75	>0.7	-0.7 to -2.1	<-2.1
80	>0.6	-0.6 to -2.0	<-2.0
85	>0.7	-0.7 to -2.2	<-2.2

*L1-4 (min. of 2 valid vertebrae), total hip, trochanter and femoral neck)

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75	>0.7	-0.7 to -2.1	<-2.1
80	>0.6	-0.6 to -2.0	<-2.0
85	>0.7	-0.7 to -2.2	<-2.2

*Fragility Fracture
*Use of corticosteroids in the last year

*L1-4 (min. of 2 valid vertebrae), total hip, trochanter and femoral neck)

FRAX: http://www.shef.ac.uk/

General Recommendation for Maintenance of Musculoskeletal Health for Postmenopausal Women and Older Men*

Weight bearing exercises which include impact: 30 minutes, three times a week

Adequate calcium intake: 1200-1500 mg per day†

Adequate vitamin D intake: at least 800 IU per day ‡

Smoking cessation

Moderate alcohol use ¤

Fall prevention strategies

* Osteoporosis Canada 2002 Clinical Practice Guidelines (16)
† (16;22;23)
‡ Less than 3 units of alcohol daily (1 unit is equivalent to 285 ml of beer, 120 ml of wine or 30 ml of spirits) (12)

Osteoporosis Treatment Efficacy (BMD and Fractures)

- Demonstrated in clinical trials and meta-analyses
- Confirmed in observational studies (real-world effectiveness)
- Demonstrated to be cost-effective, particularly in the elderly (CADTH, NICE)
- Multiple clinical guidelines are available (OC, NOF, NICE)

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Effect of Pharmacological Therapy in Postmenopausal Women Compared to Placebo on the Risk Fracture and Supporting Levels of Evidence*

Agent	Vertebral Fracture		Non-Vertebral Fracture		Hip Fracture	
	Risk	Level of Evidence	Risk	Level of Evidence	Risk	Level of evidence
Etidronate	Reduced	1a	No effect	1a	No effect	1a
Alendronate	Reduced	1a	Reduced	1a	Reduced	1a
Risedronate	Reduced	1a	Reduced	1a	Reduced	1a
Zoledronic	Reduced	1b	Reduced	1b	Reduced	1b
Raloxifene	Reduced	1a	No effect	1a	No effect	1a
Calcitonin	Reduced	1a	No effect	1b	No data	-
Teriparatide	Reduced	1a	Reduced	1a	No effect	1b

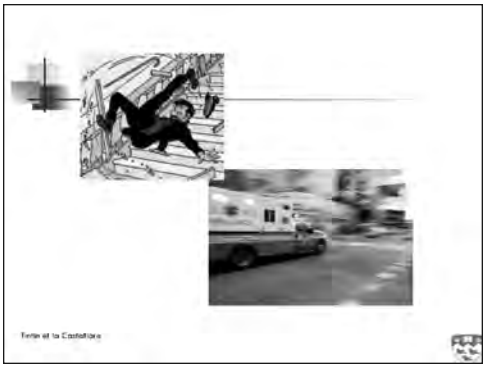
*Oxford Centre for Evidence-based Medicine Levels of Evidence (Level 1a: evidence from systematic reviews with homogeneity of randomized control trials; Level 1b: evidence from randomized clinical trials) (www.cebm.net)

Follow-up

- Adherence to recommendations
- BMD
- Bone turnover markers ?
  - Osteocalcin
  - C-telopeptides

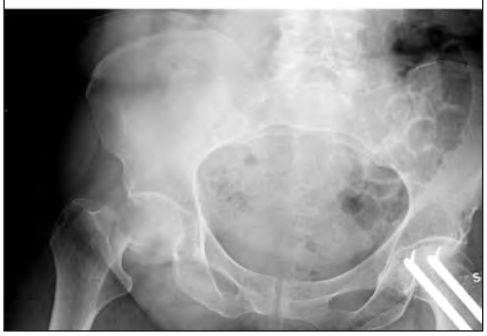
Adherence

- Compliance and Persistence
- Barriers
  - Patients' beliefs
  - Physicians'
  - Process of care



Mrs T

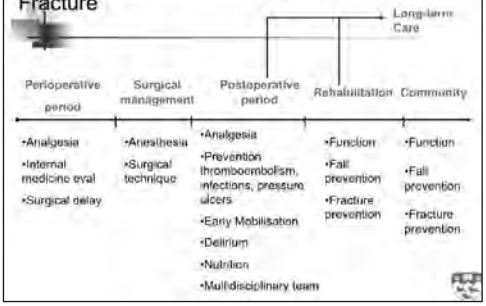
- 72 years old, lives with her husband
- Takes risedronate, calcium and vit D
- Falls in her living room in the evening
- Severe pain in her right hip
- Previous hip fracture 2 years ago
- Brought to the ER...



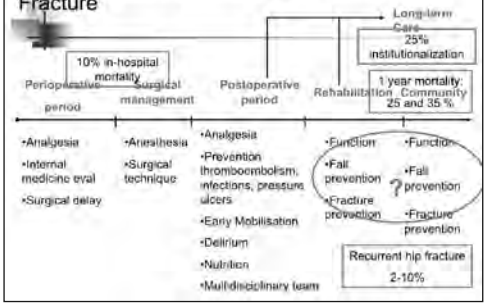
Fracture of the Hip

- Fracture of the hip is the most serious consequence of osteoporosis
- 300,000 hip fractures occur annually in the United States
- 30,000 hip fractures in Canada each year
- 90% of hip fractures are secondary to osteoporosis
- Associated with:
  - Recurrent hip fracture rate of 2 to 10 %
  - Mortality rates of 12% to 35% in the year following the fracture

Clinical Trajectory Following a Hip Fracture



Clinical Trajectory Following a Hip Fracture



Post-Fracture Care

- Ultimate Goal is to regain and maintain pre-fracture function and to reduce recurrent fracture risk through:
  - Rehabilitation program
  - Fall prevention
  - Osteoporosis management
  - Adherence to management plan

What therapy, if any, will you prescribe for Mrs T

- Switch to alendronate?
- Switch to zoledronic acid?
- Switch to teriparatide?
- Continue only calcium and vitamin D?

Falls in Canada

- Most common cause of injury in the elderly
- One in three persons over the age of 65 is likely to fall each year
- Responsible for 70% of injury-related hospital-days for the elderly
- Cause more than 90% of hip fractures
- Fall-related injuries costs have been estimated at $ 2.8 billion/ year
- Falls are directly responsible for 40% of all elderly admissions to nursing homes or long-term care facilities

Mr Smith

- 78 y o man
- Acute mid-thoracic back pain while moving furniture
- Known for COPD 2ndary to smoking
- No previous fractures
- Previous intermittent courses of oral prednisone for exacerbation

Workup should include (at a minimum)

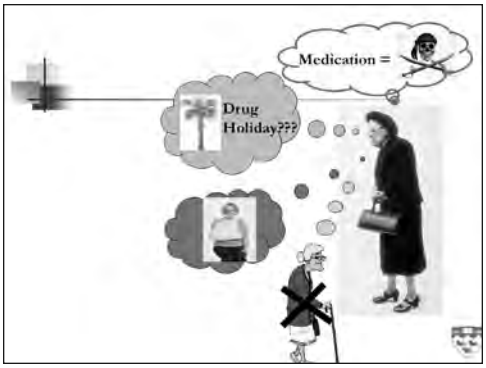
- Physical exam
  - Height measurement
  - Wall to occiput distance (>0cm, LR+ 4.6)
- Calcium, Serum protein electrophoresis
- XRay thoracic and lumbar
- Bone Scan
- Bone mineral density

Management

- Analgesia
- Calcitonin
- Bed rest?
- Back brace?
- Vertebroplasty?
- Anti-osteoporosis medication, vitamin D and calcium

Vertebroplasty

- 2 recent RCTs
  - No difference in outcomes (pain) at 30 days between sham procedure and vertebroplasty
  - Methodological limitations: cross-overs
  - Vertebroplasty is not without risks



Mrs Smith

- 72 years old
- Humerus fracture 7 years ago
- On calcium (500mg/d), vitamin D (800 iu/d) and alendronate since then.
- Walks with a friend twice a week
- Heard about serious long term side effects- wants to stop her medication

Long-term Safety of Bisphosphonates

- Osteonecrosis of the Jaw
- Atrial fibrillation
- Renal toxicity
- Atypical fractures

Drug holiday-why?

- To avoid long-term adverse events
- Data to support that 5 year of therapy might be enough
- To reduce cost?

Osteoporosis Canada

1. The reported potential link between atypical fractures and bisphosphonates.
   - Information on atypical fractures and bisphosphonates (PDF)
2. The reported potential link between Fosamax and atrial fibrillation.
   - Information on Fosamax and atrial fibrillation (PDF)
3. The reported potential link between oral bisphosphonates and aseptic osteonecrosis.
   - Information for people living with osteoporosis (PDF)
   - Information for health care professionals (PDF)
4. The reported potential link between calcium supplement intake and vascular events.
   - Information for people living with osteoporosis (PDF)
   - Information for health care professionals (PDF)
5. Osteoporosis Canada statement on esophageal cancer and bisphosphonate use
   - We are aware of a recent publication that reports on the association between oral bisphosphonate use and esophageal cancer

Incidence of Fracture by Treatment Group (FLEX)

Table 3. Incidence of Fracture by Treatment Group

Fractures	Placebo, No. (%) (n = 437)	Pooled Alendronate, No. (%) (n = 662)	Relative Risk (95% Confidence Interval)*
Vertebral			
Clinical	23 (5.3)	16 (2.4)	0.45 (0.24-0.85)
Morphometric	48 (11.0)	60 (9.0)	0.83 (0.60-1.22)
Clinical			
Any	83 (19.0)	132 (19.9)	0.93 (0.71-1.21)
Nonspine	33 (7.6)	68 (10.3)	1.33 (0.87-2.03)
Hip	13 (3.0)	20 (3.0)	1.02 (0.51-2.10)
Forearm	19 (4.3)	31 (4.7)	1.09 (0.62-1.96)

*Adjusted for clinic and ethnicity.

Black, D. M. et al. JAMA 2009;301:1827-1835.

Take Home Messages

- Fragility fracture = Think osteoporosis AND fall management
- Fragility fracture = Think long-term interventions
- Osteoporosis = Think Persistence and Adherence to management plans = Multi-targeted interventions =communication strategies
- I rarely give drug holidays in the elderly

Further reading

Prevention of osteoporosis-related fractures among postmenopausal women and older men

Poupak Rahmani MD PhD, Suzanne Morin MD MSc

www.cmaj.ca on October 19, 2009

Tuesday, Nov. 24 – Workshop F-03

16:00 - 17:00 PEDS: ADHD in Children

Lily Hechtman, MD
Professor, Psychiatry and Pediatrics, McGill University
Director, ADHD Research, Division of Child Psychiatry,
The Montreal Children's Hospital- MUHC

Research Interests: Dr. Lily Hechtman is a Professor of Psychiatry and Pediatrics and Director of Research, Division of Child Psychiatry, McGill University. She has been involved in research with children, adolescents and adults with Attention Deficit Hyperactivity Disorder (ADHD) for many years. Adult ADHD studies have included long-term prospective follow-up studies as well as diagnostic and treatment studies. This work has been written up both in scientific journals and in a book entitled: "Hyperactive Children Grown Up", published by Guilford Press. More recently, her interests in adult ADHD has extended to a recently published book entitled: "ADHD in Adulthood: A Guide to Current Theory, Diagnosis, and Treatment" published by John Hopkins Press.

In the area of child psychiatry, Dr. Hechtman has been involved in a number of multisite, multimodal treatment studies sponsored by the U.S. National Institute of Mental Health.

Dr. Hechtman has been honoured by the American Academy of Child and Adolescent Psychiatry with an ADHD Research Award, as well as an award from the U.S. Children and Adults with ADHD organization (CHADD) for "outstanding professional achievement in medicine, education and research on Attention Disorders.

Learning Objectives

- To be aware of possible etiological factors which may affect the development of ADHD
- To be aware of how to assess and diagnose children with ADHD with a focus on differential diagnosis
- To be aware of different medications used in the treatment of ADHD, their relative advantages and limitations

Suggested Reading Materials

Weiss, M., Trokenberg Hechtman, L.,Weiss, G. (1999). ADHD in Adulthood: A Guide to Current Theory, Diagnosis, and Treatment. Baltimore and London: The Johns Hopkins University Press.

Weiss, M. & Weiss, G. (2002). Attention Deficit Hyperactivity Disorder. In M. Lewis (Ed.), Child and Adolescent Psychiatry 3rd Edition, (p. 645-669). Philadelphia: Lippincott Williams & Wilkins.

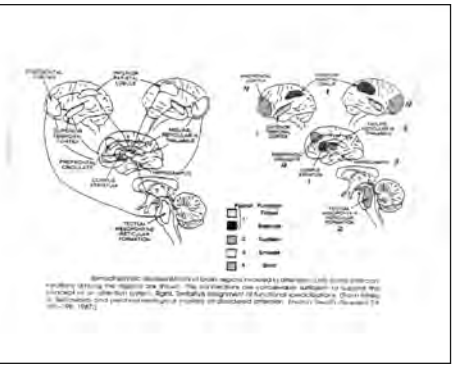
Website: http://www.caddra.ca

Diagnosis and Treatment of ADHD in Children

Lily Hechtman, MD, FRCP
Professor Psychiatry and Pediatrics
Director of Research
Division of Child Psychiatry
McGill University

Disclosure

	Research Grants	Speaker	Advisory Board	Stocks
NIMH	✓	✓		
Ely Lilly	✓	✓	✓	
Glaxo Smith/Kline	✓			
Janssen-Ortho	✓	✓	✓	
Purdue Pharma	✓		✓	
Shire	✓	✓	✓	



ADHD – DSM-IV Definition

Attention Deficit Hyperactivity Disorder (ADHD) is a neurobiological condition characterized by developmentally inappropriate levels of:

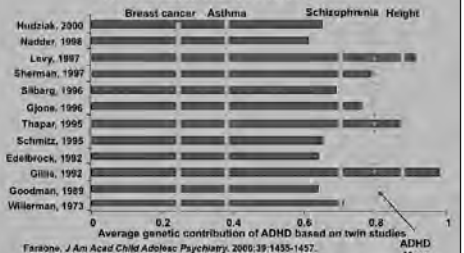
- Inattention (concentration, distractibility)
- Hyperactivity
- Impulsivity

in various combinations across school, work, home, and social settings.

Adapted from American Psychiatric Association, DSM-IV TR 2000

ADHD is Most Likely Caused by a Complex Interplay of Factors:

Twin Studies Show ADHD Is a Genetic Disorder



Faraone, *J Am Acad Child Adolesc Psychiatry*, 2000;39:1455-1457.
Hemminki, *Mutat Res*. 2001;25:11-21
Palmer, *Eur Resp J*. 2001;17:696-702.

Candidate Genes

- Candidate genes include:
  - Dopamine D4 receptor; chromosome 11(DRD4)[1]
  - Dopamine transporter; chromosome 5 (*DAT-1*)[1]
  - Monoamine oxidase A (*MAOA*)[2]
  - Vesicular monoamine transporters 1 and 2 (*VMAT-1, VMAT-2*)[3,4]
  - Synaptosomal-associated protein 25 (*SNAP-25*)[5,6]
  - Human thyroid receptor-β gene[7]
- ***No Specific X linked gene identified***

Neuroimaging Studies

- Smaller brains
- Smaller cortical areas, e.g., prefrontal cortex, cerebellum
- Smaller subcortical areas, e.g., corpus collosum, thalamus
- Hypoperfusion in these areas corrected by stimulant medication

DSM-IV Symptoms of Inattention in ADHD

- Presentation of the following symptom must occur often*
  Inattention
  - Careless
  - Difficult sustaining attention in activity
  - Doesn't listen
  - No follow-through
  - Avoids/dislikes task requiring sustained mental effort
  - Can't organize
  - Loses important items
  - Easily distractible
  - Forgetful in daily activities

* Must have 6 or more symptoms of inattention for at least 6 months to a degree that is maladaptive and inconsistent with developmental level.

Adapted from American Psychiatric Association, DSM-IV TR, 2000

DSM-IV Symptoms of Hyperactivity-Impulsivity in ADHD

- Presentation of the following symptom must occur often*
  Hyperactivity
  - Squirmy and fidgets
  - Can't stay seated
  - Runs/climbs excessively
  - Can't play/work quietly
  - "On the go"/"driven by a motor"
  - Talks excessively

  Impulsivity
  - Blurts out answers
  - Can't wait turn
  - Intrudes/interrupts others

* Must have 6 or more symptoms of hyperactivity-impulsivity for at least 6 months to a degree that is maladaptive and inconsistent with developmental level.

Adapted from American Psychiatric Association, DSM-IV TR, 2000

Symptoms

- Pervasive – many settings
- Persistent – duration 6 months
  - Onset before age 7
- Severe
  - Maladaptive, affect functioning
  - Inconsistent with developmental level
  - Clinical significant impairment, social, academic, occupational

Differential Diagnosis in ADHD: Psychiatric and Medical

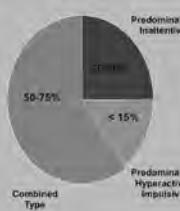
- **Psychiatric disorders can mimic ADHD**
  - Anxiety disorders
  - Mood disorders
  - Adjustment disorders
  - Learning and language deficits
  - Psychotic disorders
  - Stress
- **Medical disorders that can mimic ADHD**
  - Developmental disorders
  - Use of other medications
  - Substance use disorder
  - Seizure disorder (petit mal)
  - Sleep apnea
  - Hearing & Vision problems
  - Thyroid disorder
  - Hypoglycemia

ADHD Clinical Subtypes

Predominantly inattentive:
- Easily distracted
- Not excessively hyperactive or impulsive in behavior

Predominantly hyperactive-impulsive:
- Extremely hyperactive and impulsive
- Not highly inattentive (may have no inattentive signs)
- Often younger children

Combined type:
- Most patients
- All three classical signs of the disorder

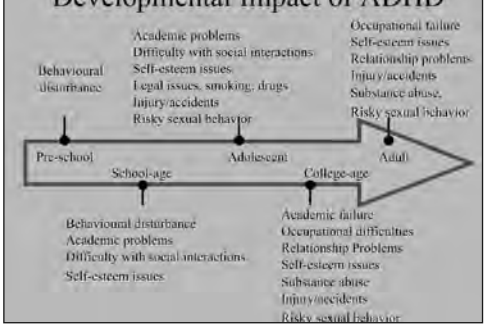


Adapted from American Psychiatric Association, DSM-IV TR, 2000

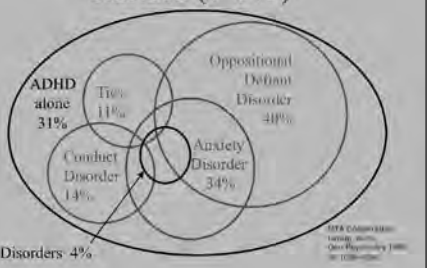
ADHD Etiology and Impact Summary

- ADHD
  - affects millions of people of both genders
  - 5-9% school age; 4% adults
  - persists through adolescence and adulthood in a high percentage of cases
  - can have negative impact on multiple areas of functioning

Developmental Impact of ADHD



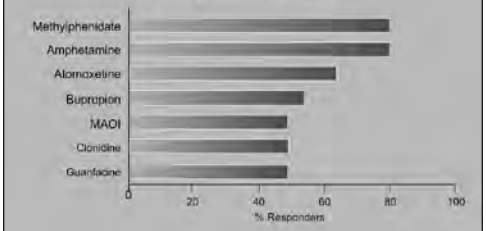
Co-occurring Disorders in Children (*n*=579)



ADHD Pharmacotherapy: Treatment Options

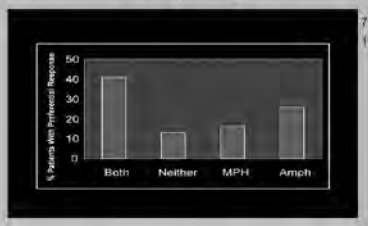
- Stimulants
  - Methylphenidate (MPH)
  - Amphetamine compounds (AMPH) (FDA-approvable)
  - Dextroamphetamine
- Atomoxetine (FDA-approved)
- Antidepressants (second-line)
  - Bupropion
  - Tricyclics
- Research
  - Modafinil
  - Cholinergic agents

ADHD Pharmacotherapy – Responsiveness

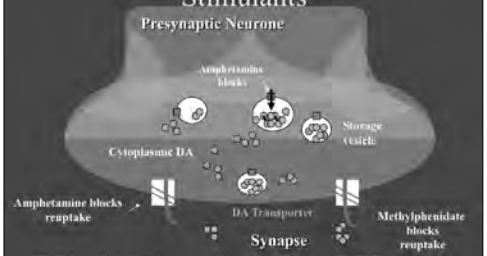


Wilens, T., Spencer, T., *Postlevel at MGH Child & Adolescent Psychopharmacology Meeting*, Boston 2000
Wilens, T., *CNS News*, 2003

Meta Analysis of Controlled Crossover Comparing Stimulants[1]



Mechanism of Action of Stimulants



CADDRA (2008) Recommendations For Dosing Psychostimulants *

Starting Dose

Starting Dose	MPH	Dexedrine	Concerta	Adderall XR	Biphentin
Child	5 mg a.m. and noon	2.5 mg a.m. and noon	18 mg a.m.	10 mg a.m.	10 mg a.m.
Adoles.	5 mg a.m. and noon	2.5 mg a.m. and noon	18 mg a.m.	10 mg a.m.	10 mg a.m.
Adult	5 mg a.m. and noon	5 mg a.m. and noon	18 mg a.m.	10 mg a.m.	10 mg a.m.

CADDRA (2008) Recommendations For Dosing Psychostimulants *

Maximum Dose

Maximum Dose	MPH	Dexedrine	Concerta	Adderall XR	Biphentin
Child	60 mg/day	30 mg/day	72 mg a.m.	30 mg a.m.	60 mg/day
Adoles.	60 mg/day	30 mg/day	108 mg a.m.	60 mg a.m.	60 mg/day
Adult	100 mg/day	60 mg/day	108 mg a.m.	60 mg a.m.	60 mg/day

Comparison of New Long-Acting Stimulants

Drug	Release Mechanism	%Dose IR	Duration	Dosages
•Biphentin	Beaded * Bi-Modal	40%	10-12 hrs.	7
•Adderall XR	Beaded* B.I.D.	50%	10-12 hrs.	6
Concerta	Water absorption, push mechanism, T.I.D.	22%	10-12 hrs.	4

*Adult indication *Can be sprinkled

ADHD: Stimulant Adverse Effects

DEX, MPH, PEM: Similar Side Effect Profiles

- Decreased Appetite
- Insomnia
- Upset Stomach
- Headache
- Irritability

Ref: Klein 1992; Wilens 1992; Pelham 1990

ADHD: Areas of Concern and Controversy with Stimulant Use

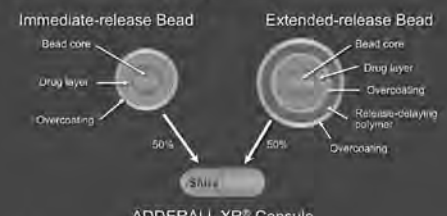
- Growth Suppression
- Development of tics
- Medication abuse
- Medicolegal concerns
- Use in adolescents
- Rebound
- Cognitive toxicity
- Cardiovascular effects

Ref: Fox 1993; Sylvester 1993; Klein 1992; Evans 1991 Swanson 1991; Johnston 1988.

CONCERTA®: OROS Technology to Clinical Efficacy

Biphentin® (CR Methylphenidate MLR™)

Adderall XR® Pulse Delivery System



ADDERALL XR® Capsule

Available in 5 mg, 10 mg, 15 mg, 20 mg, 25 mg, and 30 mg capsules

Indications for Atomoxetine

- Patients not responsive to stimulants
- Patients with significant side effects to stimulants (e.g., rebound, tics)
- Patients with Tourette's Syndrome or chronic motor tic disorders
- Epilepsy
- Comorbid Anxiety
- Abuse or diversion is a concern

Problems

- 6-8 week titration
  - 0.5mg/kg 2 weeks
  - 0.8mg/kg 2 weeks
  - 1.2mg/kg 2 weeks
- Metabolized via cytrochrome P450-206 system (10% slow metabolizers)
- Drug/drug interaction, e.g., paraxetine, fluoxetine, quinidine

Notes

Tuesday, Nov. 24 – Workshop F-04

16:00 - 17:00 HAND ON: Joint Injections

Michael R. Starr MD, FRCPC
Associate Professor, Faculty of Medicine, McGill University;
Division of Rheumatology, McGill University Health Centre

This workshop is designed as a hands on demonstration of anatomical landmark and techniques used for joint and soft tissue injections. The following notes highlight a few key principles.

Materials Needed for Aspirating and Injecting Joints

- Clean, "No Touch" technique used
- Gloves (not necessary, but may be safer in regions where human immunodeficiency virus or hepatitis cases are common)
- Providone-iodine and/or alcohol swabs
- 1%-2% lidocaine without epinephrine, or topical ethyl chloride
- 22- to 27-gauge needle for anesthetic
- 18- to 20-gauge needle for aspirating large- or moderate-sized joints
- 22- to 25-gauge needle for aspirating smaller joints
- 3 ml-5 ml syringe: anesthetic-steroid combination
- 10 ml-60 ml syringe for fluid aspiration
- Forceps; Kelleys (to allow changing syringes, etc.)
- Specimen tubes, culture container

Contraindications to Intra-Articular Corticosteroid Injections

- Periarticular sepsis
- Bacteremia
- Unstable joints
- Essentially inaccessible joints, e.g., spinal
- Intra-articular fracture
- Septic joint
- Marked juxtra-articular osteoporosis
- Failure to respond to prior injections
- Blood clotting disorders
- Probably total joint arthroplasty

Potential Sequelae from Intra-Articular and Soft Tissue Corticosteroid Injections

- Radiologic deterioration of joints – "steroid arthropathy"; Charcot-like arthropathy; osteonecrosis
- Latrogenic infection – very low incidence
- Rupture of tendon
- Tissue atrophy and fat necrosis
- Nerve damage, e.g., inadvertent injection of median nerve in carpal tunnel syndrome
- "Postinjection flare"
- Pancreatitis
- Cushing's syndrome
- Increased glucose

Typical Doses of Corticosteroid used for Injection (Usually Mixed with Lidocaine)

Structure	Dose (mg)*	Volume of Injection (ml)
Large joint		
Knee	40-60	1-4
Shoulder	40	1-4
Elbow	20-30	1-4
Medium joint		
Ankle	20-40	0.5-1
Wrist	20	0.5-1
Small joint		
Interphalangeal	5-10	0.25-0.5
Metacarpophalangeal	5-10	0.25-0.5
Metarasophalangeal	5-10	0.25-0.5
Small soft-tissue structure		
Bursa	20-40	0.5-1.5
Tendon sheath	5-20	0.25-1

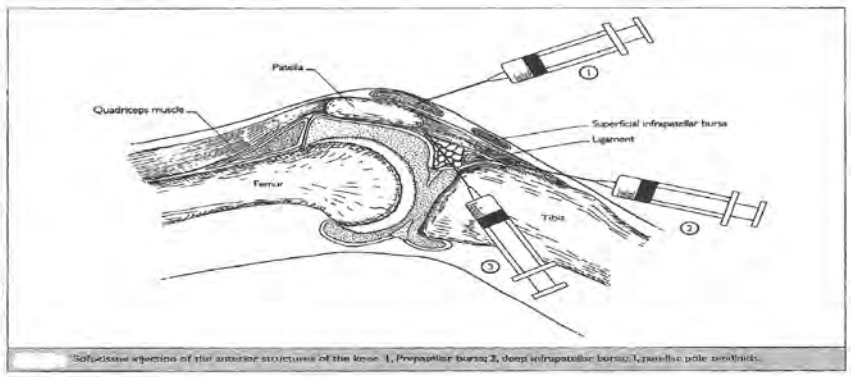
*Doses shown are for triamcinolone hexacetonide and methylprednisoione acetate

Characteristics of Synovial Fluid in Normal and Various Abnormal Conditions

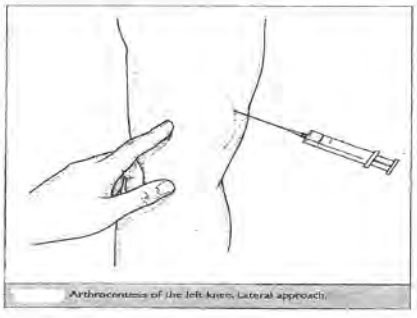
Characteristic	Condition Normal	Noninflammatory	Inflammatory	Septic
Color	Clear	Straw yellow	Yellow	Variable
Clarity	Transparent	Transparent	Hazy opaque	Opaque
Viscosity	High	High	Low	Low-high
White blood cell Count (per mm3)	0-200	200-2,000	2,000-75,000	>50,000
Neutrophils (%)	Low	Low	Medium-high	High

Reference: Genovese, M., Joint and Soft Tissue Injection, Postgraduate Medicine, Vol. 103, No. 2, February 1998

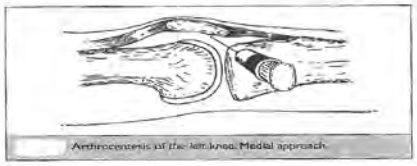
Knee Injection



Soft-tissue injection of the anterior structures of the knee. 1, Prepatellar bursa; 2, deep infrapatellar bursa; 3, patellar pole tendinitis.

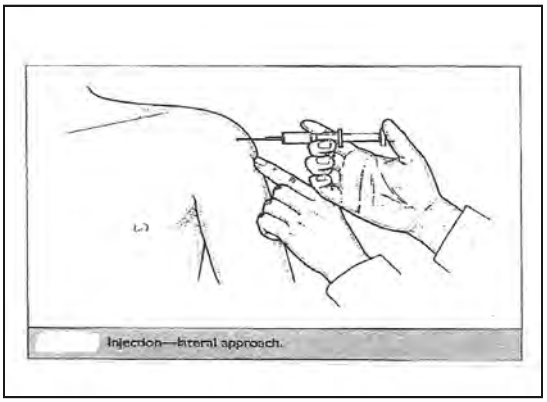


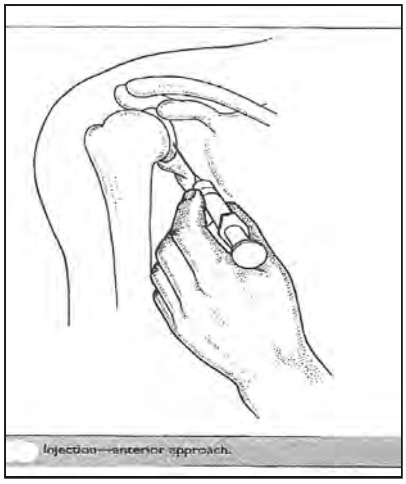
Arthrocentesis of the left knee, Lateral approach.

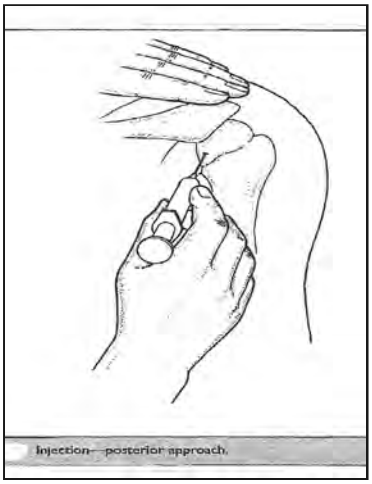


Arthrocentesis of the left knee, Medial approach.

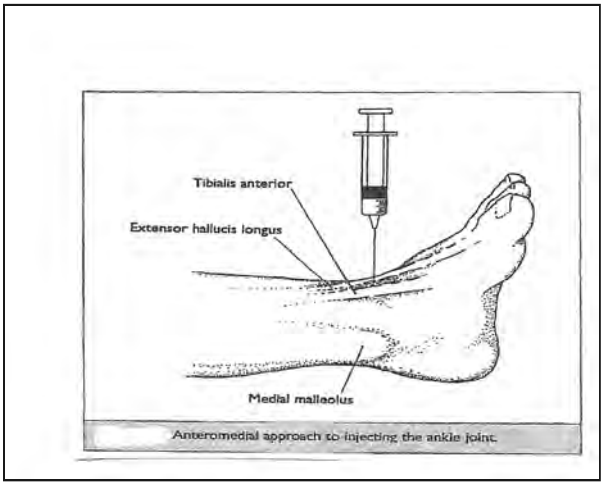
Shoulder Injection

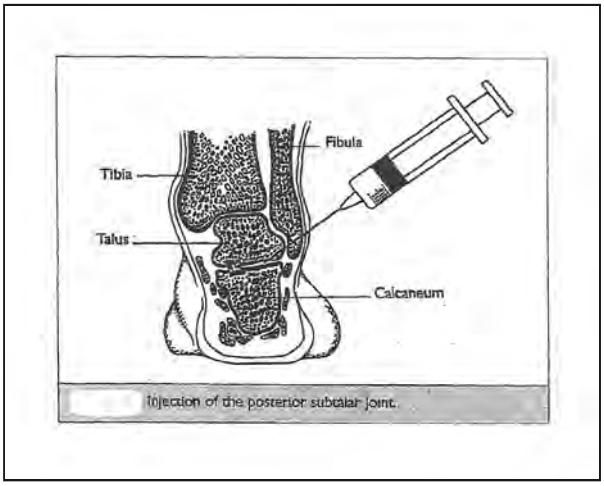


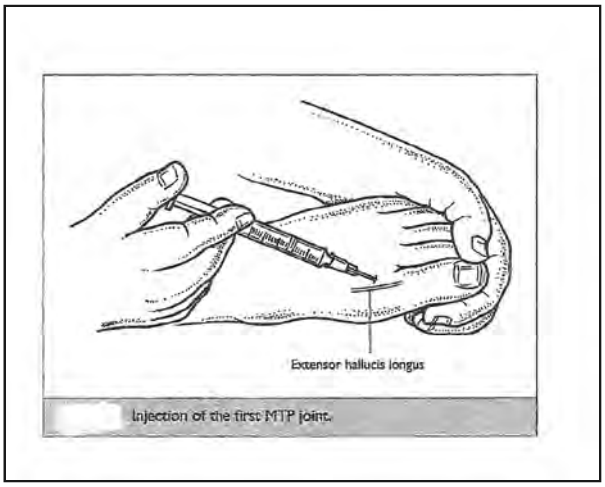




Ankle Injection





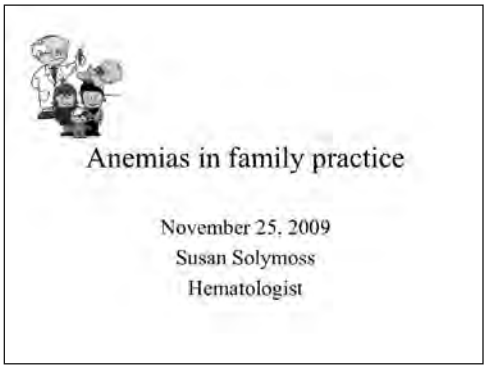


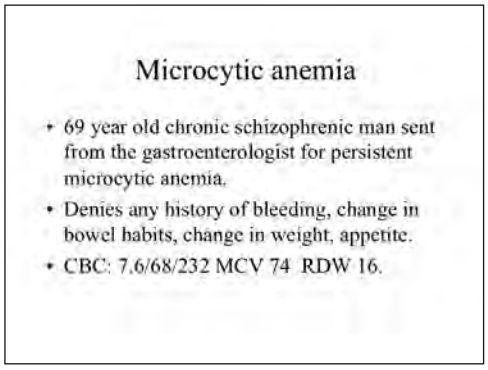
Reference: Doherty, M. Rheumatology Examination and Injection Techniques, second edition. W.B. Saunders, 1999.

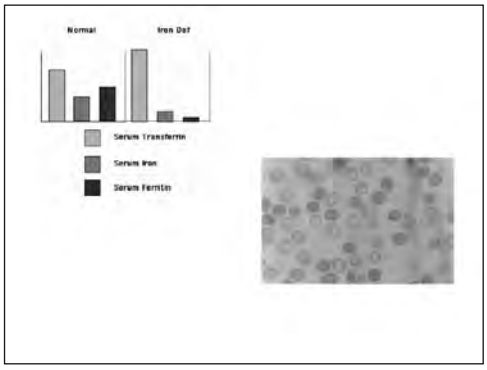
16:00 - 17:00 Anemia, Cases for Family Physician

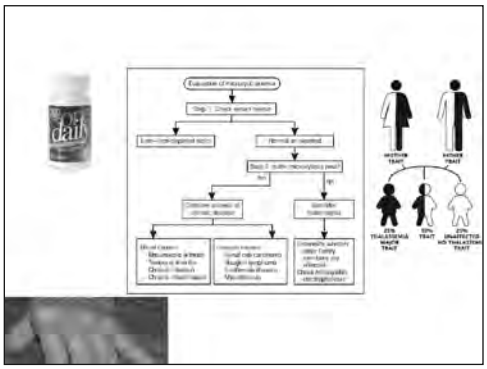
Susan Solymoss MD

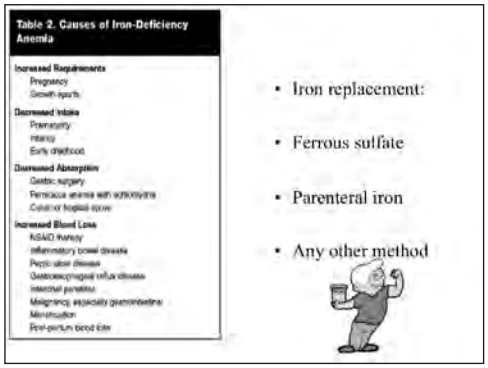
Assistant Professor, Faculty of Medicine, McGill University

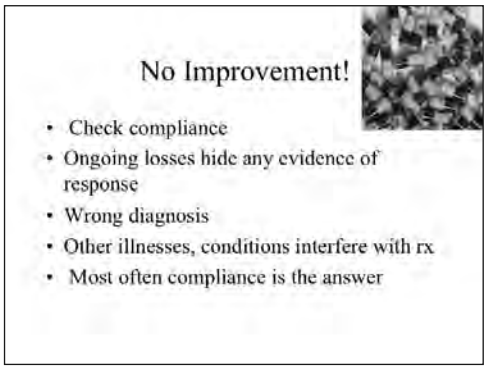


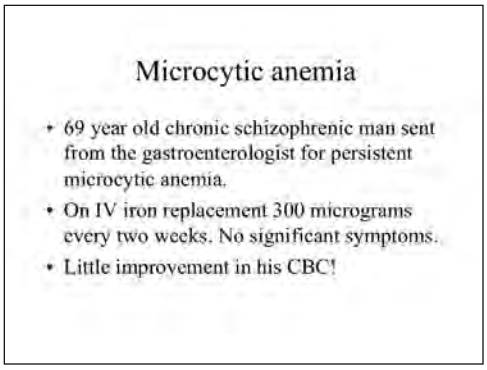


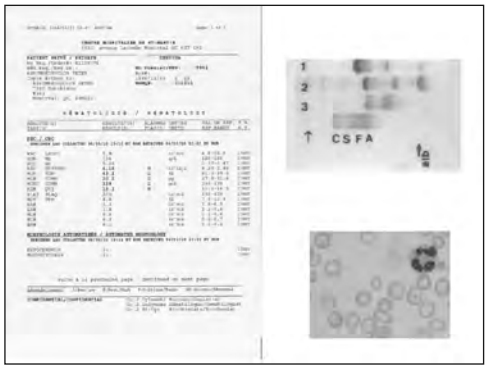


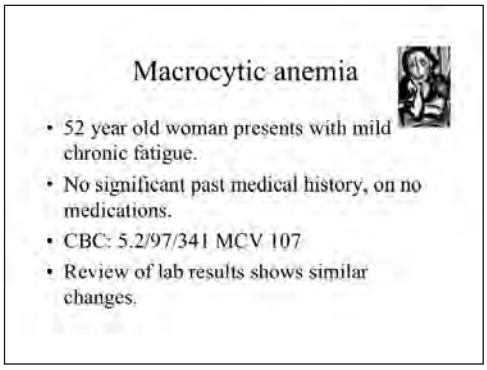


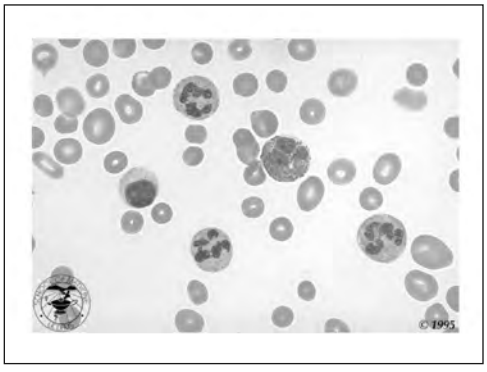


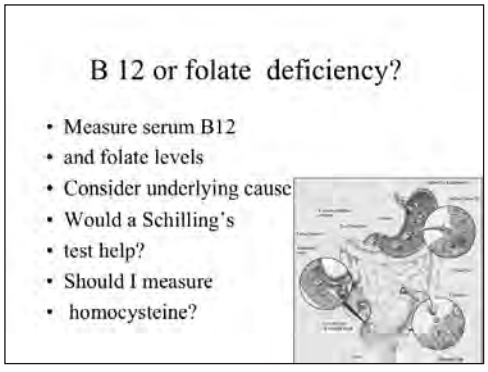


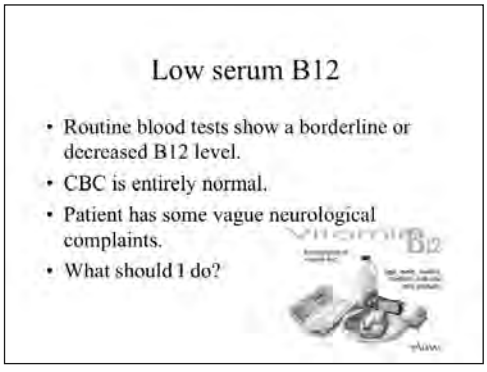












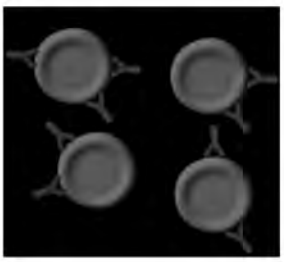
Low serum B12

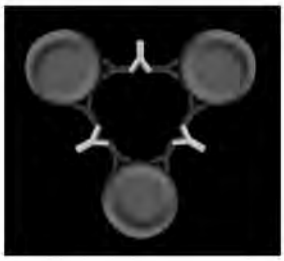
If true B12 deficiency is present, oral supplementation is often effective.

Give 1000 micrograms po daily.

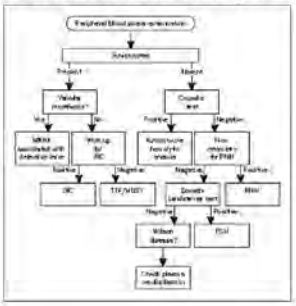
Macrocytic anemia

- Serum B12, folate are both normal, as is TSH. Bilirubin and LDH are both elevated.
- Blood smear shows reticulocytosis, and spherocytes.

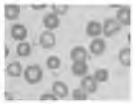


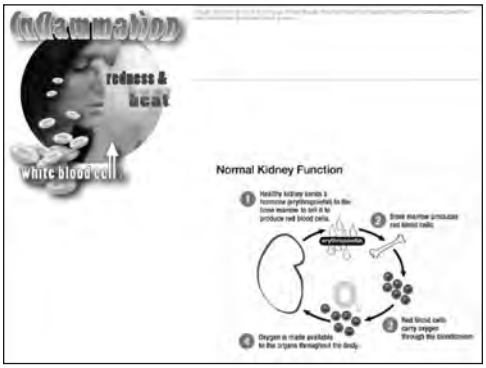


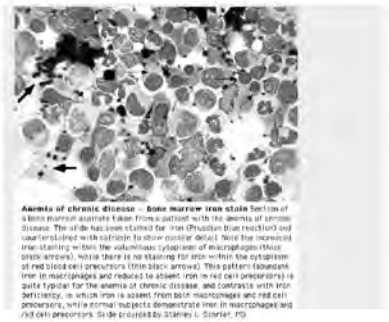
Evaluation of hemolysis



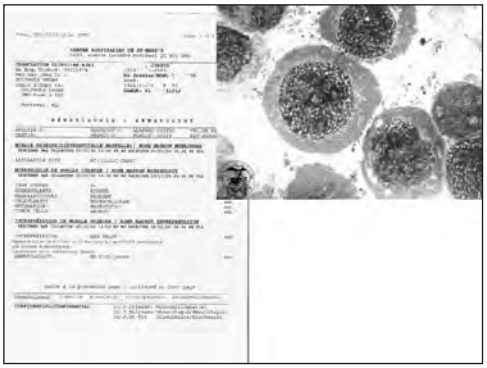
- Normochromic anemia







Anemia of chronic disease – bone marrow iron stain Section of a bone marrow aspirate taken from a patient with the anemia of chronic disease. The slide has been stained for iron (Prussian blue reaction) and counterstained with safranin to show nuclear detail. Note the increased iron staining within the voluminous cytoplasm of macrophages (thick black arrows), while there is no staining for iron within the cytoplasm of red blood cell precursors (thin black arrows). This pattern (abundant iron in macrophages and reduced to absent iron in red cell precursors) is quite typical for the anemia of chronic disease, and contrasts with iron deficiency, in which iron is absent from both macrophages and red cell precursors, while normal subjects demonstrate iron in macrophages and red cell precursors. Slide provided by Stanley L. Schrier, MD



- Transfusion therapy
- Erythropoetin

16:00 - 17:00 Laboratory Medicine, Rational Use

Julie St-Cyr MDCM, FRCPC
Director , Biochemistry Department, St. Mary's Hospital Centre;
Assistant Professor, Department of Pathology, McGill University

Research interests: I obtained my medical degree form McGill University in 1981. I then completed a residency in anatomic pathology at McGill University followed by 2 years of clinical pathology at the University Hospital of Vermont in Burlington. Upon returning to Canada in 1988, I needed to more years of internal medicine to be eligible for the medical Biochemistry exams. I then started as director of biochemistry in 1990 at SMHC and have been there ever since. I also teach clinical biochemistry to medical students at McGill.

Rational use of common biochemistry tests

DR. Julie St-Cyr
60th Annual Refresher Course
For Family Medicine

Learning objectives

- Identify the appropriate biochemistry tests recommended by the CTFPHC for the periodic health exam.
- Discuss laboratory tests that should not be ordered routinely in unselected patients.
- Review the common diagnostic tests as they apply to specific clinical conditions.

Periodic screening

- Why develop periodic screening procedures?
  asymptomatic adults harbor organic disease
  screening can detect a disease at an early stage
  early detection can alter the course of the disease.

The periodic health exam

CTF recommendations

- Do's= A & B recommendations
- Don'ts= D & E recommendations

CTF
A & B recommendations

Progressive renal disease	Urine dipstick	Adults with IDDM
Colorectal cancer	Multiphase screening with Hemocult	Adults > 50 years of age
Bacteriuria in pregnancy	Urine culture	Pregnant women
Cardiovascular events	Screening for type 2 DM	High- risk adults with HPT and hyperlipidemia

CTF
D & E recommendations

Diabetes Mellitus	Blood glucose fasting	General population
Ovarian cancer	CA125	pre and post menopausal
Pancreatic cancer	CA 19-9	General population
Prostate cancer	PSA	Males> 50
UTI	Urine dipstick/ culture	Elderly ambulatory males,
		elderly

Clinical Care & Research

Bacteriuria, asymptomatic	Against the use of UA in asymptomatic males and females except in 2 groups
Diabetes type 2	AAFP recommends screening in adults with HPT and hyperlipidemia

Clinical Care & Research

Colorectal cancer	AAFP strongly recommends screening adults 50 and older with FOBT annually
Lipid disorders	AAFP strongly recommends screening males >35 and females >45

Clinical Care & Research

Ovarian and pancreatic cancer	AAFP recommends against the use of u/s or serum markers in asymptomatic adults
Thyroid disease	AAFP recommends against screening for thyroid disease in non neonates or patients<60

Clinical Care & Research

Prostate cancer	Insufficient evidence to recommend for or against routine screening for prostate cancer using PSA

Common diagnostic tests in *unselected* ambulatory patients

A consequence of automation and social changes

Sequential multiple analyzer or SMA

Ambulatory screening

- Biochemical profiles are not routinely indicated for screening asymptomatic adults.
- Specific components of biochemical profiles that are not indicated for screening include: electrolytes
  liver enzymes

Not screening tests!

- Specific components of biochemical profiles that are not indicated for screening include: Total protein and albumin
  Amylase
  Uric acid
  Tumor markers
  Protein electrophoresis

Not screening tests

Sodium

1. not indicated in unselected ambulatory patients in the absence of manifestations of disease.
2. Useful in patients with the following signs and symptoms: rapid weight change, rapid change in fluid balance, rapid change in mental status, clinical evidence of dehydration or volume depletion

Not screening tests

Potassium

1. Screening is not indicated in unselected populations.
2. Is indicated in patients with chronic renal disease.
3. Is indicated in patients with hypertension at time of diagnosis, before initiating therapy

Not screening tests

4. Is indicated in patients with signs and symptoms suggestive of altered serum potassium concentration such as generalized or proximal muscle weakness, new atrial tachycardias

Not screening tests

Total CO2 or bicarbonate

1. Is not indicated in ambulatory patients in whom there are no signs or symptoms of disease.
2. Acid-base abnormalities do not occur until well into the course of chronic pulmonary, cardiac, renal or GI disease

Liver enzymes

- Enzymes of hepatocellular necrosis: AST and ALT
- Enzymes of cholestasis: Alk Phos and GGT

Liver enzymes

- Useful for diagnosing hepatocellular inflammation or obstruction as in patients with jaundice, on certain drugs, with alcohol abuse

The NEW ENGLAND JOURNAL of MEDICINE

Liver enzymes

Not screening tests!

Amylase

1. useful for diagnosing acute pancreatitis.
2. Not useful for: diagnosing pancreatic cancer.
3. Increases can be due to macroamylasemia

Not screening tests!

Tumor markers such as CEA, CA 125, CA 19-9:

1. are not useful as a screening assay for cancer detection in the normal population
2. Results can not be interpreted as absolute evidence of the presence or absence of cancer

Not screening tests!

- 3.serum markers are not specific for malignancy and values may vary by method
- 4. useful for evaluating patients' response to therapy
- 5. predicting recurrence

Not screening tests!

Uric acid

1. useful for assessment and management of patients with kidney stones, particularly uric acid stones.
2. serum uric acid levels are elevated in states of uric acid overproduction such as in leukemia and polycythemia

Not screening tests!

Serum protein electrophoresis:

1. Useful for monitoring patients with monoclonal gammopathies
2. Diagnosis of monoclonal gammopathies
3. Not considered an adequate screening test for monoclonal gammopathies when used alone

Common diagnostic tests used in *selected* ambulatory patients

Canadian Diabetes Association

CDA guidelines

- Screening for type 2 DM
- Glycemic targets
- Chronic kidney disease

CDA guidelines

- Screen for type 2 DM using a FPG *every 3 years* patients ≥ 40 years of age.
- A 75-g oral GTT is indicated when the FPG is 6.1-6.9 mmol/L and may be indicated when FPG is 5.6-6.0 mmol/L and if ≥ risk factors.

CDA guidelines

- Risk factors:
  - history of IGT or IFG
  - history of gestational DM
  - HPT
  - dyslipidemia
  - obesity

CDA guidelines

- Glycemic targets:
  Unchanged since 2003:

Type 1 or type 2	A1C (%)	FPG or PG	2 hr PG
diabetes	≤ 7	4.0-7.0	5.0-10.0

Home glucose monitoring

- Individuals conducting SMBG should receive initial instruction and periodic re-education regarding HGM
- In order to ensure accuracy of BG meter readings, meter results should be compared with the laboratory measurement of simultaneous venous FPG at least annually and when indicators of glycemic control do not match meter readings

CDA guidelines

- Screening for **CKD**:
  screen for microalbuminuria by measuring albumin and creatinine on a random urine to calculate the albumin to creatinine ratio or ACR.

CDA guidelines

- persistent microalbuminuria = 2/3 positive ACR tests over a 1-8 week interval
- ACR is positive if ≥ 2.0 and < 20.0 in men and ≥ 2.8 and < 28.0 in women.

CDA guidelines

- Screening for **CKD**:
  order ACR and eGFR annually in type 1 patients after 5 years
  order ACR and eGFR annually and at time of diagnosis in type 2 patients

DOQI

National Kidney Foundation

What is Chronic Kidney Disease

- *The presence of Kidney Damage or an eGFR < 60 ml/min/1.73m²* and
- *Present for ≥ 3 months* and
- *Not treated with dialysis or transplant*

The diagnosis of CKD is only present in patients with eGFR ≥60ml/min if other abnormalities (i.e. proteinuria, hematuria, anatomical) are also present.

Who should be tested for CKD?

CSN endorses a case finding approach to testing for CKD, which should be focused on high-risk groups.

CSN does not endorse mass population screening for CKD with either serum creatinine based tests or with urine dipstick testing.

Who should be tested for CKD?

- Patients with diabetes mellitus
- Patients with hypertension
- Patients with heart failure
- Patients with atherosclerotic coronary, cerebrovascular or peripheral vascular disease
- Patients with unexplained anemia
- Patients with a family history of ESRD
- First nations peoples

Why use eGFR?

It gives the health care practitioner a different sense as to a patient's level of renal function that they may not have appreciated by using simple serum creatinine measurements.

Measuring renal function: what's eGFR?

GFR

- ***Glomerular filtration rate (GFR)****: is the volume of fluid filtered from the renal glomerular capillaries into the Bowman's space per unit time.*
- *Normal for a 20 year old is ~ 120ml/min*

Methods to assess GFR

- Serum urea
- Serum creatinine
- Serum cystatin C
- Timed urine collections
  - Creatinine clearance
  - Inulin clearance
- Calculated GFR calculations
  - based on serum creatinine
  - many formulas including Cockcroft Gault and MDRD
- Nuclear medicine methods

Problems with timed collections

- Cumbersome
- Prone to error
- No longer recommended in most situations

Creatinine based approximations

1) Cockcroft-Gault equation

$$\text{CrCl (ml/min)} = \frac{(140 - \text{age}) \times \text{actual weight (kg)} \times 1.2 \text{ (if male)}}{\text{SCreat } (\mu\text{mol/L})}$$

Weight probably not available for lab to calculate

2) MDRD (Modification of Diet in Renal Disease)

6 variable or abbreviated version

$$\text{GFR(ml/min/1.73m2)} = 170 \times (\text{PCr})^{-0.999} \times (\text{Age})^{-0.176} \times (0.762 \text{ if female}) \times (1.21 \text{ if African American}) \times (\text{serum urea})^{-0.170} \times (\text{Albumin})^{+0.318}$$

Lab has patient age and gender – can do abbreviated version

eGFR equation provisos

- eGFR calculations may be less reliable in:
  - individuals with near normal GFR (>60 ml/min/1.73m²)
  - individuals with markedly abnormal body composition
    - extreme obesity
    - cachexia
    - paralysis
    - amputations
- *Controversies exist as to the applicability of these formulae to various ethnic groups and the very elderly*

Estimate of Glomerular Filtration Rate (eGFR)

- It is not recommended that clinicians rely on serum creatinine measurements alone when assessing kidney function.
- CSN calls for the reporting of kidney function as an estimate of glomerular function rate (eGFR) using equations and standardized creatinine measurements
- If neither eGFR reporting, nor calculators are available to a physician, tables based on serum creatinine and other variables are available to provide approximations of eGFR.

Is it just about GFR?

Should also assess urine protein losses

- 24 hour urines are no longer recommended
  - For same reasons as with GFR
- Urine dipsticks are affected by hydration status

Quantify protein excretion with random urine for:

- Urine albumin to creatinine ratio or
- Urine protein to creatinine ratio

What tests to order?

- Assess kidney function with
  - eGFR
    - As reported by lab
    - As calculated using equations (and PDA!)
    - As estimated by tables
  - Quantification of protein with random urine samples
    - Urine albumin to creatinine or
    - Urine protein to creatinine

Persistent significant proteinuria

- Persistent significant proteinuria is defined as:

  2/3 urine samples showing positive dipstick or

  ACR> 60 mg/mmol

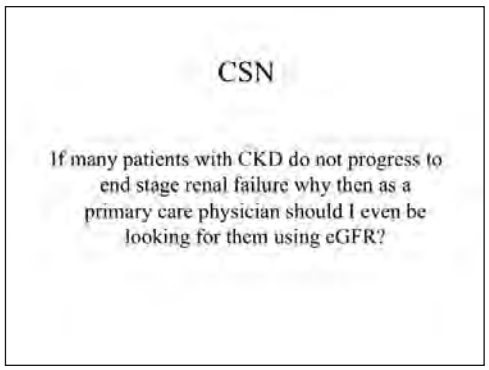
  or

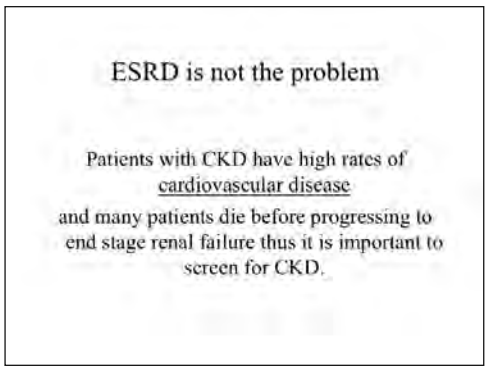
  PCR> 100 mg/mmol

CSN

CSN recommends that most patients with non-progressive CKD can be managed by non-nephrologists without referral.

The recognition that many patients with an eGFR between 30 and 60 ml/min/1.73m² do not have a high risk of progressive kidney disease is important.


CSN
If many patients with CKD do not progress to end stage renal failure why then as a primary care physician should I even be looking for them using eGFR?


ESRD is not the problem
Patients with CKD have high rates of cardiovascular disease
and many patients die before progressing to end stage renal failure thus it is important to screen for CKD.

Notes

Notes

Tuesday, Nov. 24 – Workshop F-07

16:00 - 17:00 Approach to Pneumonias

Michael D. Libman MD
Department of Medical Microbiology and
Division of Infectious Disease, MUHC;
Associate Professor, Faculty of Medicine, McGill University

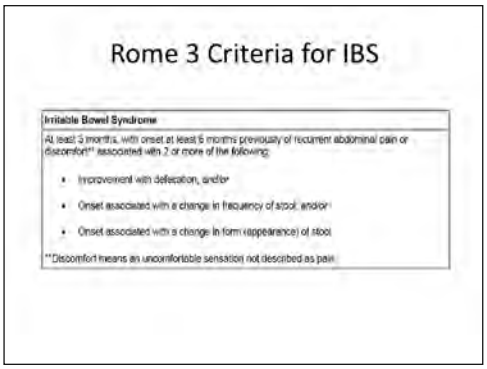
Research interests: Dr. Libman is currently Director of the Division of Infectious Diseases at McGill University and the McGill University Health Centre, and interim director of the J.D. MacLean Centre for Tropical Diseases at McGill University. He is also affiliated with St. Mary's Hospital, and consultant in microbiology and infectious diseases for the Quebec arctic region known as Nunavik. His primary interest is in tropical and travel medicine, as well as laboratory parasitology.

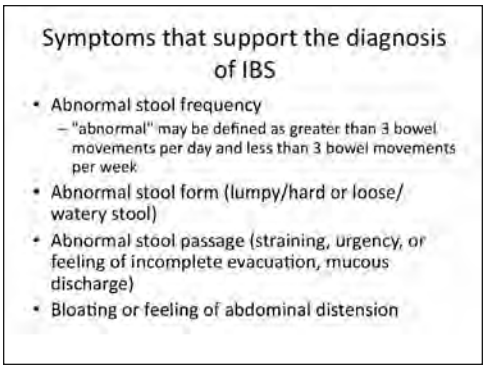
Notes

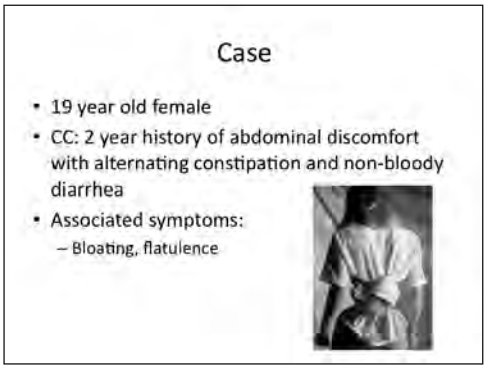
16:00 - 17:00 IBS Diagnosis and Management

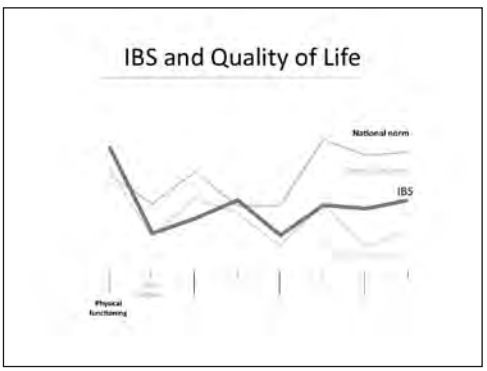
Gad Friedman MDCM, FRCP
Division of Gastroenterology, McGill University & MUHC;
Assistant Professor, School of Medicine, McGill University

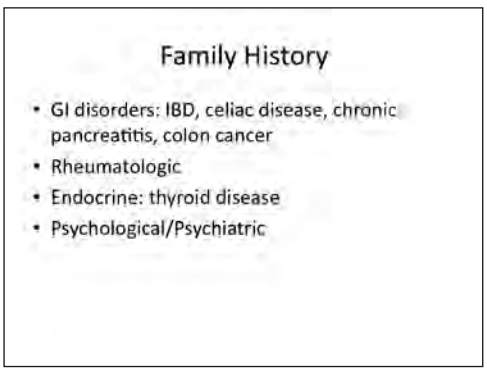
Research Interests: I have been a member of the Division of Gastroenterology of the Jewish General Hospital for over 11 years. After finishing medical school at McGill University, I completed m,y Internal Medicine residency at the Jewish General Hospital followed by a Gastroenterology fellowship at McGill. I subsequently did a year fellowship in interventional endoscopy with a focus on ERCP at the Montreal General Hospital. Although my primary interest is endoscopy with a focus on pancreaticobiliary disease, I enjoy a varied practice with large segment devoted to patients with inflammatory bowel disease. In the past two years, I have become involved in capsule endoscopy and hopfully will be starting capsule endoscopy at the Jewish General in the near future.











Food Triggers

- Caffeine
- Alcohol
- Spicy, fatty foods
- Tomato sauces
- Junk food
- Wheat products
- Dairy products
- Raw fruits, vegetables
- Eating too fast
- Eating too much
- Eating at irregular times
- Eating under stress

"I DIVORCED HIM AFTER I DISCOVERED THAT WHEN I EAT ALONE, I DON'T GET INDIGESTION."

Medications

- ASA/NSAIDs
- Antidepressants/antipsychotics
- Cardiac: CCB, ß blockers
- Antibiotics
- Calcium supplements

Red Flags

- Weight loss
- Rectal bleeding
- Arthritis, skin rash
- Nighttime symptoms
- Family history of colorectal cancer, IBD
- Age of onset >50 years of age
- Abnormal laboratory or physical exam findings

Case (cont.)

- Past medical history: migraines
- Medications: OCP, Advil prn
- Allergies: none
- Family history: thyroid disease, diverticulosis
- Habits: social smoker
- Social History: university student

Limited Screen for Organic Disease

- Physical exam
- Stool for occult blood
- Blood work
  - CBC, ESR/CRP, SMA-7, Ca, TSH, LFT, amylase
  - Celiac serology
  - Lactose, fructose and lactulose breath tests - not routine
  - Food allergy testing – not recommended

Table 3. Prevalence of organic diseases in patients meeting symptom-based criteria for IBS

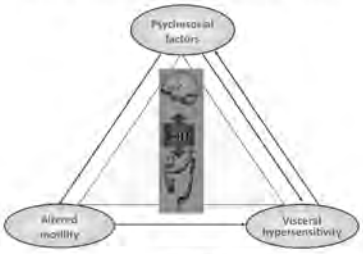
Organic GI disease	IBS patients (%)	General population (%)
Colitis/IBD[a]	0.51–0.98	0.3–1.2
Colorectal cancer[a]	0–0.51	0–6 (varies with age)
Thyroid dysfunction[b]	4.2	5–9
Gastrointestinal infection[b]	0–1.5	NA
Celiac sprue[b]	3.6	0.7
Lactose maldigestion[b]	38	26

[a]Data from Cash *et al.* (67). [b]Data courtesy of Moayyedi, *et al.* (personal communication, unpublished).

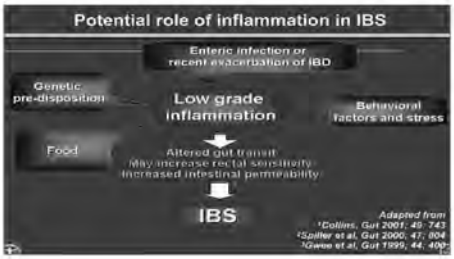
"The bowels are at one time constipated, another lax, in the same person. How the disease has two such different symptoms I do not profess to explain"

Cummings 1849 on irritable bowel syndrome

Brain-Gut Connection in IBS



Potential role of inflammation in IBS



Support for role of bacterial overgrowth in IBS

- Post-infectious IBS
- Altered enteric flora in IBS
- Probiotics
- Abnormal lactulose breath tests in IBS
- Efficacy of antibiotics in treating IBS

Treatment Options in IBS

- Lifestyle
  - Diet
  - Habits
- Psychological Issues
- Pharmacotherapy

Education

Patient Comments

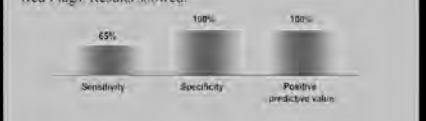
- "Doctors just say it's all in your head"
- "The doctors don't take it seriously"
- "No one ever told me about IBS"

Communicate the Positive IBS Diagnosis

- Inspire patient confidence
- Reassure the patient that he or she does not have cancer or another deadly disease
- Address the high accuracy of the diagnosis
- Discuss the high persistence of this diagnosis
- Emphasize nonpsychiatric pathophysiology of IBS

Positive Diagnosis of IBS: Validity of a Symptom-Based Approach

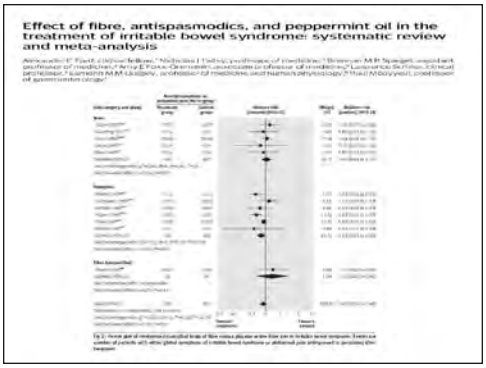
- A 2 year retrospective study confirmed the validity of an approach combining the Rome® criteria and absence of Red Flags. Results showed:



- At 2 years follow-up, no patients required revision of diagnosis

Therapeutic Options for Patients with IBS

- Antispasmodics
- Bulking agents
- Antidiarrheals
- Antidepressants
- Tegaserod/Alosetron
- Psychotherapy
- Alternative treatments



Bulking Agents

- 14 trials assessed bulking agents
  - wheat bran, corn fiber, calcium polycarbophil, psyllium, ispaghula husk
- Bulking agents effective at improving stool bulk and frequency
  - may be effective for constipation
- Bulking agents are not more effective than placebo at relieving global IBS symptoms

Grade B recommendation

Excess Bran = Gas

THE BLOATING HORSE.

Loperamide

- Only antidiarrheal agent studied in RCTs
- Loperamide successfully decreased stool frequency and improved consistency
  - is effective for diarrhea

Grade B recommendation

Antispasmodic Agents

- Dicyclomine (Bentylol) 10 mg QID more effective than placebo
  - 69% of patients experienced anticholinergic side effects vs. 16% of controls
- Global improvement reported with hyoscyamine (Levsin) without measuring improvement in specific symptoms

Grade B recommendation

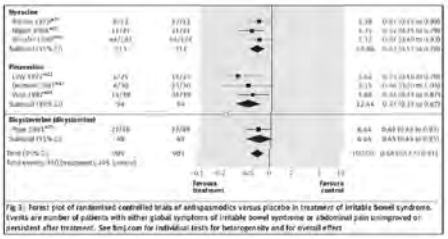
Symptomatic Therapies

Pinaverium Bromide (Dicetel®)

- Mechanism of action: antispasmodic (calcium-channel blockade)
- Efficacy:
  - Improvement in quality of life
  - No significant differences between antispasmodics and placebo for constipation, abdominal distention

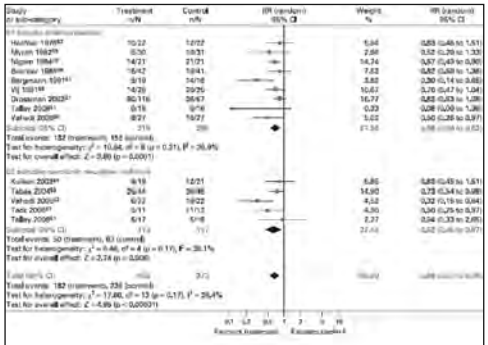
Trimebutine Maleate (Modulon®)

- Mechanism of action: antispasmodic (anticholinergic effects)
- Efficacy:
  - Improvement in abdominal pain at eight weeks, but not at four weeks compared to placebo
  - No difference between placebo and trimebutine in chronic idiopathic constipation



Antidepressants

- Meta-analysis of all antidepressant trials in IBS reported an odds ratio of 8 for improvement of pain and 4.4 for global well being[1]
- On average 3.2 patients needed to be treated (95% CI: 2.1 to 6.5 patients) to improve 1 patient's symptom[2].
- Low dose TCA (10-75 mg) are used
  - Nortryptilline, Desipramine



Antibiotics

- Rifaximin (Xifaxan®) is a nonabsorbable antibiotic with activity against gram positive and negative aerobes and anaerobes
- RCT of Rifaximin 400 mg tid for 10 days was superior to placebo for global symptoms primarily bloating

Probiotics

- Most are lactic acid producers, lactobacilli and bifidobacteria
- Thought to decrease inflammation, decrease colonic bile acid, and alter motility
- Trials of Bifidobacteria and Lactobacillus (i.e., VSL-3) have been inconsistent
- Most improvement in flatulence

The Utility of Probiotics in the Treatment of Irritable Bowel Syndrome: A Systematic Review

B. infantis 35624 has shown efficacy for improvement of IBS symptoms. Most RCTs about the utility of probiotics in IBS have not used an appropriate study design and do not adequately report adverse events. Therefore, there is inadequate data to comment on the efficacy of other probiotics. Future probiotic studies should follow Rome II recommendations for appropriate design of an RCT.

Serotonin Agonist/Antagonists

- Tegaserod(Zelnorm®)
  - 5-HT4 receptor partial agonist
    - increases velocity of propulsion through the colon
    - reduces visceral sensitivity
  - Indication: women constipation predominant IBS
- Alosetron(Lotronex®)
  - $5HT_3$ receptor antagonist
    - leading to slower transit, increased colonic compliance and decreased sensation
  - Indication: women with diarrhea predominant IBS[3]

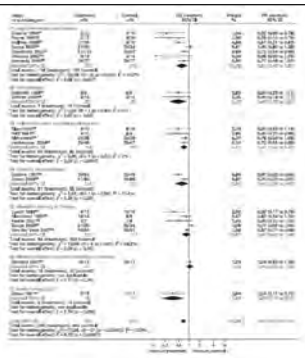
Both no longer available

Lubiprostone (Amitiza®)

- Locally-acting Type-2 chloride channel activator
- Patients with chronic constipation showed increased sBM and decreased straining. Improvement seen within 24-48 hrs[1].
- Phase 3 trial in IBS-C showed improvement in global symptoms[2]

Behavioral Therapies

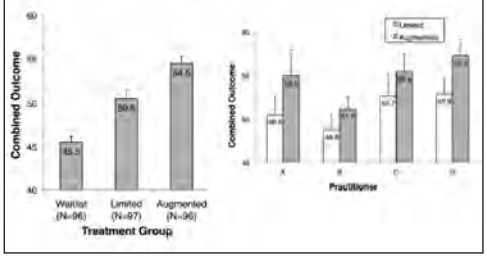
- Relaxation therapy
- Biofeedback
- Hypnotherapy
- Cognitive therapy
- Dynamic psychotherapy



Take Home Message

- Make a "Positive Diagnosis"
  - Inspire patient confidence
  - Explain the pathophysiology
- Tailor treatment to predominant symptoms
  - Be a "healer"

Patient and Practitioner Influences on the Placebo Effect in Irritable Bowel Syndrome



Thank - you

17:00 - 17:45 Satellite Symposium

Chair • **Ivan Rohan**

Pharmacotherapy of Mood and Anxiety Disorders: New Evidence for Improving Response and Remission Rates

Hani Iskandar MD

Supported through an unrestricted educational grant from AstraZeneca.

Notes

Notes

Wednesday, Nov. 25 – Breakfast Satellite

07:00 - 07:45 Breakfast Satellite Symposium

Chair • **Daniel E. Lalla**

Management of Osteoporosis and Fracture Risk in the Elderly

Martin Cohen MD, FRCPC

Supported through an unrestricted educational grant from Procter & Gamble

Notes

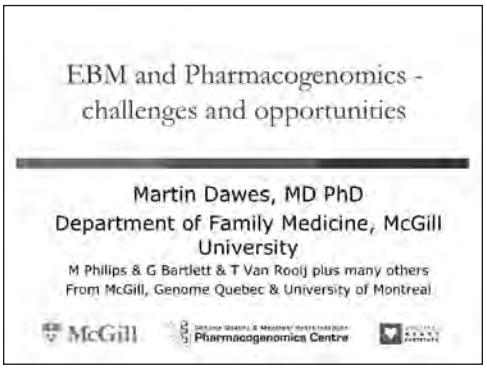
Notes

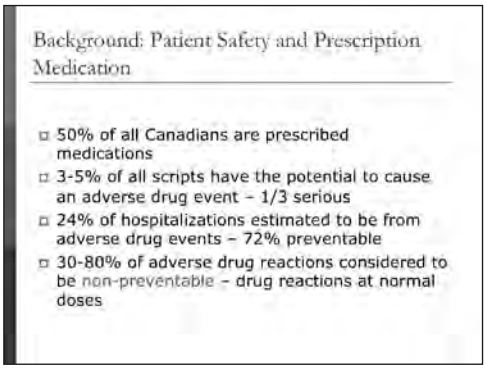
08:00 - 08:30 EBM and Pharmacogenomics - Challenges and Opportunities

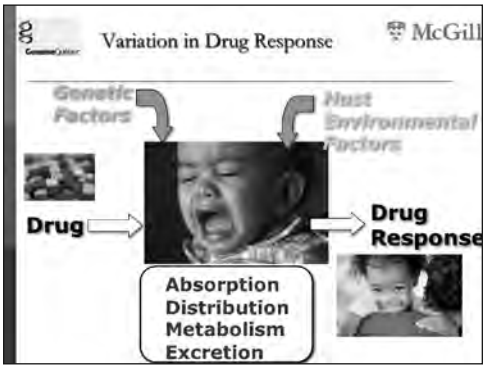
Martin Dawes MBBS, MD, FRCGP
Professor and Chair, Department of Family Medicine, McGill University

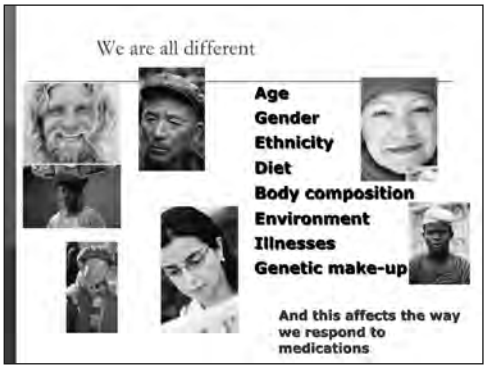
Research Interests: Dr Dawes has been a family physician since 1983 working full time for the first 10 years. He completed his PhD on weight gain in pregnancy in 1992. At that time he helped setup up the process for multi-centred audits for primary care in Oxfordshire using data from electronic records. In 1995 he developed a multi-disciplinary Masters programme in Evidence Based Health Care that is run at the University of Oxford. In 2000 he became Director of the Centre for Evidence-Based Medicine. Since October 2002 he has been chair of Family Medicine at McGill University.

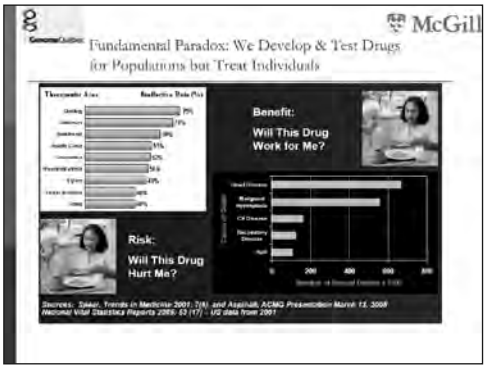
His clinical research includes pharmacogenomics as it relates to prescribing in primary care, and hypertension and in particular automated blood pressure monitoring. He also undertakes research into knowledge translation in particular information retrieval.











Pharmacogenetic Variation in Drug Metabolism

McGill

Polymorphic cytochrome P450 2D6 (CYP2D6)

CYP2D6 metabolizes drugs from many therapeutic classes.

5-8% of Caucasians are phenotypically '**poor metabolizers**' (PMs) homozygous for defective CYP2D6 function.

Multiple SNPs in the *CYP2D6* gene produce allelic variants that impair enzyme activity, ~ 106 alleles to date.

Examples of Pharmacogenetic Variation in Drug Metabolism

McGill

Polymorphic cytochrome P450 2D6 (CYP2D6)- poor activity of protein

Example #1

PMs cannot activate codeine to its analgesic metabolite morphine – lack of efficacy in pain relief.

Pharmacogenomics

- physicians do not know how a patient will react to a medication
- pharmacogenomics is being implemented and tested in specialist acute-care settings
- 1 in 4 primary care patients are prescribed a medication that causes adverse reactions due to genetic variability

Feasibility Project Objectives

- Develop the informatics pipeline to perform pharmacogenomics testing
- Determine if the testing can be completed and information received by physicians in time to modify dosing (24 hour period)
- Assess if the new buccal swabs provide the same quality of genetic information as blood samples
- Assess the cognitive impact of pharmacogenomics test results on treating physicians
- Provide information for design of randomized control trial of pharmacogenomics testing in primary care

Feasibility Project

- Medication: Warfarin
- Major adverse reaction: 7.2 major bleeds and 1.3 fatal bleeds per 100 patient years
- High variation in effective dosage between patients
  - 20% of dose variation - patient factors
  - 30-35% of dose variation - genetics
- Managed in primary care as well as acute care settings

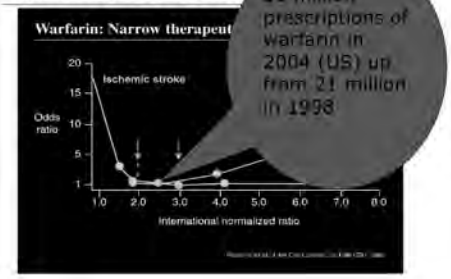
WARFARIN

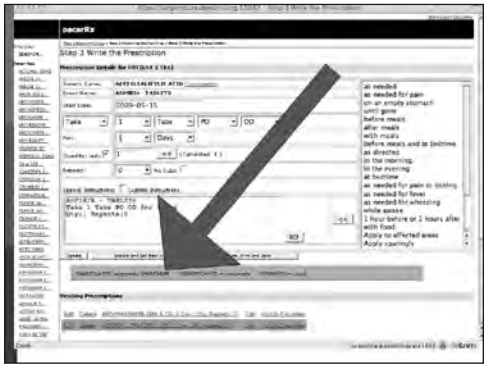
- Patients with atrial fibrillation who receive warfarin compared with placebo are less likely to have a stroke (NNT = 18 at 1.6 years)

Warfarin can make you bleed - badly

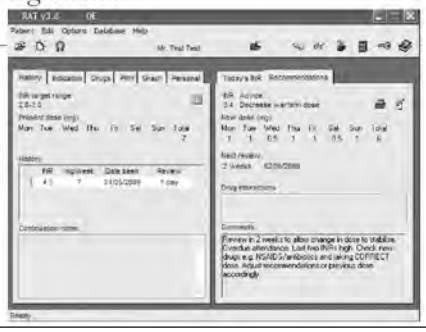
- Number needed to harm (bleed) 77
- 2% per year
  If you have a major bleed
  13.4% case fatality < 3/12 on Tx
  9.1% >3/12 on Tx

One third of the time peo… Warfarin are not in ta…





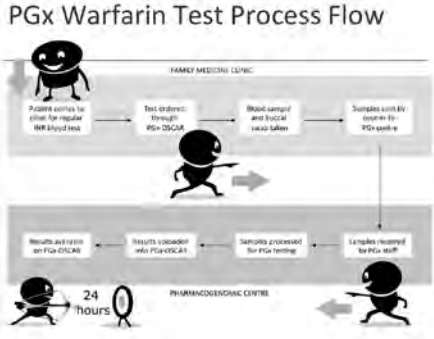
Algorithm



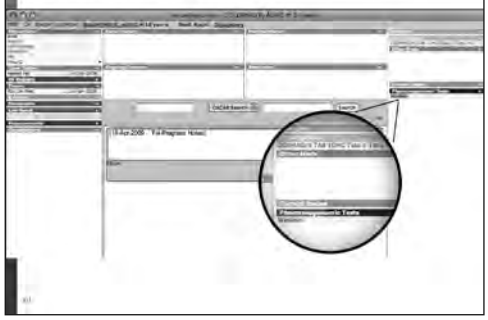
Algorithms

- Improve speed to desired INR at induction
- Improve time in INR range during maintenance
- But only by 3% or so – from 68% to 71%
- 6 to 10% of time is spent with INR>target
- High variation in effective dosage between patients
  - 20% of dose variation - patient factors
  - **30-35% of dose variation - genetics**

PGx Warfarin Test Process Flow



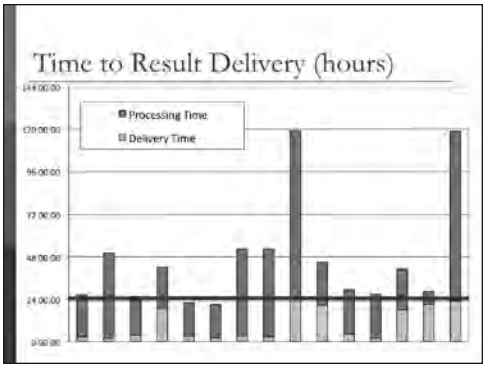
Test Results Pending

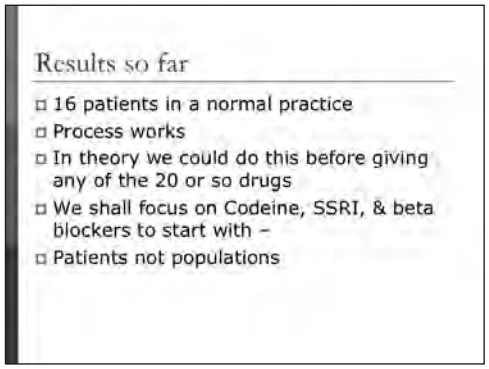


Order PGx test


Test Results Available


Dosing Guidance
Test Results


Time to Result Delivery (hours)
Processing Time
Delivery Time


Results so far
16 patients in a normal practice
Process works
In theory we could do this before giving any of the 20 or so drugs
We shall focus on Codeine, SSRI, & beta blockers to start with –
Patients not populations

Notes

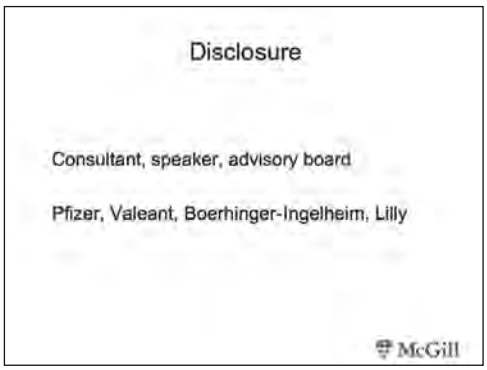
08:30-09:00 What's New in Pain Management?

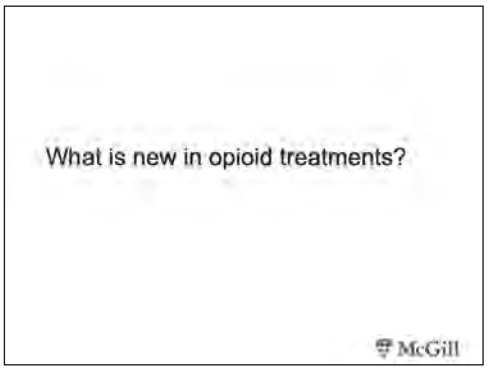
Mary-Ann Fitzcharles MD
Associate Professor, Division of Rheumatology, McGill University;
Rheumatologist, The Montreal General Hospital – MUHC

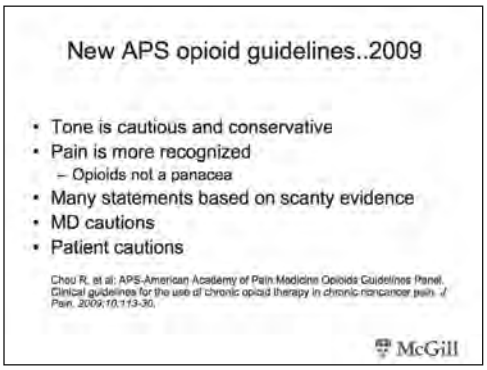
Research Interests: Mary-Ann Fitzcharles is currently an Associate Professor of Medicine in the Division of Rheumatology at McGill University. She received her medical education at University of Cape Town, South Africa, and completed specialist training in rheumatology at The London Hospital, Whitechapel, London, England.

She has been on faculty at McGill University since 1984, and has been a consultant rheumatologist to the McGill Pain Centre, at the Montreal General Hospital for the past 5 years. Academic activities have included being an examiner for the Royal College of Physicians of Canada Internal Medicine Specialist Examinations for 15 years, a member of Medical Admissions committee for medical students to McGill University for 15 years, and Director of Postgraduate Medical education at Royal Victoria Hospital, and member of postgraduate medical education board of McGill University.

In the past 10 years research interests have been in the area of pain and rheumatic diseases. Publications have been in the area of chronic pain in fibromyalgia, alternative treatments use in rheumatic diseases, and more recently evaluation of the pain experience in rheumatoid arthritis and osteoarthritis. Other research activities include evaluation of new compounds in the management of osteoarthritis, rheumatoid arthritis, psoriatic arthritis and fibromyalgia syndrome.







Opioid guidelines APS…. 2009

- Risk benefit ratio
  - Function
  - Constantly re-evaluate
- Opioids alone are not a magic potion
  - Co analgesics, non pharm measures
  - Best care is multimodal
- Pain is seldom 0..accept ↓ 2-3 points

McGill

Caution….opioid guidelines 2009

- Meticulous follow up needed
- No best opioid
  - Long-acting opioids…anecdotal benefit
- Driving cautions
- Long term effects and safety not known
- Be alert to indications of abuse
  - Do we truly need a contract for every patient?
  - Random drug urine checks

McGill

Patient needing more Opioids….

- Tolerance
  - Higher doses, but pain relief
- Hyperaglesia
  - Progressively higher doses, but persisting pain
  - Long time opioid use
  - Needing short acting
  - Shivering in am, or in day
- Addiction

McGill

Opioid side effects

- Depression
- Cognitive changes
  - Risk trauma, falling
- Hormonal changes
- Oedema

McGill

Tramadol

- Atypical analgesic
  - Acts on μ-opioid receptor, also serotonin, norepinephrine
  - Metabolite has strong μ-opioid
- Advantages
  - Less resp depression, less addiction
- Disadvantages
  - Kidneys, liver,20% protein bound

McGill

Fibromyalgia?

Proposed new criteria

McGill

Why new criteria for FM

- Old ACR criteria
  - for research
  - 20 years old
  - Only address pain
  - !!!!! Tender points…..a sore point for many
- New
  - Clinically applicable
  - Take into account symptoms other than pain
  - Addressing real concepts of FM

McGill

Which symptoms of FM are important to patient?

- Pain
- Fatigue
- Cognitive changes
- Mood
- Other somatic symptoms
- Activities
- Goals

McGill

Clinical Diagnostic and Severity Criteria for Fibromyalgia

American College Rheumatology, 2009

F Wolfe, Daniel Clauw, MA Fitzcharles, Don L. Goldenberg, KA Harp[1], RS Katz, PJ Mease, KD Michaud, Anthony S. Russell, IJ Russell, JB Winfield[10] and MB Yunus

McGill

Process for new criteria

- Committee think tank
- 55 centers, 1002 FM and controls
- MD and patient evaluations
- 134 variables….Random Forest data mining

McGill

New proposed criteria..

- Widespread pain 1-19
  - body regions
- Symptom severity 0-12
  - Unrefreshed sleep
  - Fatigue
  - Cognitive disturbance
  - Somatic symptoms
- NO TENDER POINTS

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Results

- Widespread pain index
  - Total of 19 body areas
  - best predictor
- Symptom severity (SS scale) correlates with many FM severity measures
- 80% agreement with old ACR criteria
- Applicable to clinical settings

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FM diagnosis

Pain >6, SS >4

Pain 3-6, SS >9

NO TENDER POINTS

McGill

Symptom based management of FM

- F – Fatigue
- I – Insomnia (sleep quality)
- B – Blues
- R – Rigidity (stiffness)
- O – 'Ow' (pain and work disability)

Boomershine CS, Crofford LJ. A symptom-based approach to pharmacologic management of fibromyalgia. *Nat Rev Rheumatol*. 2009;5(4):191-199.

McGill

FM: Pharmacologic treatments

- Multimodal treatment
- Anchor drug
  - Gabapentinoids, NSRI antidepressants
- Very low doses
- Best if can address > 1 symptom
- Combinations of drugs (never tested)
- No trials of opioids, stimulants

McGill

FM - sympathetic over activity

- Adrenergic nervous system
  - Adrenergic receptor maintains BP
  - Inverse relationship BP and pain
  - Genetic polymorphism of beta-2 adrenergic receptor..
    (G-protein coupled receptor- mediator of catecholamines)
- COMPT
  - enzyme degrades catecholamines
  - Haplotype met-val more pain
  - COMPT inducible by oestrogens
  - Do not clear catecholamines ↑ pain

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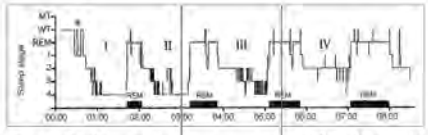
Sleep is necessary....

Sleep must be good quality.....

Sleep:3-5 non-REM et REM cycles



First 1/3 of the night
- Slow wave sleep (St 3&4) dominance

Last 1/3 of the night
- REM stage dominance

Disruption of sleep stages - memory, attention and PAIN

McGill

The need for good sleep

- Sleep architecture
- Drugs interfere with architecture
  - Alcohol, benzos, opioids
- Good sleep
  - 4-5 cycles, 6-7 hrs, not fragmented
- Gabapentinoids for sleep, improve architecture

McGill

Not every pain needs a pill

McGill

Questions about pain

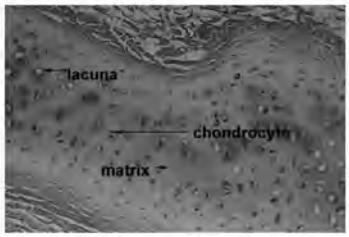
- Why is patient presenting
  - Worry about something serious
  - True suffering
  - Just curious!!!
- Exact features of pain
  - Timing
  - Effect on function
- Does pain need treatment

McGill

The 75 yr man with knee OA....

- Knees are painful
  - Expand on pain history
- X-ray shows "bad" arthritis
- What does this mean to patient?
- The examination
  - Some OA but
  - ++ anserine bursitis

The delinquent chondrocyte



No treatment yet

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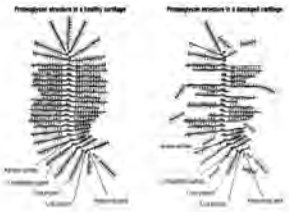
Proteoglycan ageing



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How to treat OA..

- Does pain need treatment
- Self management measures ++++
  - Self medication, education, exercise
- No longer daily pills
  - ↓ weight ↑ exercise
  - Barefoot or soft shoes
  - pm acetaminophen, a little NSAID
  - TOPICAL NSAID
  - Strong analgesics..Tramadol, opioids
  - injections

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Arthroscopy for OA

No benefit

Surgery vs Best care

(lavage + debridement + best care)

Mod-sev OA
- 92 and 86 pts
- 2 yrs follow up
- Womac & SF-36

No difference - endpoint or interim visits

Kirkley: NEJM Sept 2008

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Topical treatments

Please compound

Diclofenac 3%, 8% or 10% in Glaxal
ibuprofen 8%
Lidocaine 10%

Apply 2-4 times/day
100gm repeat*6

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Topical NSAIDS

Meta-analysis
- 25 RCT various pains
- 8/13 better than placebo
- Safety = placebo

- High conc in joint tissue
- Use if target tissue is close to skin
- Minimal systemic effect

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The mechanical back pain

- Normal part of life
- Watch for "red flags"
- Self management measures ++++
  - Self medication, education, exercise
- Activity and exercise
- Meta-analyses mostly
  - No evidence for most treatments

McGill

The miracle ball

- $20 on the internet
- Self massage at home
- Ideal for mechanical truncal pain

Which pain patient never gets better?

The catastrophizer

- Mostly innate trait, some cultural, family effect
- We do not know how to address

McGill

Summary

- New opioid guidelines….cautious
- Fibromyalgia…not just pain
- Fibromyalgia treatment …FIBRO
- Think about sleep
- What else besides a pill
  - Self management strategies
  - topicals

McGill

Notes

Wednesday, Nov. 25 – Morning Plenary

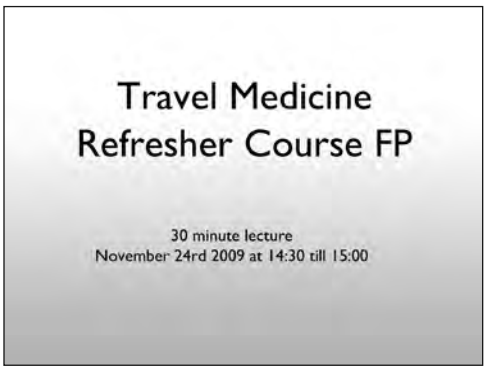
09:00 - 09:30 Travel Medicine

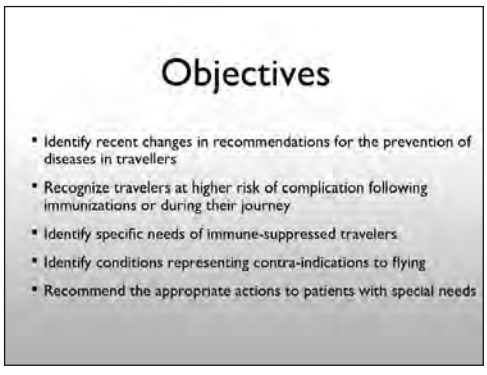
Dominique Tessier MD, CCFP, FCFP
Family Physician, Clinique médicale du Quartier Latin;
Chargée d'enseignement clinique, Université de Montréal;
Family physician, Post-exposure prophylaxis clinic,
Hôpital Saint-Luc du CHUM

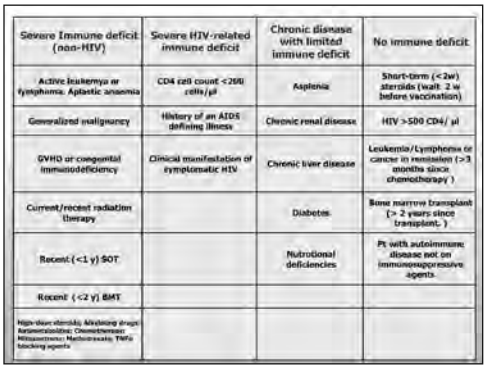
Research interests: Dominique Tessier graduated in medicine from the University of Montréal in 1981 and is certified and Fellow of the College of Family Physicians of Canada. She is a Past-President of the College of Family Physicians of Canada.

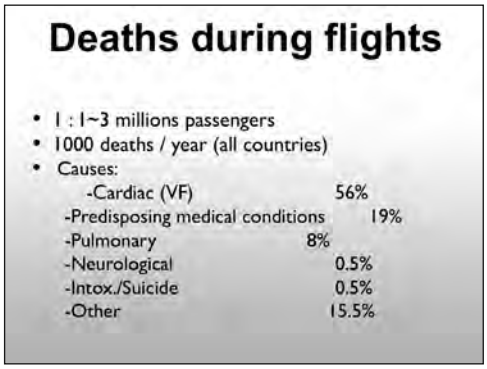
Her current practice includes providing services aimed at reducing travel-health problems to corporations and individuals across Canada. She is a family physician and Clinical Instructor at the Family Medicine Department of U. of Montréal. An important proportion of her practice is devoted to infectious diseases, including HIV/AIDS care. Her additional areas of interest include diversity and equity, women's health, violence against women and education.

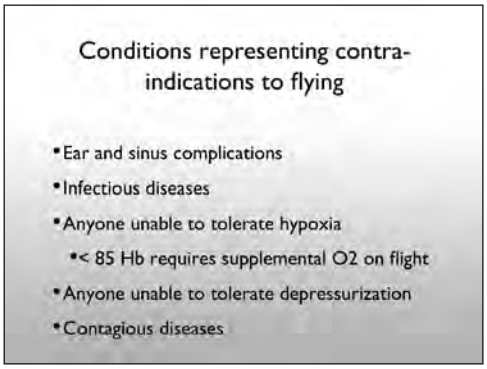
She is committed to educating and expanding public awareness on prevention and care of problems related to immunizations and Travel Medicine. With Dr Martin Brizard, she recently launched Bleu, a company providing training and services to support Health care professionals in their practice. She loves to travel!











Severe Heart Disease

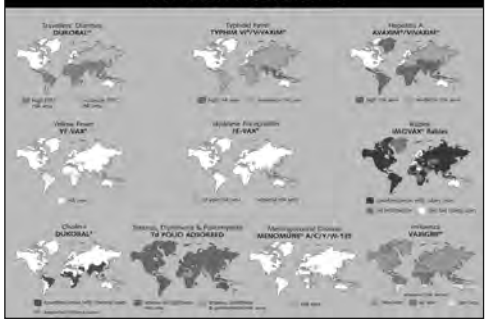
- No travel within at least 7 days of a **heart attack**
- **Stable Angina** is not usually a problem
- Travel following **angioplasty** is usually permitted after 3 to 5 days

Deep vein thrombosis

Alitalia

- **Hypercoagulability**
  - dehydratation (ROH - ⇩ fluids – dry air)
  - Hypoxia → ⇩ fibrinolytic ¢ endothelial activity
- **Veinous stasis**
- **Venous compression (poplit)**

Immunisations

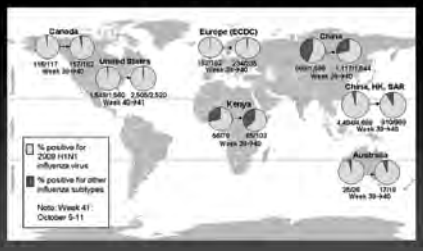


Vaccin dcaT

- Administrer une seule dose, à titre de rappel, aux personnes âgées de 7 à 17 ans, quel que soit le vaccin anticoquelucheux reçu antérieurement en primovaccination (à cellules entières ou acellulaire).
- Administrer une seule dose aux personnes âgées de 18 ans ou plus qui n'ont pas reçu le vaccin comprenant le composant acellulaire contre la coqueluche en particulier :
  - les parents ou les futurs parents de nourrissons et les autres contacts étroits (ex : grands-parents);
  - le personnel et les stagiaires des centres de la petite enfance;
  - le personnel et les stagiaires des écoles primaires et secondaires;
  - les travailleurs de la santé, incluant les stagiaires.

Vaccine against zona

Map: International Co-circulation of 2009 H1N1 and Seasonal Influenza



Hajj pilgrims

Seasonal Flu (Influenza) and H1N1 Flu Virus

- All travellers are strongly recommended to get the seasonal flu vaccine and, if available, the H1N1 vaccine at least two weeks prior to their departure for the Hajj.
- The age of Hajj pilgrims and Umrah performers should be above 12 and less than 65 years. Children and the elderly are advised to postpone their travels this year.
- The government and Ministry of Health of Saudi Arabia has also advised that persons with chronic diseases, immunocompromised individuals and pregnant women should postpone the Hajj (and Umrah) pilgrimages this year for their own safety.

Possible 2009 H1N1 Flu Screening for International Travelers

- When you travel internationally, officials may ask you to:
  - Pass by a scanning device that checks your temperature. In some countries this may be done before you disembark at your destination.
  - Have your temperature taken with an oral or ear thermometer.
  - Fill out a sheet of questions about your health.
  - Review information about the symptoms of 2009 H1N1 flu.
  - Give your address, phone number, and other contact information.
  - Be quarantined for a period of time if a passenger on your flight is found to have symptoms of 2009 H1N1 flu.
  - Contact health authorities in the country you are visiting to let them know if you become ill.
  - If you have a fever or respiratory symptoms or are suspected to have 2009 H1N1 flu based on screening, you may be asked to: Be isolated from other people until you are well. Have a medical examination. Take a rapid flu test (which consists of a nasal swab sample). Be hospitalized and given medical treatment, if you test positive for 2009 H1N1 flu.

Neuramidase Inhibitor

- Oseltamivir (Tamiflu®) 75mg :
  - 1 co die X 10 jours suite à un contact
  - 1 co bid pour 5 jours au début des symptômes
- Zanamivir (Relenza) Rotadisk 5 mg :
  - 2 inh die X 10 jours suite à un contact
  - 2 inh bid pour 5 jours au début des symptômes

Invasive pneumococcal and meningococcal disease: association with influenza virus and respiratory syncitial virus activity

- Correlations were determined between population-based data on IPD and MD during 1997–2003 and influenza virus and RSV surveillance data.
  - Incidence rate ratios of disease during periods of high influenza virus and RSV activity over the peri-seasonal and summer baseline periods were calculated.
  - The analyses comprised 7266 and 3072 cases of IPD and MD.

Meningococcal Vaccines

- Meningococcal polysaccharide vaccine (MPSV, Menomune)
  - 2 years of age and older
  - subcutaneous injection
- Meningococcal conjugate vaccine (MCV, Menactra)
  - 2 through 55 years of age
  - intramuscular injection

MCV Recommendations

- Recommended for:
  - all adolescents, preferably at 11 or 12 years of age
  - unvaccinated college freshmen living in a dormitory

Meningococcal Vaccine Recommendations

- Recommended for certain high-risk persons:
  - persistent complement component deficiency
  - functional or anatomic asplenia
  - HIV infection
  - microbiologists who are routinely exposed to isolates of ***Neisseria meningitidis***
  - military recruits
  - travelers to and U.S. citizens residing in countries in which N. meningitidis is

Meningococcal Vaccine Recommendations

- MCV is the preferred vaccine for persons 2 through 55 years
- MPSV should be used for persons 56 years of age or older, or if the person has a precaution for MCV (e.g., a history of Guillain-Barre' syndrome)

MCV Revaccination Recommendations

- High-risk persons who should be revaccinated with MCV:
  - persistent complement component deficiency
  - anatomic or functional asplenia
  - HIV infection
  - frequent travelers to or persons living in areas with high rates of meningococcal disease

Evidence of Measles, Mumps, and Rubella Immunity for Healthcare Personnel (HCP), USA

- Appropriate vaccination against measles, mumps, and rubella
  - 2 doses of measles and mumps vaccine
  - at least 1 dose of rubella vaccine, or
- Laboratory evidence of immunity, or
- Laboratory confirmation of disease
- Physician-diagnosed disease no longer recommended as evidence of measles or mumps immunity

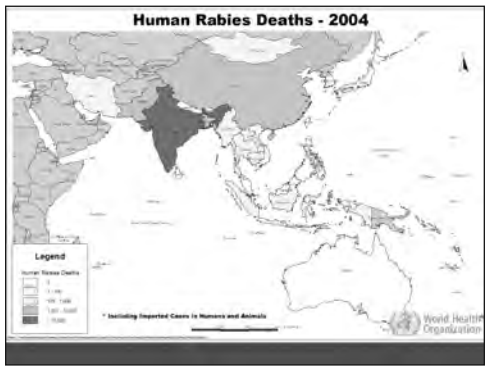
•FDA Approves New Indication for Gardasil to Prevent Genital Warts in Men and Boys

- The U.S. FDA today approved use of the vaccine Gardasil for the prevention of genital warts (condyloma acuminata) due to human papillomavirus (HPV) types 6 and 11 in boys and men, ages 9 through 26.
- Each year, about 2 out of every 1,000 men in the United States are newly diagnosed with genital warts.
- This vaccine is the first preventive therapy against genital warts in boys and men ages 9 through 26

FDA Approves New Indication for Gardasil to Prevent Genital Warts in Men and Boys

- Gardasil effectiveness was studied in a randomized trial of 4,055 males ages 16 through 26 years old.
- The results showed that in men who were not infected by HPV types 6 and 11 at the start of the study, Gardasil was nearly 90 percent effective in preventing genital warts caused by infection with HPV types 6 and 11.
- Studies were conducted to measure the immune response to the vaccine in boys ages 9 through 15.

Immuno déficicience sévère (non-VIH)	Immuno déficience sévère chez VIH	Maladie chronique avec déficit immunitaire modéré	Non immuno déprimé
Leucémie active ou lymphome. Anémie aplastique	CD4 <200 cells/ µl	Asplénie	Stéroïdes court terme (<2semn.)
Generalized malignancy	SIDA	Maladie rénale chronique	VIH >500 CD4/ µl
GVHD ou immunodéficience congénitale	Manifestation clinique d'un VIH symptomatique	Chronic liver disease	Leucémie/Lymphome en cours ou remission (>3 months since chemotherapy)
Radiothérapie en cours		Diabète	Greffe de moelle osseuse (> 2 ans)
Greffe d'organe récente (<1 an)		Déficiences nutritionelles	Maladie auto immunitaire sans traitement immunosuppresseur agents
Greffe moelle osseuse (<1 an)			
High-dose steroids, Alkylating drugs, Antimetabolites, Chemotherapy, Mitoxantrone, Methotrexate, TNFα blocking agents			



High mountain sickness prevention Acétazolamide (Diamox)

- 125 mg (1/2 co.) à 250 mg (1 co.) bid
  - 24 heures avant d'atteindre 2500 à 3000 m
  - Poursuivre x 2 à 5 jours
- OU
- 125 mg bid ou 250 mg au coucher si symptômes
  - La dose au coucher est la plus importante
  - C.I. : Glaucome

High mountain sickness prevention and treatment Dexamethasone (Décadron)

- Améliore la dysfonction endothéliale causée par l'hypoxie
- Stimule la réabsorption de l'eau et du sodium a/n alvéolaire
- Augmente la genèse d'oxyde nitrique
- 4-8 mg Bid en prévention
- 8 mg stat puis 4 mg Qid en traitement

High mountain sickness prevention and treatment Nifedipine (Adalat)

- Anticalcique 'vasodilatateur'
- Adalat PA 20 mg Bid en prévention
- Adalat 10 mg s/l puis PA 20 Bid en traitement
  - Diminuer la dose en présence d'insuffisance hépatique ou d'anti-hypertenseur

High mountain sickness prevention and treatment Salmétérol (Serevent)

- 2 inhalations 50 µg (100 µg) bid
- ↓ 50% (74% à 33%) le risque d'œdème pulmonaire lorsque débuter 24h avant le départ

Notes

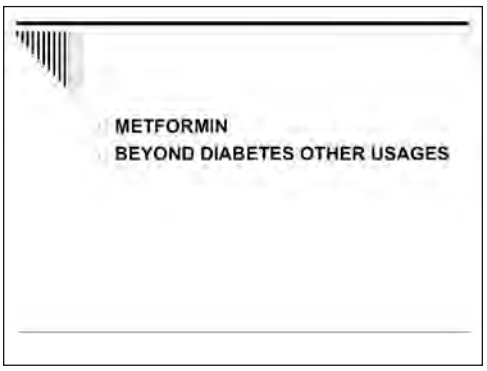
Wednesday, Nov. 25 – Morning Plenary

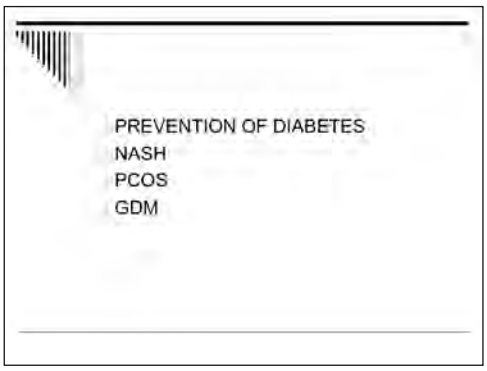
09:30-10:00 Metformin, beyond Type 2 Diabetes

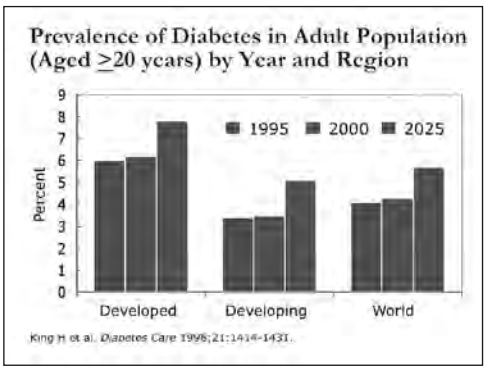
Tina Kader MD, FRCPC, CDE
Assistant Professor , Department of Medicine, McGill University;
Certified Diabetes Educator

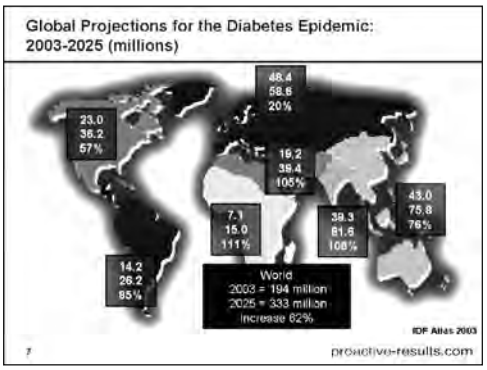
Research Interests: Dr. Tina Kader is a staff physician in the JGH's Division of Endocrinology, a certified diabetes educator and a member of the Executive Committee of the Canadian Diabetes Association. She received her medical degree from McGill University and completed post graduate studies in internal medicine and endocrine training at the JGH and McGill.

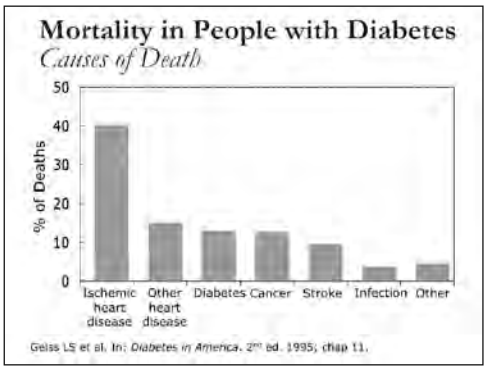
Delivering approximately 60 lectures a year, Dr. Kader is committed to educating the public, medical students, residents, general practitioners and fellow endocrinologists about type 2 diabetes. Among her interests are new research and treatments for diabetes, as well as new techniques in diabetes related education.

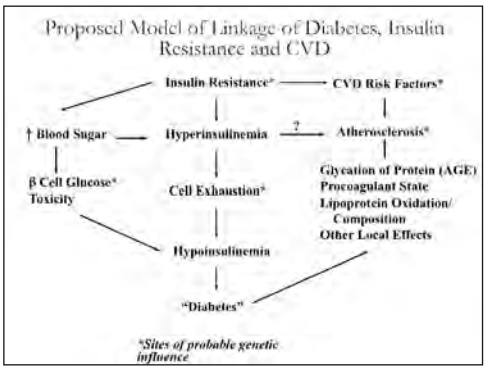




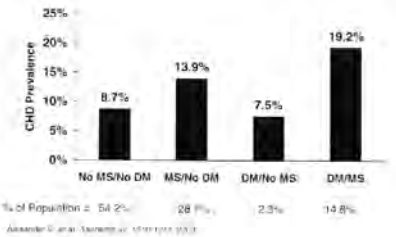


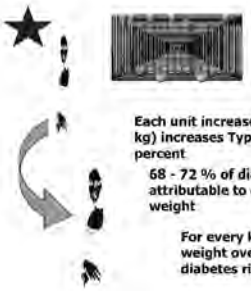






Prevalence of CHD by the Metabolic Syndrome and Diabetes in the NHANES Population Age 50+





Each unit increase in BMI (about 2.7 - 3.6 kg) increases Type 2 diabetes risk by 12.1 percent

68 - 72 % of diabetes risk in the U.S. is attributable to or associated with excess weight

For every kilogram increase in weight over 10 years, Type 2 diabetes risk increases 4.5 %

Ford et al. *Amer J Epidemiol* 146:214,1997

Feasibility of Prevention

Prevention of type 2 diabetes should be feasible since:

- There is a long period of glucose intolerance that precedes the development of diabetes
- Screening tests can identify persons at high risk
- There are safe, potentially effective interventions

Progression of IGT to Type 2 Diabetes

- The rate of progression is 1-9% per year, depending on the population
- Obesity, higher fasting glucose level, prior history of GDM, and other factors increase the rate of progression

Edelstein et al. Diabetes 1997; 46:701-10.

Finnish Diabetes Prevention Study (NEJM 2001;344:1343-50)

- 352 middle-aged IGT (172 men,350women)
- Followed 3.2 years
- Lifestyle goals : 5%weight loss, 30mins exercise/day
- 7 sessions first year,quarterly thereafter
- Weight loss 4. 2kg (4.7%) in Intervention 0.8Kg (0.9%) in Controls at 1 yr, 3.5v(0.8Kg at 2 yrs
- Incidence: Diabetes reduced by 58%
- 32 v 78/1,000 person years

DPP Goals: Primary

- To prevent or slow the development of type 2 diabetes in persons with impaired glucose tolerance (IGT)

Primary Outcomes

- Annual Fasting plasma glucose (FPG) and 75 gm OGTT
  - FPG $\geq$ 126 mg/dL (7.0 mmol/L) or 2-hr $\geq$ 200 mg/dL (11.0 mmol/L), confirmed with repeat OGTT
- Semi-annual FPG
  - $\geq$ 126 mg/dL, confirmed

Study interventions

Eligible participants

↓

Randomized

↓

Standard lifestyle recommendations

Intensive Lifestyle (n = 1079)	Metformin (n = 1073)	Placebo (n = 1082)

Lifestyle Intervention

- Program supervised by a case manager
- 16 session core curriculum (20-22 hours over 16 weeks)
  - Nutrition (keep food diaries, count grams of fat)
  - Exercise (goal of $\geq$ 150 minutes per week)
  - Stress management
- Monthly visits post-core curriculum
  - Minimum of every-other-month contact must be in person

Metformin

- Approved for use in type 2 diabetes
- Mechanism of action
  - Lowers hepatic glucose production
  - Increases glucose uptake/utilization
  - Decreases intestinal glucose uptake
  - Does not stimulate pancreatic insulin production
- Metabolism
  - Excreted unchanged in urine
  - Significant renal tubular excretion
  - Half-life ~ 6 hours

Interventions

Medications

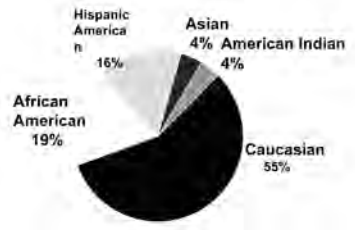
Metformin- 850 mg per day escalating after 4 weeks to 850 mg twice per day

Placebo- Metformin placebo adjusted in parallel with active drugs

Eligibility Criteria

- Age $\geq$ 25 years
- Plasma glucose
  - 2 hour glucose 140-199 mg/dl (7.8- <11.1 mmol/L) and
  - Fasting glucose 95-125 mg/dl (5.3- <7.0 mmol/L)
- Body mass index $\geq$ 24 kg/m^2
- All ethnic groups goal of up to 50% from high risk populations

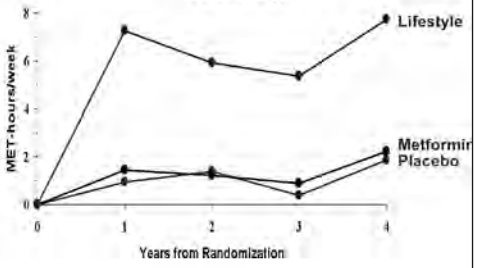
Study Cohort



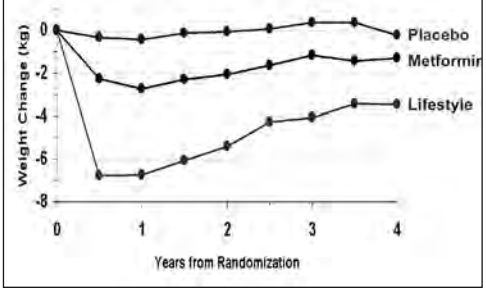
Lifestyle Intervention: Physical Activity Results

- 74% of volunteers assigned to intensive lifestyle achieved the study goal of $\geq$ 150 minutes of activity per week at 24 weeks

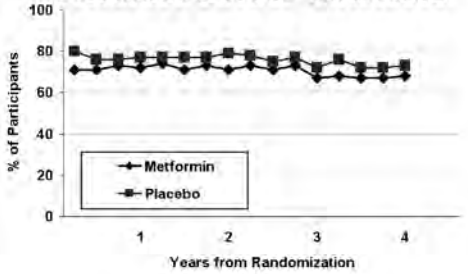
Mean Change in Leisure Physical Activity



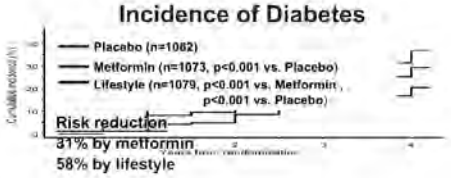
Mean Weight Change



Percent Taking ≥ 80% of Prescribed Dose of Medication



Incidence of Diabetes

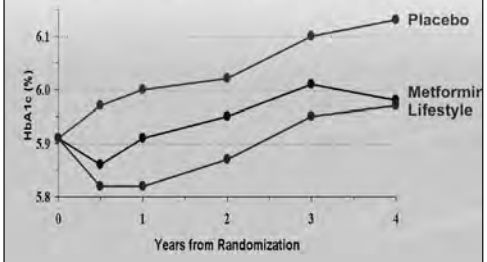


Risk reduction
31% by metformin
58% by lifestyle

Effect of Treatment on Incidence of Diabetes

	Placebo	Metformin	Lifestyle
Incidence of diabetes (percent per year)	11.0%	7.8%	4.8%
Reduction in incidence compared with placebo	—	31%	58%
Number needed to treat to prevent 1 case in 3 years	—	13.9	6.9

Mean Change in HbA_{1c}



Adverse Events
(rates per 100 person years)

	Placebo	Metformin	Lifestyle
Death	0.16 0.10	0.20	
Hospitalization	7.9 8.0	8.4	*
GI Symptoms	30.7 12.9	77.8	*
Musculoskeletal	21.1 24.1	20.0	

Background

- The DPP has demonstrated significant reductions in the incidence of Diabetes using either Intensive Lifestyle or Metformin
- Cardiovascular Disease is the leading cause of early Mortality and Morbidity in Diabetes
- At present the DPP has insufficent events to evaluate treatment effects on CVD outcomes
- The metabolic syndrome is a high CVD risk state, associated with glucose intolerance and insulin resistance, that is a target for intervention with lifestyle and possible drug therapy.

Cumulative Incidence of MS by Treatment Group

Risk reduction:
Lifestyle vs. Placebo 41%*
Metformin vs. Placebo 17%*
Lifestyle vs. Metformin 29%*

* $p < 0.05$; * $p < 0.001$

Conclusions

- Lifestyle and Metformin both reduce the incidence of Diabetes and the Metabolic Syndrome in subjects with impaired glucose tolerance
- Lifestyle is more effective than Metformin
- Though these results suggest that DPP interventions may reduce the incidence of CVD in IGT subjects, further follow up, with CVD outcomes, is needed

Nonalcoholic Fatty Liver Disease (NAFLD)

Objectives

- Terminology
- Epidemiology
- Pathogenesis
- Clinical Presentation
- Diagnosis
- Prognosis
- Treatment

NAFLD

- Spectrum of liver disease associated with hepatic steatosis that occurs in the absence of excessive alcohol consumption
- Diagnosis of exclusion – no test exists that can distinguish NAFLD from alcoholic fatty liver
- Often associated with insulin resistance and features of the metabolic syndrome

Terminology

- **NAFL** = nonalcoholic fatty liver
  - Characterized by steatosis without inflammation or fibrosis
  - Benign and reversible condition
  - Main cause of elevated liver enzymes among the general population

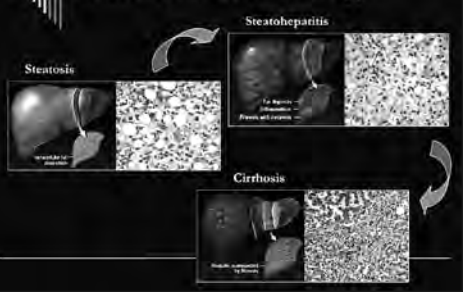
Terminology (cont)

- **NASH** = nonalcoholic steatohepatitis
  - Fatty liver accompanied by hepatocellular damage plus inflammation
  - Occurs in ~20% of NAFLD cases
  - Transition from NAFL to NASH not clearly demarcated

Terminology (cont)

- **Cirrhosis** – regenerative nodules and formation of fibrosis/scar tissue
  - Up to 20% of patients with NASH progress to advanced liver disease (cirrhosis or liver failure)
  - Risk of HCC in NASH-related cirrhosis is comparable to HCV

Spectrum of NAFLD Pathology



Risk Factors

Metabolic Syndrome: cluster of disorders including central obesity, insulin resistance with or without type 2 DM, dyslipidemia and hypertension
- >90% of patients with NAFLD have at least one component of the metabolic syndrome
- Approximately 1/3 have the complete syndrome

Morbid Obesity

Four studies evaluating > 600 morbidly obese patients undergoing gastric bypass
- All patients underwent intraoperative liver biopsies
- Prevalence of NAFL ranged from 30-90% and NASH was documented in 33-42%.
- ***> 2/3 of morbidly obese patients undergoing gastric bypass surgery have NAFL/NASH***

Abrams GA, et al. Hepatology 2004;40:475-483; Fransidides CT, et al. J Gastrointest Surg 2004;8:845-855; Barshes NR, et al. Obes Surg 2004;14:43-51; Baumer C, et al. Arch Surg 2003;138:1340-1345

Type 2 Diabetes Mellitus

Recent study surveyed 100 patients with type 2 DM and used U/S to screen for NAFLD
- Detected fatty liver in 50% of patients
- Performed subsequent liver biopsy in those with NAFLD:
  - NAFL: 13%
  - NASH: 86%
  - Fibrosis: 22%

Bonnet et al. J Hepatol Hepatol 2003;19:444-448

Dyslipidemia

Canadian study used U/S to screen 95 adults with dyslipidemia
- Detected fatty liver in 50%
- Steatosis was particularly common in individuals with moderate to severe hypertriglyceridemia or mixed dyslipidemia
- Hypertriglyceridemia and mixed dyslipidemia increased the risk for hepatic steatosis by ~5-fold

Assy N, et al. Dig Dis Sci 2000;45:1929-1934

Other Factors Associated with NAFLD

- Abdominal surgeries: bariatric surgery, extensive small-bowel resection
- Nutritional: rapid weight loss, TPN
- Drugs/Toxins: corticosteroids, methotrexate, amiodarone, tamoxifen
- Other metabolic disorders: carnitine deficiency, lipodystrophy, dysbetalipoproteinemia

Pathogenesis

- Pathogenesis is not fully understood
- Insulin resistance is thought to be the key pathogenic feature leading to hepatic fat accumulation
  - Insulin resistance causes an increase in free fatty acid (FFA) influx into the liver → drives hepatic triglyceride production
  - Increased serum insulin and glucose levels also promote de novo lipogenesis by upregulating lipogenic transcription factors
  - NAFLD may in turn result in hepatic insulin resistance, which is thought to be triggered by hepatic TG accumulation → may exacerbate overall insulin resistance

Adams LA, et al. Gastroenterology 2005;129:113-121

Pathophysiology

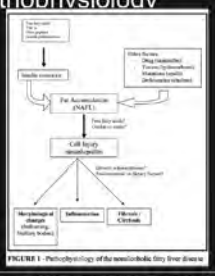


FIGURE 1 - Pathophysiology of the nonalcoholic fatty liver disease

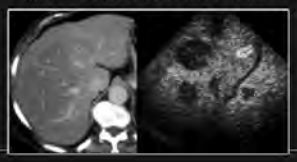
Salgado W, et al. Acta Cir. Bras. 2006; 21

Clinical Presentation

- Variable clinical presentation
- Typically asymptomatic, but may have hepatomegaly and abdominal discomfort
- Liver enzymes may be normal in >75% of cases, making them insensitive in detecting NAFLD
  - When increased, usually only modestly and limited to aminotransferases
  - ALT upper limits of normal: <30 in M, <20 in F

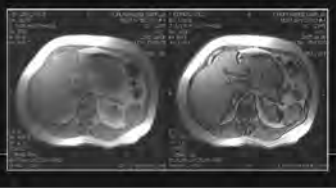
Diagnosis

- Diagnosis of NAFLD can often be made by imaging studies, including U/S, CT or MRI – detects presence of fat



Diagnosis (cont.)

- MR spectroscopy accurately measures hepatic triglyceride content
  - Has advantage over U/S, CT and MRI as it is quantitative rather than qualitative

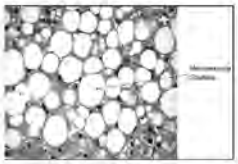


Diagnosis (cont.)

- No imaging studies can differentiate between the histological subtypes of benign steatosis or aggressive NASH, or stage the degree of fibrosis
  - Need tissue for staging and to make diagnosis of NASH

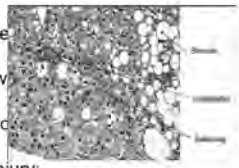
Histology

Histologic diagnosis NAFL requires presence of ≥ 5% steatosis
- Indistinguishable from alcoholic fatty liver



Histology

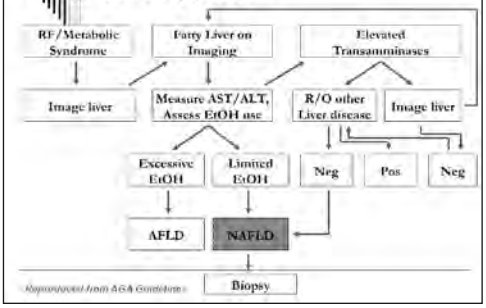
- NASH involves presence of steatosis with evidence of inflammation and hepatocyte injury:
  - Ballooning
  - Mallory bodies



Histology

- Histologic evidence of steatohepatitis may disappear with progression to cirrhosis
  - Thus, significant proportion of cryptogenic cirrhosis is likely related to unrecognized NASH

Diagnostic Approach



Reproduced from AGA Guidelines

Prognosis

- NAFLD is associated with an increased risk of all-cause death
  - Likely related to complications of insulin resistance (i.e. vascular disease) and progressive liver disease (i.e. cirrhosis, HCC)
- Presence of NAFLD exacerbates the severity of liver injury from other hepatotoxic agents, such as HCV and EtOH
- Risk factors associated with advanced hepatic fibrosis include age > 45yr, DM, increased BMI and AST/ALT ratio of >1

Matteoni C, et al. Gastroenterology 1999;116:1413-1419.

Prognosis (cont.)

- Patients with bland steatosis (NAFL) have a benign liver-related prognosis
  - 1.5% develop cirrhosis
  - 1% die from liver-related causes over 10-20 years
- Almost 30% of patients with NASH and fibrosis become cirrhotic within 5-10 years
  - Those with biopsy-proven NASH have a liver-related death rate of ~10%
- NASH cirrhosis may develop into HCC
  - ~13% of cases of all HCC are related to NASH cirrhosis
  - Endstage NAFLD accounts for ~5-10% of liver transplants

Matteoni C, et al. Gastroenterology 1999;116:1413-1419.

Treatment

- Aim to improve insulin sensitivity and modify underlying metabolic risk factors
  - Diet and exercise
  - Insulin Sensitizing Agents (metformin, TZD)
  - Lipid lowering medications (statins, fibrates)
- L-Carnitine supplementation

McCullough AJ. N Engl J Med 2006; 355: 2361-3.

Treatment (cont.)

- Beneficial according to preliminary studies:
  - Insulin sensitizers: TZD > metformin
- Benefit unproven by preliminary studies
  - Lipid lowering agents
  - Antioxidants
  - Probiotics (animal models only)
- Not beneficial
  - Ursodiol

Metformin vs. TZD

- One of 5 Metformin studies observed improvement in liver histology
- Three of 3 TZD studies demonstrated improvement in histological features
- All Metformin and TZD studies revealed decrease in aminotransfereases

McCullough AJ. N Engl J Med 2006; 355: 2361-3.

Metformin vs. TZD

- Tiikkainen and colleagues compared Metformin and Rosiglitazone
  - Demonstrated that Rosiglitazone, and not Metformin:
    - Improved peripheral insulin sensitivity
    - Decreased hepatic steatosis
    - Increased serum adiponectin (inhibits FFA uptake, stimulates FFA oxidation and lipid export, enhances insulin sensitivity)

Tiikkainen M, et al. Diabetes 2004; 53:2169-2178.

Pioglitazone

- Belfort et al performed first placebo-controlled trial investigating Pioglitazone in patients with biopsy-proven NASH and glucose intolerance or type 2 DM
  - Improvement in hepatic histologic features, except fibrosis
  - Decrease in peripheral and hepatic insulin resistance
  - Decrease in serum aminotransfereases
  - Increase in serum adiponectin

Belfort R, et al. N Engl J Med 2006; 355: 2297-307

Study Limitations

- Small sample size – only 47 patients
- Short study period – 6 months
- ***Shows promise, but need more info***

Summary

- NAFLD = main cause of elevated liver enzymes among the general population
- Spectrum of disease ranging from benign steatosis to cirrhosis
- Insulin resistance is thought to be the key pathogenic feature
- Often associated with the metabolic syndrome
- Goal of treatment is to improve insulin sensitivity and modify underlying metabolic risk factors

PCOS: Goals

- Identify patients with risks for or with Dx of PCOS
- Assess patients appropriately for PCOS and associated disease states
- Prescribe therapy to treat complaints and prevent sequelae

PCOS: Defined? I

- ACOG and NIH (1990): hyperandrogenism and chronic anovulation excluding other causes
- Stein and Levanthal (1935): association of amenorrhea with polycystic ovaries and variably: hirsutism and/or obesity

PCOS: Epidemiology

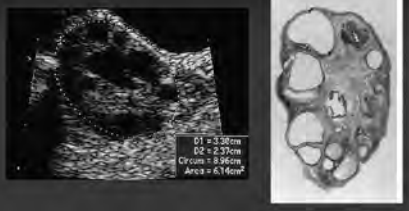
- Prevalence: 4-6% females
  - Probably same world wide
- No difference between blacks and whites
- 75% of women w/ irregularity or infertility

PCOS: Signs and Symptoms

SYMPTOMS	SIGNS
Menstrual irregularity	**Hirsutism, acne**
Infertility	**Obesity**
Hirsutism, acne, etc	Ovarian enlargement
Obesity	Acanthosis nigricans

PCOS: Signs and Symptoms II

PCOS: Imaging and Pathology

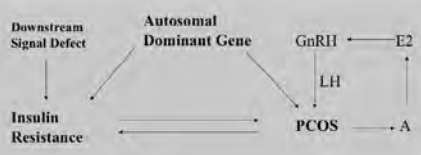


PCOS: Pathopysiology

What we think we know.

- "Vicious cycle"
- Abnormal gonadotropin secretion
  - Excess LH and low, tonic FSH
- Hypersecretion of androgens
  - Disrupts follicle maturation
  - Substrate for peripheral aromatization
- Negative feedback on pituitary
  - Decreased FSH secretion
- Insulin resistance, Elevated insulin levels

PCOS: Current theories of pathopysiology



A=androgens, E2=estradiol

PCOS: Case 1 - Hx

- J.D. 31yof
- Menstrual irregularity,LMP 5 months prior
  - Irregular since menarche
  - Getting longer over time
- Sexually active and uses condoms
- 40lb weight gain over past six months
- Previous U/S w/ ovarian cysts
- ROS: hair growth on her chin and chest
- Meds: HCTZ, Effexor, atenolol

PCOS: Case 1 - PE

- BP 126/96, Weight 248lbs
- Skin: dark hair on chin and chest, moderate to severe acne on face and back
  - no acanthosis nigricans
- Abd-obese, tender RLQ, no R/G, no abd striae
- Pelvic exam – nl ext genitalia no clitoromegaly, norm appearing cervix
- Bimanual: Uterus/adnexa not palpated
- U/S: Normal appearing ovaries

PCOS: Differential Dx

- Androgen secreting tumor
- Exogenous androgens
- Cushing's syndrome
- Nonclassical congenital adrenal hyperplasia
- Acromegaly
- Genetic defect in insulin metabolism
- Primary hypothalamic amenorrhea
- Primary ovarian failure
- Thyroid dz
- Prolactin dz

PCOS: Case 1 Work-up

- Total or free testosterone
- +/- LH and FSH
- Pelvic U/S
- Fasting glucose
- Fasting lipid profile
- (SHBG, Insulin)

PCOS: Case 1 Treatment

- Oligomennorhea
  - OCPs, Progestins, insulin-sensitizing agents
- Hirsutism
  - OCPs, Antiandrogens, ISAs, Eflornithine
  - Mechanical treatments
- Obesity
  - LIFESTYLE MODIFICATIONS
  - Metformin

Case 1: Outcomes

- Laboratory analysis: Nl TSH and prolactin, mild elevation of testosterone, LH:FSH 3:1
- Treatment: Diet and exercise counseling, metformin 850mg bid.
- Patient reported resumption of menses and thereafter lost to f/u

PCOS: Infertility

- WEIGHT LOSS
- Clomiphene citrate 50-100mg QD +/- dexamethasone
- Gonadotropins
- Metformin
- Ovarian Drilling

PCOS: Associated Disorders

- Diabetes
- Hyperlidpidemia (LDL, Triglycerides)
- Obesity
- Hypertension
- CAD?
  - Incr in Risk Factors, but not mortality

PCOS: Associated Disorders

- Endometrial CA
- Ovarian CA?
- +/- Breast CA
- NO increase in Osteoporosis
- Eating disorders
- Psychiatric dz

Pharmacologic Management of Type 2 Diabetes

The following factors and the information shown in Table 1 and Figure 1 should also be taken into account:

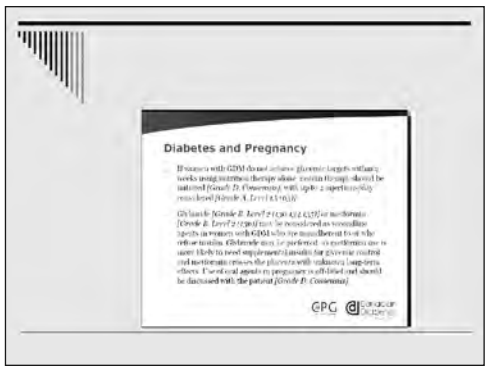
- Metformin should be the initial drug used in both overweight patients *[Grade A, Level 1A (52)]* and nonoverweight patients *[Grade D, Consensus]*.
- Other classes of antihyperglycemic agents, including insulin, should be added to metformin, or used in combination with each other, if glycemic targets are not met, taking into account the information in Figure 1 and Table 1 *[Grade D, Consensus]*.

CPG Canadian Diabetes

Diabetes and Pregnancy

1. Women with type 2 diabetes who are planning a pregnancy or become pregnant should:
   a. Switch from oral antihyperglycemic agents to insulin *[Grade D, Consensus]*. This should preferably be done prepregnancy, except in the setting of PCOS, where metformin can be safely used for ovulation induction *[Grade D, Consensus]*.The safety of metformin beyond ovulation induction in women with type 2 diabetes remains unknown *[Grade D, Consensus]*.
   b. Receive an individualized insulin regimen to achieve glycemic targets, with consideration given to intensive insulin therapy *[Grade A, Level 1 (65)]*.

CPG Canadian Diabetes



Notes

Notes

Wednesday, Nov. 25 – Workshop G-01

10:30 - 11:30 ER "Zebras Run with Horses"

Joe Nemeth MD
Assistant Professor, Emergency Medicine, McGill University;
Attending Physician, Emergency Department,
McGill University Health Center, MUHC

Notes

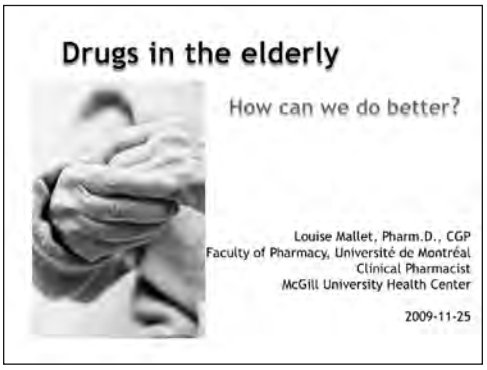
Wednesday, Nov. 25 – Workshop G-02

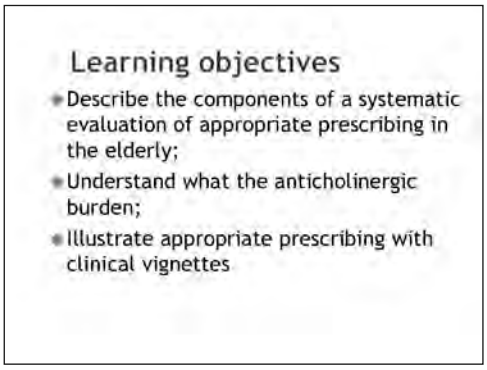
10:30 - 11:30 GER: Drugs in the Elderly

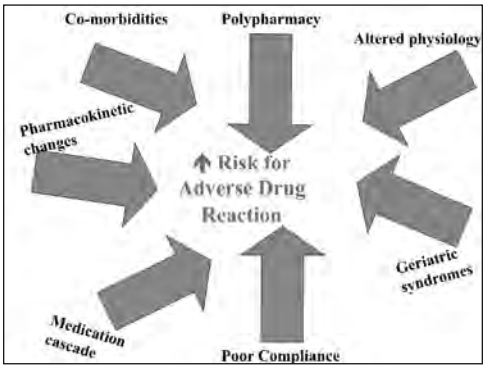
Louise Mallet BSc (Pharm), PharmD, CGP
Professor in Clinical Pharmacy, Faculty of Pharmacy, University of Montréal;
Clinical Pharmacist in Geriatrics, MUHC

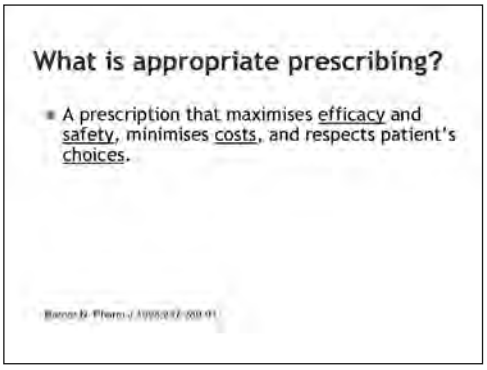
Research interests: Louise Mallet is Clinical Professor of Pharmacy at the University of Montreal. She is also a clinical pharmacist specializing in geriatrics at McGill University Health Center (Royal Victoria Hospital). She is responsible to teach topics in geriatrics in the pharmacotherapy course and has developed an elective course in geriatrics for pharmacy students and pharmacists at the Faculty of pharmacy.

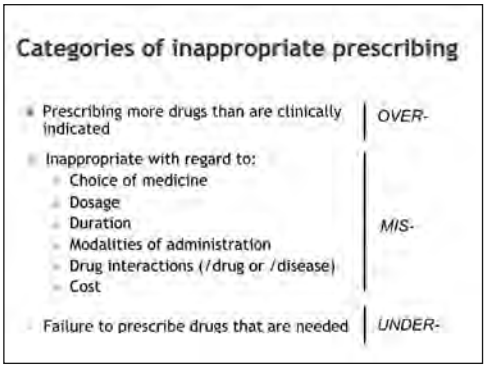
Dr. Mallet is the co-editor of two pharmaceutical textbooks and has authored or co-authored more than two dozen book chapters and close to 150 articles for refereed and professional journals. Dr. Mallet has received research funding to investigate such topics as reducing medication-related falls and training caregivers for the elderly.











How should we review prescribing for an elderly patient?

Some pre-requisites

- Have access to patients' records
  - Past medical Hx, drug Hx, laboratory data, evolution, calculate creatinine clearance, weight
- See the patient/carer !
  - Drug history, compliance, chronology of events for medication history, medication reconciliation
- Communicate with other HCPs
  - Physicians, nurses, physiotherapists, community pharmacists,...
  - Continuity of care

Explicit instruments

- The Beers' criteria
  - Drugs to avoid, risks > benefits
  - Drugs - drugs in certain diseases

Beers et al., Arch Int Med 1997;157:1531 - Arch Int Med 1997;157:1531 and 2003;163:2716-24

Explicit instruments

- The Beers' criteria

Beers 1997	Beers 2003 - additions
Amitriptyline Diazepam, flurazepam,clorazepate, triazolam,... Propoxyphene Ticlopidine, Dipyridamole Indomethacin Loraz.>3 mg, alpraz.>2mg VKA + aspirin / NSAID ...	Amiodarone Fluoxetine Cimetidine Nitrofurantoin Oestrogens ...

There is a role for inappropriate prescribing screening tools in everyday clinical practice.

They should enhance, not replace good clinical judgement.

(Hamilton et al., BMC Geriatrics 2009;9:5)

Implicit instruments

- The Medication Appropriateness Index (MAI)
  - 10 questions per drug

1. Valid indication?
2. Appropriate choice?
3. Correct dose?
4. Modalities of treatment correct?
5. Modalities of treatment practical?
6. Clin. significant drug-drug interactions?
7. Clin. significant drug-disease interactions?
8. Duplication?
9. Appropriate duration?
10. Cost?

Hanlon et al. Am J Med 1996;100:428-37

"Any symptom in an elderly patient should be considered a drug side effect until proved otherwise."

J Gurwitz, M Monane, S Monane, J Avorn
Brown University Long-term Care Quality Letter 1995

Prescription cascade

Drug n° 1
⇒ Side effect interpreted as a new disease
⇒ Addition of drug n° 2
⇒ Side effet interpreted as a new disease
⇒ .....

Rochon et coll. BMJ 1997

11/13/09

"Prescribing Cascade"

- **Common causes of polypharmacy in elderly**
- **Some common examples**
  - Ibuprofen ->HTN->antihypertensive therapy
  - Metoclopromide ->Parkinsonism ->Sinemet
  - Nifedipine -> edema ->furosemide
  - Gabapentin -> edema ->furosemide
  - Ciprofloxacin ->delirium ->risperidone
  - Sudafed ->urinary retention ->alpha blocker
  - Enalapril -> dizziness -> Serc -> falls
  - Bupropion -> insomnia -> Mirtazapine

11/13/09

Mrs AL

90 yo, 52 kg. lives alone. Reports dizziness and falls in past month.

Medications

Ditropan 5 mg 2 x/ j
Synthroid 0.075 mg 1/j
Hydrochlorothiazide 25 mg in morning
Serax 15 mg at bedtime when needed

Lab tests:

TSH	3,4
Creatinine	85 umol/L
Na	135 mmol/L
K	3,0 mmol/L

11/13/09

So What's the Problem?

My Doctor said "Only 1 glass of alcohol a day". I can live with that.

11/13/09 20

Application to drug interactions

Panel 5: Team approach to the prevention of drug interactions (widely generic)

Pharmacist
- Develop a therapeutic relationship with the patient and caregiver to assess attitudes, preferences, and drug compliance
- Document a complete up-to-date drug history, including over-the-counter medications, health supplements, alcohol, and vitamins
- Review medications for actual drug interactions, screen for drug–disease interactions, and for drugs that are metabolised primarily via cytochrome (CYP) isoenzymes
- Detect and document actual drug interactions, in liaison with prescriber and patient, follow up, suggest drugs with a lower risk of interactions according to the patient's drug profile
- Monitor for adverse outcomes from potential drug interactions
- Educate the patient and caregiver on non-prescription drug use, nutritional supplements, and potential drug–food interactions
- Educate members of the health-care team on drug interactions
- Document and report any adverse drug event
- Reconcile active drug lists and pharmaceutical care plan on transition between care settings, to promote continuity of care

Prerequisites
Know your CYPs!
Communicate
Document
Educate
Follow-up

Mallet L et al. Lancet 2007;370:185-91.

Some questions

For every patient

- Could the presenting complain be related to an ADE?
- Are there diseases or symptoms that are undertreated?
- What does the patient think about the medicines prescribed?

For every medicine prescribed

1. Valid indication?
2. Appropriate choice?
3. Correct dose?
4. Modalities of treatment correct?
5. Modalities of treatment practical?
6. Clin. significant drug-drug interactions?
7. Clin. significant drug-disease interactions?
8. Duplication?
9. Appropriate duration?
10. Cost?

When you evaluate prescribing,

never forget to ask (if possible)

the patient's point of view!

Don't anticipate that the patient will disagree with what you want to propose

Spinewine et al., BMJ 2005;331:985-9

BMJ 11 oct 2003

Anticholinergic Burden

- The ***cumulative*** effect of taking ***multiple*** medications with anticholinergic properties
- Factors that may influence ACh burden:
  - Multiple drugs with Ach effects
  - Drug exposure, anticholinergic potency of drugs involved
  - Co-morbid conditions
  - Pharmacokinetic changes
  - Drug interactions
  - Blood-brain barrier integrity
- Physicians can reduce ACH burden by avoiding potentially inappropriate agents, especially when initiating new therapies

Rudd KM, et al. Pharmacotherapy 2005;25:1592-1601

Clinical importance of Ach burden

- Growing body of evidence which indicates measurable and clinically relevant adverse effects in the elderly
- Impact on CNS is documented
- Other potential adverse outcomes may also result in significant disability for seniors
- Patients with dementia and delirium may be at higher risk for negative clinical outcomes.

Ach effects

Dry Mouth	↓ communication, malnutrition, mucosal damage, denture misfit, dental caries, ↑ risk of serious respiratory infection 2° to loss of antimicrobial activity of saliva
Mydriasis and ↓accommodation	Narrow angle glaucoma, increased risk of accidents/falls
Constipation	Fecal impaction
Urinary hesitancy	Urinary retention
Tachycardia	Worsening angina
Decreased sweating	Heat stoke or hyperthermia
CNS	Delirium, dementia, confusion, sedation, agitation

Tune LE. J Clin Psychiatry 2001;62(Suppl 21):11-14

Mrs Robinette

- 85 yo woman, lives alone
- Her daughter visits her once a week. Had 2 falls in past few weeks. Daughther finds her mother different, more confused since her last visit.
- Medical history: Hypertension, hypothyroidism, constipation, chronic pain, history of depression
- Weight: 45 kg
- B.P: 180/70 lying and 130/70 standing
- Pulse 72
- MMSE 24/30

Medications

- Amodipine 5 mg qd
- HCTZ 12.5 mg qd
- Levothyroxine 0.05 mg qd
- Risperidone 0.5 mg at lunch and supper
- Gabapentin 300 mg tid
- Senokot 2 tablets at bedtime
- Acetaminophen 50 mg qid prn pain
- Dimenhydramine 25 mg at bedtime prn

Mrs. Robinette

Your evaluation

Mr. S.

- 85 yo man admitted for falls: now in ER
  - Amitriptyline 100 mg at bedtime
  - Nexium 40 mg 1 x/j
  - Amlodipine 10 mg 1 x/j
  - Lévothyroxine 0.175 mg 1 x/j
  - ECASA 80 mg 1x/j
  - Atorvastatine 20 mg 1x/j
  - Metformine 850 mg 3x/j
  - Nitropatch 0.2mg 12/24: 9h00 on 21h00 off
- Received metoclopramide 10 IV, Gravol 50 IV, Dilaudid 2 mg s.c. during the night

Mr. S.:
How do we start

Which 3 things you are going to do differently in your [illegible]?

Questions

louise.mallet @umontreal.ca

Notes

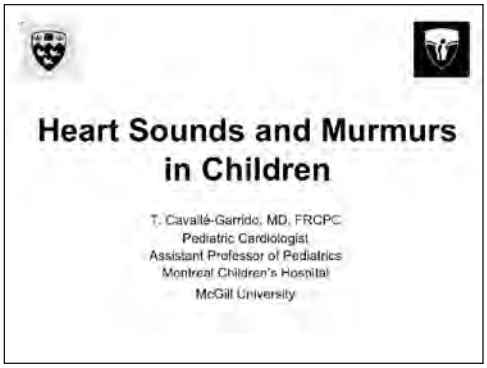
Wednesday, Nov. 25 – Workshop G-03

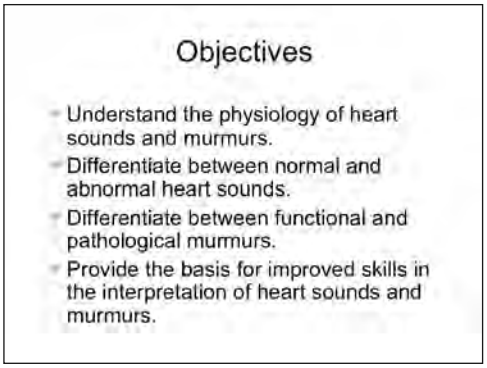
10:30 - 11:30 Peds - Heart Sounds and Murmurs in Children

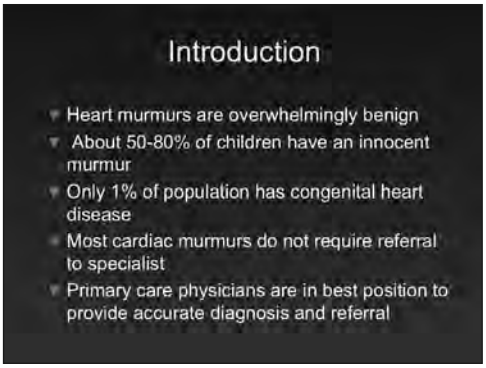
Tiscar Cavalle-Garrido MD

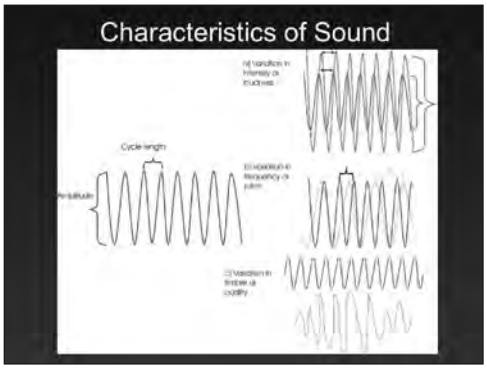
Assistant Professor, Department of. Pediatrics, McGill University;
Staff Physician, Division of Pediatric Cardiology, The Montreal Children's Hospital – MUHC

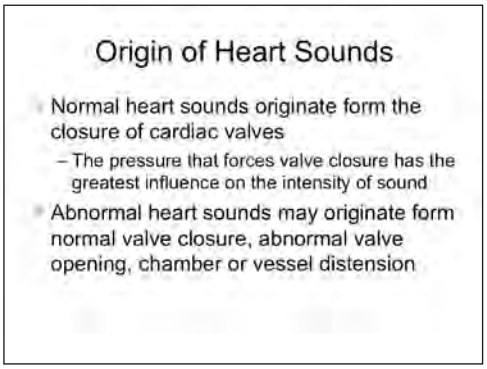
Research Interests: I trained in Pediatrics and Pediatric Cardiology in Spain. Thereafter, I subspecialized in fetal echocardiography at the Hospital for Sick Children in Toronto. For personal reasons I decided to stay in Canada. To be able to practice medicine I re-trained both in Pediatrics and Cardiology at the Hospital for Sick Children, University of Toronto. I worked as a Staff Cardiologist at the Hospital for Sick Children and St. Michael's Hospital in Toronto until I joined the Montreal Children's Hospital in March of 2008.

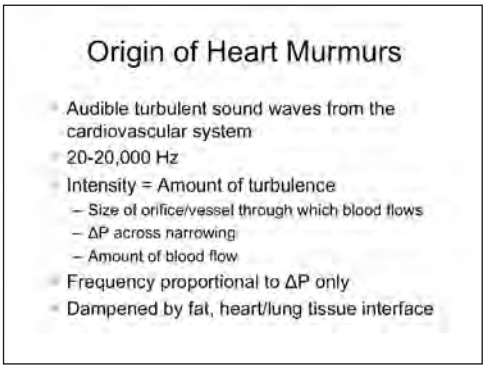


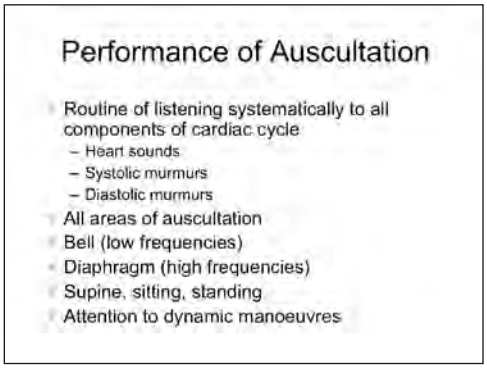


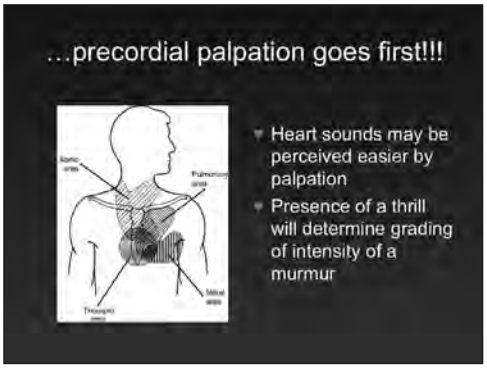


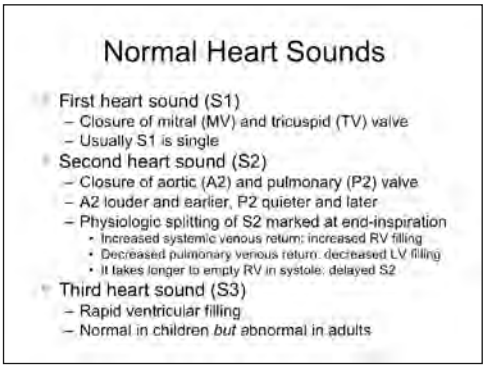


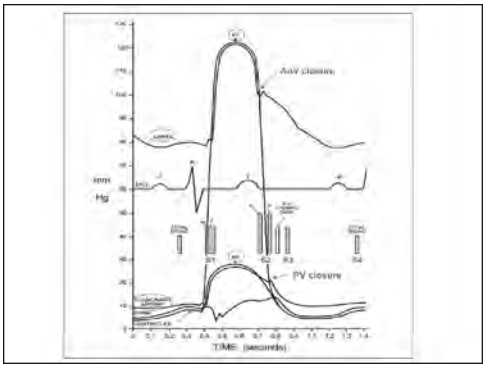








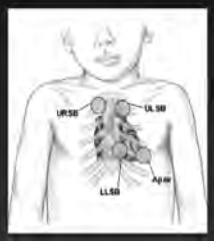




Abnormal Heart Sounds

- Abnormal S2
  - Loud P2: Pulmonary hypertension
  - Widely or fixed split S2
    - RV volume overload
    - Delayed RV conduction
- Fourth heart sound (S4)
  - Distension of stiff ventricles during atrial contraction
  - Indicates diastolic dysfunction

Clicks and Snaps

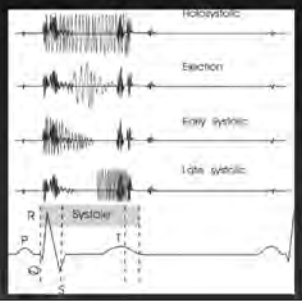


- Early systolic ejection clicks
  - Opening of stenotic semilunar valves
    - Pulmonary valve stenosis
    - Aortic valve stenosis
  - Right after S1
  - "Sail catching wind"
- Mid-systolic clicks
  - MV prolapse
- Opening snaps
  - Opening of stenotic MV / TV
  - Right after S2

Classification of Heart Murmurs

- Intensity
  - Grade 1: Barely audible
  - Grade 2: Audible and constant
  - Grade 3: Loud with no thrill
  - Grade 4: Loud with thrill
  - Grade 5: Heard with stethoscope just touching chest
  - Grade 6: Heard with stethoscope off the chest
- Timing and duration
- Location and radiation
- Configuration
- Pitch and quality

Systolic Murmurs



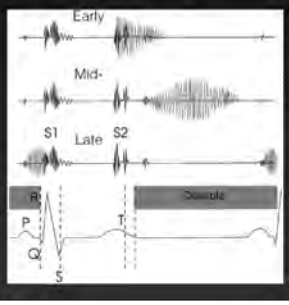
VSD, MR, TR

Outflow obstruction
Innocent murmurs

Tiny VSDs

MR of MV prolapse

Diastolic Murmurs



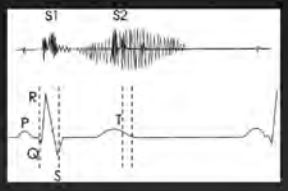
AI, PI

MV or TV stenosis
↑ Flow thru MV/TV

MS or TS
during A systole

ALWAYS
PATHOLOGIC!!!

Continuous Murmurs



PDA
Coronary fistula

Venous hum:
innocent

Innocent Murmurs

(functional, benign, physiological, flow murmurs...)

- Early systolic ejection murmurs (except venous hum)
- Short duration
- Low intensity (grade 1 or 2)
- Louder when supine

Still's Murmur

- Origin unclear
- From infancy to adolescence
- Typically 3-7 yrs
- Early ejection systolic
- Grade 1-3/6
- Low-medium pitch
- Musical, vibratory
- Maximal at LLSB
- Loudest when supine
- Decreases with Valsalva

Differential Diagnosis of Still's Murmur

- Hypertrophic obstructive hypertrophic cardiomyopathy
  - Murmur louder with Valsalva, softer with squatting
  - Suggestive history
- Small ventricular septal defect
  - If closing, VSD may be only heard in early systole
  - In general, VSD murmurs are pansystolic and regurgitant

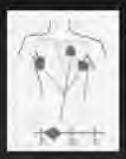
Pulmonary Flow Murmur

- From children to young adults
- Early to mid ejection systolic
- Grade 2-3/6
- Rough
- Maximal at 2-3 ICS at LSB
- Best heard supine
- Augmented with full exhalation
- Decreased upright, with held inspiration
- Exaggerated with decreased AP diameter of chest

Differential Diagnosis of Pulmonary Flow Murmur

- Atrial septal defect
  - Hyperdynamic RV impulse
  - Wide / fixed split S2
  - Mid-diastolic flow rumble
- Pulmonary stenosis
  - Systolic thrill
  - Ejection click
  - Longer, higher pitched murmur
  - Delayed, softer P2

Peripheral Pulmonary Stenosis



- Origin in branch pulmonary arteries
- In neonates and infants < 1 year
- Early or mid ejection systolic
- Rarely just beyond S2
- Grade 1-2/6
- Low to medium pitch
- Maximal in axilla and back
- Changes with HR and SV

Differential Diagnosis of Benign PPS

- Branch pulmonary artery hypoplasia
  - Murmur may be indistinguishable
  - Higher pitched, extending beyond S2
  - Persist beyond first months of life
- Atrial septal defect
  - Murmur of ASD rarely heard in this age group
- Pulmonary stenosis
- Patent ductus arteriosus
  - Usually a continuous murmur

Supraclavicular or Brachiocephalic Systolic Murmur

- Turbulence in carotid arteries
- Children and young adults
- Early to mid ejection systolic
- Abrupt and brief
- Variable intensity
- Low-medium pitched
- Maximal above clavicles, to neck
- Diminished by shoulder hyperextension

Differential Diagnosis of Supraclavicular Flow Murmur

- Valvar aortic stenosis
  - Ejection click
  - Location
  - Not decreased by shoulder hyperextension
- Carotid obstruction
  - Exceedingly rare in pediatric population
  - Higher pitched
  - Extension into diastole

Aortic Systolic Murmur

- Older children and adults
- Short ejection systolic murmur
- Low-medium pitch
- Best heard in aortic area of auscultation
- Secondary to increased cardiac output: fever, hyperthyroidism, anemia, anxiety
- Athletes: due to ↓HR and ↑stroke volume

Differential Diagnosis of Aortic Flow Murmur

- Difficult to distinguish from benign murmur
  - Referral often indicated
- Hypertrophic obstructive hypertrophic cardiomyopathy
  - Murmur louder with Valsalva, softer with squatting
  - Suggestive history
- Fixed subaortic obstruction

Venous Hum

- Children of any age
- Continuous murmur
- Variable intensity
- Variable quality
- Maximal at low anterior part of neck, lateral to SCM muscle
- More frequent in right side
- Originated in the jugular vein
- Best heard sitting-up
- Decreases on lying or compressing jugular vein

Differential Diagnosis of Venous Hum

- Patent ductus arteriosus
  - On left side
  - Louder systolic component
  - No change with maneouvers

Features That Increase the Likelihood of Cardiac Pathology

- Cardiovascular symptoms
- Family history (e.g., Marfan syndrome or sudden death in young family members
- Malformation syndrome (e.g., trisomy 21)
- Increased precordial activity
- Decreased femoral pulses
- Abnormal second heart sound
- Clicks
- Loud or harsh murmur
- Increased intensity of murmur when patient stands

Murmurs in the Asymptomatic Newborn

- Few hours of life
  - Closing PDA or tricuspid regurgitation
- Systolic murmur after S1 *(may be innocent)*
  - Aortic or pulmonary stenosis
  - PPS
- Systolic murmur obscuring S1 *(always pathologic)*
  - VSD
    - As PVR drops: louder
    - If PVR does not drop: no murmur but loud P2
  - AVV regurgitation

McCrindle BW, et al. Factors prompting referral for cardiology evaluation of heart murmurs in children. Arch Pediatr Adolesc Med 1995;149:1277-9

- General providers are generally accurate in their assessment of likelihood of disease
- Only a minority of murmur patients are referred for cardiology consultation
- "Newly heard" murmurs due to lack of continuity of primary care resulted in inappropriate referrals
- Parental anxiety is the most common non-clinical reason for referral
- Additional testing is unnecessary and cost-ineffective
- Education of providers and parents needed to ensure that evaluation of heart murmurs is cost-effective both in detecting disease and reassuring families

Take Home Points

- The majority of patients with heart murmurs have a normal heart
- Innocent murmurs can be diagnosed with an adequate physical examination
- Additional testing is usually not necessary
- A detailed history and physical exam is required to put murmur in context
- Cardiology referral if red flags present

Recommended Reading & Links

- Menash V. Heart murmurs. Pediatr Rev 2007;28(4):e19-22
- Silberbach M, Hannon D. Presentation of congenital heart disease in the neonate and young infant. Pediatr Rev 2007;28:123-131
- Pelech A. The physiology of cardiac auscultation. Pediatr Clin N Am 2004;1515-1535
- Saunders n. Innocent heart murmurs in children. Can Fam Physician 1995;41:1507-1512
- McConnell M, Adkins S, Hannon D. Heart Murmurs in Pediatric Patients: When Do You Refer? Am Fam Physician 1999;60:558-65
- McCrindle BW, et al. Factors prompting referral for cardiology evaluation of heart murmurs in children [Letter]. Arch Pediatr Adolesc Med 1995;149:1277-9.
- http://pedsinreview.aappublications.org/cgi/content/full/28/4/e19/DC1
- http://www.wilkes.med.ucla.edu/intro.html
- http://depts.washington.edu/physdx/heart/demo.html
- http://www.dundee.ac.uk/medther/Cardiology/hsmur.html

Notes

10:30 - 11:30 Hands On – Steroid Injections

Michael Stein, MDCM, FRCPC

Assistant Professor, Department of Rheumatology,
Faculty of Medicine, McGill University

Hands on - Steroid Injections

Michael Stein MDCM, FRCP(C)
November 24 2008

McGill

Disclosure

- Abbott Pharmaceuticals
- Schering Plough
- Roche
- Bristol-Myers Squibb

McGill

Agenda

- Materials
- Corticosteroids
  - Dosages and Equivalency
- Contraindications and Complications
- Practical Approach using Patient Partner
  - Joint injection
  - Tendon and Bursa Injection

McGill

Materials

- Alcohol wipes
- Povidone-iodine wipes
- Sterile and nonsterile gloves
- Sterile drapes
- Needles and syringes
- Local anesthetic
- Corticosteroid preparation
- Laboratory tubes for culture or other studies (aspiration)
- Hemostat
- Adhesive bandage

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Corticosteroid Equivalency

Corticosteroid	Anti-inflammatory	Mineralocorticoid	Half-life
Prednisone	20 mg	100 mg	12-36 hr
Betamethasone	2 mg	No effect	36-72 hr
Methylprednisolone	16 mg	No effect	12-36 hr
Hydrocortisone	80 mg	80 mg	8-12 hr
Dexamethasone	2 mg	No effect	36-72 hr
triamcinolone acetonide	16 mg	No effect	18-36 hr
Triamcinolone hexacetonide	8 mg	No effect	18-36 hr

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Containdications

Absolute

- Local cellulitis
- Septic arthritis
- Acute fracture
- Bacteremia
- Joint prosthesis
- Achilles tendonopathy
- Anaphylaxis history

Relative

- No relief with previous injection x 2
- Coagulopathy
- Anticoagulation
- Osteoporosis
- Diabetes
- Psoriatic plaque over possible injection site

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Complications of Joint Injections

- Septic arthritis
- Hemarthrosis
- Post-injection synovitis
- Damage to internal structures
- Soft-tissue atrophy and depigmentation
- Systemic effects
- Pain

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Why can't I aspirate fluid?.....

- No fluid in joint/bursa
- Tissue obstructing needle lumen
- Needle is not in joint
- Tense muscles around joint

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Steroid dose for Joint Injections

JOINT	Methylprednisolone	Triamcinolone hexacetamide
Shoulder	40 - 80 mg	20 - 40 mg
Elbow	20 - 40 mg	10 - 20 mg
Wrist	20 - 40 mg	10 - 20 mg
Finger joint	10 mg	5 mg
Knee	40 - 80 mg	20 - 40 mg
Ankle	20 - 40 mg	10 - 20 mg
Metatarsal joints	10 mg	5 mg

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Steroid dose for Bursa/Tendon Injection

Tissue	Methylprednisolone	Triamcinolone hexacetamide
Rotator cuff	40 mg	20 mg
Bicep tendon	20 mg	10 mg
Olecranon bursa	10 mg	5 mg
DeQuervains	10 mg	5 mg
Flexor tendon	10 mg	5 mg
Trochanteric Bursa	20 -40 mg	10 -20 mg
Pre-patellar bursa	10 mg	5 mg
Plantar Fascia	20 mg	10 mg

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Knee

- Mix 1ml lidocaine with 40 mg methylprednisolone
- 1½ x 22 gauge needle
- Approach:
  - supra-patella bursa
  - medial or lateral sub-patellar
  - Anterior with flexed knee

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Shoulder

- Gleno-humeral joint injection
- 1½ x 22 gauge needle
  - Anterior: 1 cm below and lateral to coracoid. Aim posteriorly
  - Posterior: posterior angle of acromion. 1 cm lateral and posterior. Aim for coracoid
  - Sub-acromial bursa: posterior angle of acromion. 1 cm latereral. Aim horizontally below acromion

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Shoulder

- Acromio-clavicular joint injection
  - Shrug shoulder to identify joint
  - Mix .2 ml lidocaine and 10 mg methylprednisolone with 1½ x 25 gauge needle
- Bicep tendon
  - Identify tendon by ER and IR shoulder
  - Mix .5 ml lidocaine and 20 mg methylprednisolone with 1½ x 25 gauge needle

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Ankle

- Mix 0.5 ml lidocaine with 20 mg methylprednisolone with 1½ x 22 gauge needle
- Identify space between med malleolus and ant. Tibialis tendon
- Aim towards lateral malleolus

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Olecranon bursa

- Extend elbow
- Aspirate/inject from superior/apical pole with .3 lidocaine and 10 mg methylprednisolone with a 20 gauge needle

McGill

Greater Trochanteric Bursa

- Mix 1 ml lidocaine with 40 mg methylprednisolone using 1½ x 22 gauge needle
- Inject bursa in circumferential fashion

McGill

Plantar Fascia

- Mix 1ml lidocaine with 20 mg methylprednisolone using 1½ x 25 gauge needle
- Medial aspect of heel
- Inject in circumferential fashion in mid heel

McGill

Trigger Finger

- Mix 0.1 ml lidocaine with 10 mg methylprednisolone using 1 x 30 gauge needle
- Inject base of finger, 45° towards tip
- Pierce until feel "scratchy" tendon, withdraw and inject
- Splint 24 hr

McGill

References

1. Fam A et al. Musculoskeletal Examination and Joint Injections Techniques. 1st edition. Moseby. 2005.
2. http://www.emedicine.com/
3. Rheumatology Examination and Injection Techniques. Ehrlich. JAMA.1999; 282: 697-698.

McGill

Thank you

McGill

Wednesday, Nov. 25 – Workshop G-05

10:30 - 11:30 Diabetes

Tina Kader MD, FRCPC, CDE
Assistant Professor , Department of Medicine, McGill University;
Certified Diabetes Educator

Notes

Wednesday, Nov. 25 – Workshop G-06

10:30 - 11:30 Pain Management

Mary-Ann Fitzcharles MD
Associate Professor, Division of Rheumatology, McGill University;
Rheumatologist, The Montreal General Hospital – MUHC

Notes

10:30 - 11:30 Evidence Based Medicine in Real Clinics, No Ivory Towers

Martin Dawes MBBS, MD, FRCGP
Professor and Chair, Department of Family Medicine, McGill University

Evidence Based Medicine in Real Clinics, No Ivory Towers

Dr Martin Dawes

Disclosures

- Works a bit for pharma as a consultant on trial design (two ongoing studies – one just funded by CIHR on knowledge translation – the other at the development stage and can't disclose).

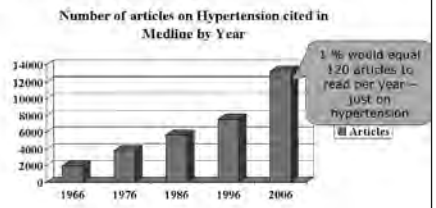
Objectives

- Learn about your variation in practice (interactive)
- Learn about places to get information (interactive)
- Learn that appraisal is important – but that you don't have time
- Learn how to do it for real using trusted sources of low bias (interactive)

Increasing Medical Knowledge

- 27Kg of Guidelines
- New scientific papers per day
  - 3,000
- Medline New articles
  - 1,000
- Randomised controlled trials
  - 46

Increasing Knowledge



Are you up to date?

- Child with high fever but low risk – why give an antipyretic? Harnden 2006 BMJ
- Febrile convulsions – no evidence
- Reduces temperature –
  - Why do you want to do that? – no evidence
  - Ibuprofen better than acetaminophen – but so what
- Do give based on distress
- 50% of parents may incorrectly dose

Pull technology

- Searching databases
- Medline

Primary Literature Databases

- Mostly non-validated "raw" research
  - Medline
  - UpToDate: textbook with variable transparency of LOE
  - (Thanks Fred Tudiver)

How many questions do you need to ask?

- Before you search, ask:
  - Is it an answerable question (PICO)?
  - What's the best place you'll find the answer?
- Avoid browsing: check specific sources
- Go for best level of evidence

Asking (answerable) questions

- Background questions:
  - What's the latest on fever in children?
- Foreground questions:
  - Specific, managing patient with a problem
    - (P) patient and/or problem
    - (I) intervention
    - (C) comparison
    - (O) outcomes.
  - So – lets go to Medline
  - 18 million articles

Why PICO Medline?

"Fever and children" n= 26,678

- P Fever and children and
- I acetaminophen and
- C ibuprofen and
- O seizure or fit

- n=11

Results


Arch Pediatr Adolesc Med. 1999 Jun;149(6):632-7 Links

Antipyretic efficacy of ibuprofen and acetaminophen in children with febrile seizures.

Department of Pediatrics, University Hospital, Rotterdam, The Netherlands.

OBJECTIVE: To compare the antipyretic efficacy of ibuprofen syrup (5 mg/kg per dose) and acetaminophen syrup (10 mg/kg per dose) in children with a history of febrile seizures. DESIGN: Randomized, multicenter, double-blind, single-dose trial. SETTING: Two outpatient departments of a university-affiliated teaching hospital. PATIENTS: Seventy outpatients (mean age, 2.1 years; range, 10 months to 4 years) who had visited the hospital because of a febrile seizure were randomized to treatment in a temperature of 38.5 degrees C or higher. INTERVENTIONS: Study medication was given every 6 hours for 1 to 3 days. Rectal temperatures were recorded at 0, 2, 4, 6, 12, and 24 hours after the first dose. MAIN OUTCOME MEASURES: The temperature 4 hours after the first dose, the mean temperature during treatment, and the highest temperature during treatment were evaluated. Analysis of covariance corrected for the initial temperature, age, weight, and cause of the fever. RESULTS: Ibuprofen lowered the initial temperature from 39.1 degrees C to a mean temperature of 37.7 degrees C during treatment; acetaminophen covered the initial temperature from 39.2 degrees C to 38.0 degrees C. Ibuprofen reduced fever 0.35 degree C more than did acetaminophen at 4 hours (95% confidence interval [CI], -0.58 to -0.02). The mean temperature was 0.26 degree C lower during ibuprofen treatment (95% CI, -0.59 to 0.07); the highest temperature was 0.30 degree C lower (95% CI, -0.13 to 0.73). In 22 patients, a second fever was treated with the opposite medication than the first. In the cross-over analysis, the respective differences were 0.66 degree C (95% CI, -1.29 to -0.04), 0.40 degree C (95% CI, -0.83 to 0.03), and 0.36 degree C (95% CI, -0.81 to 0.09) in favor of ibuprofen. CONCLUSIONS: Ibuprofen and acetaminophen are effective antipyretic agents in children with a history of febrile seizures. Ibuprofen yielded significantly greater fever reduction than did acetaminophen 4 hours after the first dose. Research is needed on the value of antipyretic agents for the prevention of febrile seizure recurrence.

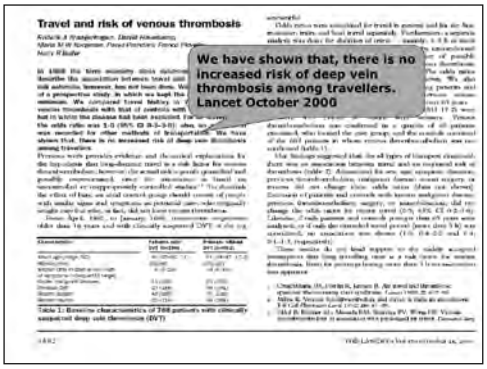
Push technology

- POEMS
- BMJ updates
- ....

Take Nothing For Granted

Critically Appraise

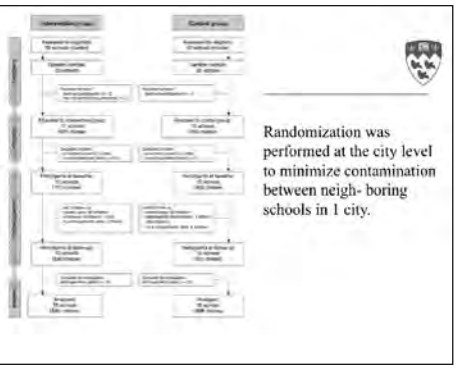
- Deafness
- Headache
- Neuralgia
- Cures all aches and pains



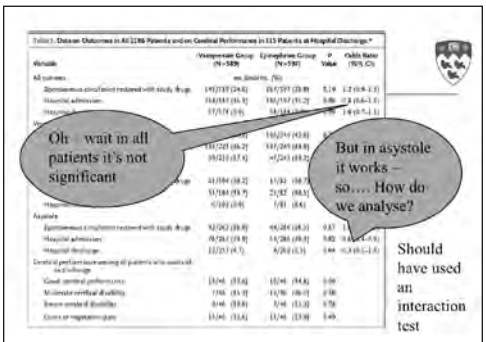
6 months later

Interpretation We conclude that symptomless DVT might occur in up to 10% of long-haul airline travellers. Wearing of elastic compression stockings during long-haul air travel is associated with a reduction in symptomless DVT.

Lancet 2001; **357:** 1485–89
See Commentary page 1461



Randomization was performed at the city level to minimize contamination between neigh- boring schools in 1 city.



Does the way the trial is performed matter?

- No Randomisation
  - Overestimates effectiveness by 20%
- No Allocation concealment
  - Overestimates effectiveness by 30%
- No Blinding
  - Overestimates effectiveness by 15%

Juni P, Altman DG, Egger M. Systematic reviews in health care: Assessing the quality of controlled clinical trials. *BMJ* 2001;323(7303): 42-6.

What does this show?

- Trial design is more important than anything else
- Poor trial design often overestimates the effect
- To interpret research we should be aware of the weaknesses of a trial
- Critical appraisal is necessary

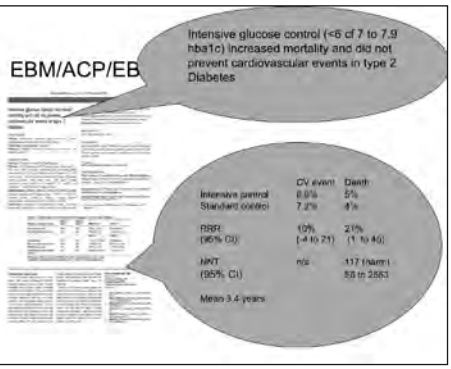
Evidence appraisal

- Time consuming
- Requires practice
- Can sometimes be difficult to see the wood for the trees
- Boring
- Look at pre-appraised evidence

Secondary Literature Databases

- Prevalidated, usually concise
  - T.R.I.P.
  - ACP Journal Club
  - JFP POEMS
  - **Essential Evidence +**
  - Evidence Based group of journals/periodicals
  - Cochrane Database of Systematic Reviews
  - D.A.R.E.
  - BMJ Updates
  - FPIN Clinical Inquiries, etc
  - National Guideline Clearinghouse
  - Bandolier
  - (Thanks Fred Tudiver)

EBM/ACP/EB



Decision- Where to search?

- Appraised
- Guideline?
- Clinical calculator
- Patient information resources
- Medline (never)
- Google (hardly ever)

Validity for Tx

- More than one study- Systematic reviews
- RCT
- All the rest over emphasise the effect
- Cohort
- Case control
- Case series
- Case reports

Real Life

- Is this important?
- Topical aqueous 2% lignocaine eardrops reduced ear pain in children with acute otitis media
- Review: infection rates do not differ for wounds cleansed with water or saline
- Review: many adults still have pain and subjective instability at 1 year after acute lateral ankle sprain
- Telmisartan and ramipril were equivalent, but their combination increased adverse events in vascular disease or diabetes
- Review: symptom-based action plans reduce acute care visits more than peak flow-based plans in children with asthma
- Review: delayed or immediate prescriptions of antibiotics have similar clinical outcomes in respiratory infections (the former results in 30% less AB's)

Knee Pain

A. Do intra-articular corticosteroids or physical aids improve symptoms of OA of the knee?

Trade offs

- The more complete the evidence the less up to date it is
- Guidelines – maybe 3 years out of date (not CHEP)
- Systematic review – maybe 2 years out of date
- Appraised evidence – 1 year
- Pre-appraised - now

- antibiotics and otitis media and pain **TRIP site**
- CBT and schizophrenia **EBM site**

Why are you searching?

- Confirm what you are doing works?
- Refresh your memory?
- Learn something new?

- Your searches are susceptible to bias.
- The expert at the CME talk is susceptible to bias

No one knows how to do this

- Is your practice different from your neighbours?
- Is your practice changing over time
- What are the 2 most important things you need to know today?
- Use an electronic medical record so you can see what you are doing

Last word

- Look things up
- Ask questions
- Never take anything for granted – from anyone

Notes

Wednesday, Nov. 25 – Workshop G-08

10:30 - 11:30 Travel Medicine, Malaria and other Diseases

Dominique Tessier MD, CCFP, FCFP
Family Physician, Clinique médicale du Quartier Latin;
Chargée d'enseignement clinique, Université de Montréal;
Family physician, Post-exposure prophylaxis clinic,
Hôpital Saint-Luc du CHUM

Research interests: Dominique Tessier graduated in medicine from the University of Montréal in 1981 and is certified and Fellow of the College of Family Physicians of Canada. She is a Past-President of the College of Family Physicians of Canada.

Her current practice includes providing services aimed at reducing travel-health problems to corporations and individuals across Canada. She is a family physician and Clinical Instructor at the Family Medicine Department of U. of Montréal. An important proportion of her practice is devoted to infectious diseases, including HIV/AIDS care. Her additional areas of interest include diversity and equity, women's health, violence against women and education.

She is committed to educating and expanding public awareness on prevention and care of problems related to immunizations and Travel Medicine. With Dr Martin Brizard, she recently launched Bleu, a company providing training and services to support Health care professionals in their practice. She loves to travel!

Notes

Wednesday, Nov. 25 – Satellite Symposium

11:30 - 12:15 Satellite Symposium

Chair • **Peter Rohan**

New Horizons in Fibromyalgia: Bringing Hope Through Better Patient Care

Martin Cohen MD, FRCPC

Supported through an unrestricted educational grant from Pfizer

Notes

Notes

Wednesday, Nov. 25 – Afternoon Plenary

13:00 - 13:30 David J.G. Tector Memorial Lecture

Susie Tector MDCM, CCFP-EM
Attending physician, Emergency Department, Montfort Hospital (Ottawa)

Research interests: Susie Tector grew up in the beautiful Eastern Townships of Quebec. She attended Mount Allison University in New Brunswick for her undergraduate degree and McGill University in Montreal for medical school. She then did her residency in family medicine and emergency medicine at Queens University in Kingston, Ontario. She currently lives in Ottawa where she works in a community hospital emergency department and part time as a coroner for the Province of Ontario. For the past four years, she has spent 3 - 6 months per year overseas with the the medical-humanitarian organization Medecins Sans Frontieres - MSF (Doctors Without Borders). She has worked in Darfur (Sudan), Chad, the Democratic Republic of Congo, and most recently in Pakistan. She feels privileged to be able to do this type of work, and is looking forward to her next mission with MSF.

Notes

Wednesday, Nov. 25 – Afternoon Plenary

13:30 - 14:00 Pandemic Update

Brian J. Ward MSc, MDCM, DTM&H
Associate Professor, Department of Medicine, Division of Experimental Medicine, McGill University;
Associate Professor, Centre for the Study of Host Resistance, Montreal General Hospital

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Research interests: Dr Ward received medical and research training at McGill, Oxford (Rhodes Scholar 1977), Johns Hopkins and the University of London. A past Chair of the McGill Infectious Diseases Division, he is currently Associate Director of the Research Institute of the McGill University Health Center (Fundamental Science). He is also Co-director of the McGill Vaccine Evaluation Center, Associate Director of the Center for Tropical Diseases and Director of the National Reference Center for Parasitology. He has served or continues to serve on a number of national and international advisory committees that deal with international health and vaccine use. His current research interests include retinoid-virus interactions, vaccine development and evaluation, novel anti-parasitic strategies, new diagnostic tools and factors that influence HIV transmission. He has authored >125 research articles, book chapters and reviews. This work has been supported by a range of public and private institutions as well as industry.

Notes

14:00 - 14:30 Breast Cancer Detection

John R. Keyserlingk MD, FACS
Medical Director, Surgical Oncology, Ville Marie Medical Center

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Research Interests: Dr. John R. Keyserlingk is a Fellow of a number of societies, including the Royal College of Physicians and Surgeons of Canada, the American College of Surgeons, the American Society of Head and Neck Surgeons and the American Society of Clinical Oncology. He specialized in both General Surgery and Otolaryngology, and completed a Fellowship in Surgical Oncology at the Royal Marsden Hospital in London. He is an Assistant Professor of Surgery at McGill University and a member of the Department of Surgery at the Université de Montréal. He is a staff surgeon in the Division of General Surgery, Surgical Oncology and Otolaryngology at St.Mary's Hospital, the Department of Surgery at the Sacré Coeur Hospital, the Division of General Surgery at the Montreal General Hospital and the Department of Otolaryngology at the Royal Victoria Hospital. He is currently the Medical Director of the Ville Marie Medical and Women's Health Center which includes the Ville Marie Multidisciplinary Breast Cancer and the Ville Marie Oncology Center.

RECENT CONCEPTS REGARDING BREAST CANCER PREVENTION, DETECTION & MANAGEMENT

60th Annual Refresher Course
for Family Physicians McGill University
November 23 to 25, 2009
Hilton Montréal Bonaventure

J. R Keyserlingk

Surgical Oncologist

Ville Marie Multidisciplinary Breast and Oncology Center

1. INTRODUCTION:

Breast cancer prevention, early detection and optimal management have all contributed to improve survival, which is particularly pertinent when dealing with the currently increasing number of new breast cancer cases.

2. PREVENTION:

Prior prevention trails have already documented the ability of SERMS (Tamoxifen & Raloxifen), and soon Aromatase Inhibitors (Aromasin), to decrease the incidence of breast cancer in higher risk patients. Structured Fitness Programs and Risk Assessment expertise are already integral components of prevention strategies and being integrated into contemporary Breast Centers.

2.1 RISK ASSESSEMENT:

Breast Centers are now held accountable to identify and provide special guidance and care for patients with increased risk for breast cancer. As the ability to detect patients at higher risk for cancer has increased in recent years, so too has the need for options to manage higher cancer risk. Potential advantages of incorporating risk assessment into the contemporary Breast Centers include genetic expertise, offering a family approach to risk management and ensuring that appropriate targeted management is seamlessly accessible to moderate and high risk patients. The success of the cancer risk assessment process is largely based on a good level of coordination, comprehension and collaboration between genetic, oncology, radiology and primary care services to ensure an effective risk assessment where at-risk patients are identified and proper health management is offered. Optimal care means providing patients with information and opportunities to benefit from appropriate risk evaluation approaches, possibly including genetic testing, as well as risk reduction strategies such as chemoprevention and prophylactic surgery.

Risk evaluation is based on two steps:

- A comprehensive Risk Questionnaire completed by the patient and returned by mail (copy available from the Ville Marie Risk Assessment Clinic at www.villemariemed.com).

- Evaluation of breast cancer risk by submitting the acquired information in #1 to different scales and software (Gail Risk, BRCAPRO, Manchester, Risk Apps etc) to best determine actual risk and recommend if genetic testing and /or preventive strategies are indicated, along with the pertinence of new imaging technologies such as Full Filed Digital Mammography & CAD, 3-4D Ultrasound and MRI .

Decisions regarding all steps in risk assessment process depend on the extended interdisciplinary team, which includes physicians, nursing, genetic counselors, imaging technicians, clinic coordinators, legal and ethics experts, as well as research and support staff.

2.2 FITNESS AND WELLNESS:

Emerging data suggests that well structured fitness programs can help prevent breast cancer, thus the current trend towards integrating Fitness & Wellness Centers into Breast Centers. These Centers are ideally managed by kinesiologists, physical educators and experts in lifestyle programming, including Yoga and nutrition. This team is usually mandated to train, instruct and advise women regarding various health-related techniques and protocols they may consider incorporating into their everyday routine. Participants and patients are motivated to embrace an optimally active and fulfilling lifestyle that could alleviate various women's health related disorders or limitations. In addition to impacting on the incidence of breast cancer, the physical benefits of exercise for breast cancer survivors include: improved physical fitness and muscular strength; enhanced immune system function; improved physical functioning; weight management and physical appearance benefits.

Physical activity programs improve emotional health including: self-esteem enhancement; quality of life and tasks of daily living improvements; enhanced mood states; improved body image; the development of perceptions of physical competence; regulatory strategies for coping with stress and positive psychological growth. Combined, exercise and social support have been identified as ways to help manage psychological distress associated with breast cancer.

To monitor the effectiveness of some of these new proactive protocols, all pertinent patient information should be entered into the e-chart for the physician and patient to follow. The accumulated data will continue to test exercise strategies that appear to prevent and help manage breast cancer. Such on-going trials are jointly and prospectively evaluated by research staff. Convenient access for those new and monitored patients entered onto clinical trails will lead to the development of many new and improved programs.

Reading Material:

Warburton DE, et al. Health benefits of physical exercise: the evidence. CMAJ 2006; 174: 801-9

3. DETECTION:

Much discussion regarding breast cancer screening is reemerging, particularly the role in patients between 40 and 50, as evidenced in the recent AETMIS Quebec report m dated 2009-10-09 and available on their web page.

Continued advances in multi-imaging imaging technologies, such as **Digital Mammography, 3D4D Ultrasound, Breast MRI, PET/CT** are having a significant impact on promoting earlier and more reliable detection and an improved appreciation of the actual tumor burden.

3.1 FULL FIELD DIGITAL MAMMOGRAPHY (FFDM):

Dr Kopans, a major contributor to breast imaging, suggested in 2003 that Digital Mammography already constituted the most significant advancement in breast imaging over the prior 3 decades. His prediction was borne out in 2005 by the large multi-center **DIMIST STUDY**, funded by the **American Cancer Society** confirming the advantages of Full Field Digital Mammography over analogue (film) mammography, including significantly higher sensitivity rates (increased ability to detect early breast cancer) in the under 50 years of age group (a 27% increase), also in the pre and peri-menopausal patients (20% increase) and in patients with very or partially dense breasts (15% increase). The accuracy of Digital Mammography was also significantly higher than that of film mammography in these three groups (difference in the area under the curve of 0.15; 0.11 and 0.15 respectively). These improved Digital Mammography detection rates, over the old film mammography, produced by first generation units and readers with limited experience, obviously carry over beyond the immediate borders of above-described groups that already constitute a significant proportion of patients undergoing imaging. Of additional interest was that analogue (film) mammography only picked up half of the breast cancers in these groups during a 455 day follow-up during the DMIST study. While film mammography still constitutes a valid tool in breast cancer detection, Digital mammography, particularly when digitally combined with **Computer Assisted Detection (CAD)**, can now be considered the golden standard, particularly for all those patients described above. In addition to this improved detection, the **DEMIST** report cited a number of additional advantages associated with **Digital Mammography** that will result in its rapid installation (in the US, over 90% of newly acquired mammography units are digital). These advantages include consistently optimal images, less recalls, less radiation for the patient, image sharing for multiple reading and reduced image loss. A recent and significantly less expensive hybrid called Computerized Radiology (CR) has appeared. This technology simply scans mammography films to digitalize them and should be recognized a totally different process and not included in the DMIST study.

Reading Material:

Pisano E, et al: Diagnostic Performance of Digital versus Film Mammography for Breast Cancer Screening - The Results of the ACRIN Trial (DMIST). NEJM, pp1773-83. October 27, 2005

Juliette The, and Kathy

Schilling; Detection of Breast Cancer with Full-Field Digital Mammography and Computer-Aided Detection; American Journal of Roentgenology;Volume 192, Issue 2;Jan, 2009.

3.2 3D4D ULTRASOUND:

Ultrasound has greatly contributed to the promotion of early breast cancer detection. The latest 3D4D US technology produces images that can provide a most detailed view of any breast abnormality. With the ability to produce real-time high-quality complex images, ultrasound is now used very frequently up-front to determine the etiology of any palpable or mammography abnormality, and also to conciliate any clinical, mammography or MRI image variations. In addition ultrasound guidance has now become an essential technique for optimizing most breast core or vacuum biopsies. The smallest abnormalities now become sufficiently clear and the last generation units can provide the breast experts with the ability to perform immediate and very precise 4D tissue harvesting on the tiniest lesions. This in turn ensures the best possible advance notice of a potential abnormality that can then translate into much easier treatment and much improved survival.

High frequency screening-monitoring Ultrasound is now being used alongside mammography. As published in the May 14th , 2008 issue of the Journal of American Medical Association, the ACRIN study, which clearly demonstrated that a single screening ultrasound examination added to a screening mammogram, resulted in improved detection of breast cancer compared with mammography alone particularly among women at increased risk of breast cancer and have dense breast tissue. Breast Ultrasound is becoming an integral part of the first level evaluation.

3.3 MRI:

Breast MRI is now an integral breast imaging modality based on accumulated and convincing data from numerous clinical trials done in the mid to late 1990s. Despite substantial differences in patient population and MRI technique, all reported significantly higher sensitivity for MRI compared to both mammography or any of the other modalities used. First published studies screening over 2000 unaffected patients, aged 25 to 70 with an estimated 15% risk of breast cancers (19% proven gene carriers) reported that 80% of the tumors were detected by MRI versus 33% by conventional film mammography, and tumors picked up by MRI were smaller than those detected by film mammography. Five subsequent trials in high risk patients produced similar results, with MRI sensitivity ranging from 71% to 100% versus 16% to 40% for film mammography. These and other subsequent trials led to the following current list of indications for breast MRI:

- to help digital mammography and high resolution ultrasound in monitoring high risk patients or patients with breast implants;
- to better evaluate the local extent and staging of established breast cancer prior to surgery or to help assess residual tumor after surgery;
- to monitor the efficacy of neo-adjuvant (prior to surgery) treatments;
- to assess local recurrence
- to seek out an occult breast primary when associated with involved axillary lymph nodes.

The American Cancer Society's recent recommendations for regular monitoring using breast MRI are listed as follows:

a) Gene carriers and their first-degree relatives and for all patients with a 20% lifetime risk as defined by the risk tools utilized by established Risk Assessment Clinics.

b) Prior chest radiation between ages 20 and 30

c) Patients included in the following categories should be considered for MRI screening based on individual risk factors: Lifetime risks over 15%; lobular carcinoma in situ; atypical lobular; ductal hyperplasia; heterogeneously dense breasts on mammography and women with a personal history of breast cancer, including LCIS.

In a recent study looking for additional occult tumors, MRI detected 30 clinically and mammography occult small node-negative and curable tumors in the contra lateral breast in 969 patients with an established breast cancer MRI detection was not marred by breast density. The authors thus recommend MRI prior to undertaking surgery or neo-adjuvant therapy. They also recommended that breast MRI should only be done in Centers with dedicated breast MRI expertise and capable of doing MRI-guided biopsy when indicated. The authors concluded that the control of breast cancer for the foreseeable future will depend mostly on early detection, careful diagnostic evaluation and therapy. They also suggested that ultrasonography, MRI, and digital mammography will improve the outcome when they are used as a substitute for, or an adjunct to, conventional film mammography for women in whom conventional film mammography screening has not been useful. While proposing MRI, the editor also reiterates that Digital Mammography has recently been shown to be a more effective imaging tool in younger women. Since conventional film mammography does not identify all breast cancers, newer imaging tools such as MRI and Digital Mammography can fill this void.

READING MATERIAL:

1. the American Cancer Society Guidelines for Breast Screening with MRI as an adjunct to mammography; Ca Cancer J Clin 2007;57;75-89.

2. MRI Evaluation of the Contraletral Breast in Women with recently diagnosed breast cancer; N Engl J Med, 2007;356;13:1295-1303

4. PET/CT:

PET/CT combines, as does MRI, both structural (CT component) and functional information (PET component). Its current role in breast cancer is to help define complete tumor burden in higher risk patients, or as a better alternative to our current metastatic workup that traditionally is limited to a Chest X-Ray, and bone scan and an abdominal Ultrasound. It can also be used in lieu of MRI when the latter is not possible.

5. VACUUM BREAST BIOPSIES:

The ability to harvest significantly more tissue using the Minimal Invasive Breast Biopsy (MIBB) approach, such as the 11 gauge Vacuum technology used by ultrasound or on the stereotaxic table, has had a significant impact on the reliability of the pathology interpretation and to reduce the need for open surgery. This process has been further enhanced with new pathology procedures that provide immediate reliable preliminary core biopsy results to reduce unnecessary anxiety.

6. MANAGEMENT:

The presence of better and more targeted treatment agents, along with new radiation, surgical and reconstructive techniques, have also contributed to reduce recurrences and increase survival. The benefits of all of the above are enhanced by minimizing clinical fragmentation and reviewing the clinical, imaging and pathology data at weekly multidisciplinary breast oncology rounds to ensure best conciliation and management.

Much emphasis is now devoted to better targeting the right therapies to the right patients. This includes reviewing the tumor markers. genomic classification of breast cancer to better choose between hormonal manipulation, chemotherapy and the use of newer targeted agents such as and Pertuzumab, Bevacizumab and Trastuzumab or PARP inhibitors. Of equal interest is the newer sequencing protocols, including neo-adjuvant therapies that can result in 50% complete pathology response prior to surgical intervention. When total mastectomy is the only alternative, new microvascular techniques can provide excellent immediate reconstruction techniques. Finally, newer radiotherapy-related technology provides with shorter and more focused radiotherapy options.

READING MATERIAL:

Kaufman M, et al. Recommendations from an expert panel on the use of neoadjuvant systemic treatment for operable breast cancer. J.Clin Onc ;24 (12)1940-49, 2006

Coubert BP, et al: Preoperative systemic therapy with trastuzamab for breast cancer.Ann Oncol:173 409-419, 2006

TRAM Flap Breast Reconstruction with Expanders and Implants.

AORN Journal | February 1, 2000| Moran, S. L.; Herceg, S.; Kurtelawicz, K.; Serletti, J. M. |

14:45 - 15:15 Helpful and Harmful Herbs

Joseph A. Schwarcz PhD
Director, Office for Science and Society, McGill University

Some Views on the Nature of Science

1. Science is a process used to search for the truth. It is not a collection of unalterable "truths." It is, however, a self-correcting discipline. Such corrections may take a long time; bloodletting went on for centuries before its futility was realized. But as more scientific knowledge accumulates, the chance of making substantial errors decreases.

2. Certainty is elusive in science and it is often hard to give categorical "yes" or "no" answers to many questions. To determine if bottled water is preferable to tap water, for example, one would have to design a lifelong study of two large groups of people whose lifestyle was similar in all respects except for the type of water they consumed. This is virtually undoable. We therefore often have to rely on less direct evidence for our conclusions.

3. It may not be possible to predict all consequences of an action, no matter how much research has been done. When chlorofluorocarbons (CFCs) were introduced as refrigerants, no one could have predicted that thirty years later they would have an impact on the ozone layer. If something undesirable happens, it is not necessarily because someone has been negligent.

4. Any new finding should be examined with skepticism. A skeptic is not a person who is unwilling to believe anything. A skeptic, however, requires scientific proof and does not swallow information uncritically.

5. No major lifestyle changes should be made on the basis of any one study. Results should be independently confirmed by others. Keep in mind that science does not proceed by "miracle breakthroughs" or "giant leaps." It plods along with many small steps, slowly building towards a consensus opinion.

6. Studies have to be carefully interpreted by experts in the field. An association of two variables does not necessarily imply cause and effect. As an extreme example, consider the strong association between breast cancer and the wearing of skirts. Obviously, the wearing of skirts does not cause the disease. Scientists, however, sometimes show a fascinating aptitude for coming up with inappropriate rationalizations for their pet theories.

7. Repeating a false notion often does not make it true. Many people are convinced that sugar causes hyperactivity in children-not because they have examined studies to this effect but because they have heard that this is so. In fact, a slate of studies has demonstrated that if anything, sugar has a calming effect on children.

8. Nonsensical lingo can sound very scientific. An ad for a type of algae states that "the molecular structure of chlorophyll is almost the same as that of hemoglobin, which is responsible for carrying oxygen throughout the body. Oxygen is the prime nutrient and chlorophyll is the central molecule for increasing oxygen available to your system." This is nonsense. Chlorophyll does not transport oxygen in the blood.

9. There will often be legitimate, opposing views on scientific issues. But the impression that science cannot be trusted because "for every study there is an equal and opposite study" is incorrect. It is always important to examine who carried out a study, how well it was designed and if anyone stood to gain financially from the results. One must be mindful of who is the "they" in "they say that..." In many cases what "they say" is only gossip, inacurately reported.

10. Humans are biochemically unique. Not everyone exposed to a cold virus will develop a cold. Response to medications can be dramatically different. Eating fish can healthy for many but deadly to those with an allergy. Like me.

11. Animal studies are not necessarily relevant to humans although they may provide much valuable information. Penicillin, for example, is safe for humans but is toxic to guinea pigs. Rats do not require vitamin C as a dietary nutrient but humans of course do. Feeding high doses of a suspected toxin to test animals over a short term may not accurately reflect the effect on humans exposed to tiny doses over the long term.

12. Only the dose makes the poison, only the dose makes the cure. It does not make sense to talk about the effect of substances on the body without talking about amounts. Licking an aspirin tablet will do nothing for a headache but swallowing two tablets will make the headache go away. Swallowing a whole bottle of pills will make the patient go away.

13. "Chemical" is not a dirty word. Chemicals are the building blocks of our world. They are not good or bad. Nitroglycerine can alleviate the pain of angina or blow up a building. The choice is ours. Furthermore, there is no relation between the risk posed by a substance and the complexity of its name. Dihydrogen monoxide after all, is just water.

14. Nature is not benign. The deadliest toxins known, such as ricin from castor beans or botulin from the Clostridium botulinum bacterium are perfectly natural. "Natural" does not equate to safe and "synthetic" does not mean dangerous. The properties of any substance are determined by its molecular structure, not by whether it was synthesized in the laboratory by a chemist or by nature in a plant.

15. Perceived risks are often different from real risks. Food poisoning from microbial contamination is a far greater health risk than trace pesticide residues on fruits and vegetables.

16. The human body is incredibly complex and our health is determined by a large number of variables which include genetics, diet, the mother's diet during pregnancy, stress, level of exercise, exposure to microbes, exposure to occupational hazards and luck!

17. While diet does play a role in health, the effectiveness of specific foods or nutrients in the treatment of diseases is usually overstated. Individual foods are not good or bad, although overall diets can be described as such. The greater the variety of food consumed, the smaller the chance that important nutrients will be lacking in the diet. There is universal agreement among scientists that increased consumption of fruits and vegetables is beneficial.

18. The mind-body connection is an extremely important one. About 40% of people will improve significantly when given a placebo and about the same percentage will exhibit symptoms in response to a substance they perceive as dangerous. The mind is capable of making a heaven of hell, and a hell of heaven.

19. About 80% of all illnesses are self-limiting and will resolve almost no matter what kind of treatment is being followed. Often a remedy receives undeserved credit. Anecdotal evidence is unreliable because positive results are much more likely to be reported than negative ones.

20. There are no geese that lay golden eggs. In other words, if something sounds too good to be true, it probably is. As H.L. Mencken said, "Every complex problem has a solution that is simple, direct, plausible, and wrong."

21. Virtually any subject or issue that arises gets more interesting and more complicated on deeper examination. Ours is a fascinating world.

22. Physicians and researchers do not try to hide effective therapies from the public for monetary gain. But peddlers of “natural therapies” often overhype their wares for monetary gain.

22. Nobody has a monopoly on being right. As Will Rogers said, "everybody is ignorant, only on different issues."

15:15 - 15:45 Chocolate and Red Wine Anyone?

Joseph A. Schwarcz PhD
Director, Office for Science and Society, McGill University

Chocolate and Flavanols

Joe Schwarcz PhD

There is something unusual about the Kuna Indians living in the San Blas Islands of Panama. Or at least there was in the 1940s when a scientific paper described their extremely low blood pressure. The cause was not genetic; Indians who had moved to the mainland did not have low blood pressure. Were they eating or drinking something on the islands that lowered their blood pressure? This is what interested Dr. Norman Hollenberg of Harvard Medical School. Examination of the Kuna lifestyle revealed that a beverage made from minimally processed cocoa beans was extremely popular. Could this be the key to the unusually low blood pressure of the natives?

Hollenberg knew that cocoa beans, like other natural products, were chemically complex. Researchers had isolated dozens of compounds from cocoa beans, as well as from chocolate made from cocoa beans. Some of these had garnered attention in terms of health, particularly a family known as the flavanols. Indeed, chocolate manufacturers had already been interested in flavanols, and the Mars Company was working on developing a tasty high-flavanol cocoa powder. This turned out to be a challenge because flavanols have an inherent bitter taste. In any case, when Dr. Hollenberg approached Mars, the company was happy to provide him with a supply of flavanols. It didn't take long before Hollenberg's studies showed that flavanols relaxed blood vessels and improved blood flow to the brain by 33 percent. Chalk one up for chocolate!

The blood vessel relaxation effect is not the only benefit that has been noted. At the University of California at Davis, Dr. Carl Keen has observed a flavanol-related "blood thinning" effect. It seems flavanols interfere with the activity of blood platelets, which make blood coagulate. The effect is similar to that of a daily baby Aspirin, which people take to ward off heart attacks, many of which are caused by blood clots. There is yet another way that compounds in cocoa may help prevent heart attacks. At the University of Scranton, Dr. Joe Vinson examined the antioxidant effect of chocolate. Why look into this? Because one of the mechanisms by which coronary arteries get clogged involves the oxidation of low-density lipoproteins (LDL, the "bad cholesterol"). Presumably if this oxidation can be curtailed, heart attack risk decreases. Vinson found, albeit only in the test tube, that cocoa powder and dark chocolate were very effective at reducing LDL oxidation. What does this mean in terms of how much chocolate people should eat? Not much, although a provocative preliminary study has found that about 35 grams of defatted cocoa, roughly what is found in 1.5 litres, or seven cupfuls, of hot chocolate can have a significant impact on preventing LDL oxidation.

And the positive studies just keep coming. Dr. Roberto Corti at the University Hospital in Zurich showed that 40 grams (1.5 ounces) of dark chocolate improved the flow of blood through the coronary arteries, whereas white chocolate, devoid of flavanols, had no effect. Dr. Jeffrey Blumberg at Tufts University randomly assigned 20 subjects to receive 100 grams of dark or white chocolate for 15 days. The lucky subjects on the chocolate diet saw their blood pressure and cholesterol drop and their response to insulin improve. Perhaps even more telling is a

study carried out at the National Institute for Public Health and Environment in Holland. For 15 years, researchers following the health status of 470 men, ages 65 to 84, discovered that those who regularly ate cocoa products had lower blood pressure. But the really exciting finding was that the men who ate the highest amount of cocoa were less likely to die from heart disease. Still, this does not mean that people with high blood pressure, or indeed anyone else, should start guzzling chocolate. But if you are looking for a dessert, dark chocolate is a better choice than a doughnut.

A CocoaVia bar may be easier to justify than a chocolate-covered doughnut. This is the Mars Company's entry into the "functional food" market. Functional foods are those that aim to deliver more than just simple nutrition or taste, and they are now a $50 billion business in North America. Each CocoaVia bar contains 100 milligrams of flavanols. This means that two of these bars a day contain an amount of flavanols shown to have an effect on blood pressure and on platelet aggregation. Mars has even incorporated into each bar 1.5 grams of phytosterols, plant-derived compounds that can lower cholesterol levels. So far there have been no human trials to demonstrate the benefits (other than to the manufacturer) of consuming CocoaVia bars. But you never know where chocolate research will go. Dr. Hollenberg's work suggests that flavanols dilate blood vessels by triggering the release of nitric oxide, the same substance that is responsible for the activity of Viagra. Now if that effect stands up to clinical trials, women may be giving men chocolates on Valentine's Day.

Antioxidants such as flavanols are also thought to have an effect on the skin. Wilhelm Stahl and colleagues in Germany decided to put the matter to a scientific test. They had women consume a cup (250 millilitres) of either high- or low-flavanol cocoa daily for a period of 12 weeks. Women in the high-flavanol group showed reduced reddening of the skin upon exposure to ultraviolet light, increased skin thickness, better skin hydration and a significant decrease in skin roughness and scaling. So chocolate seems to be good for our outsides as well as our insides. And if you are worried about chocolate causing acne, don't be. There is no scientific evidence for that common belief.

Gorging on chocolate while pregnant or lactating, however, may not be such a great idea, if we go by a report from the University of Messina in Italy. Doctors found that a baby born to a mother who was a heavy consumer of cocoa and chocolate was irritable, jittery and often cried inconsolably. All of the baby's symptoms resolved when the mother was told to give up chocolate—but one wonders whether she then became the crankiest person in the family.

Grapes and Resveratrol

Joe Schwarcz PhD

They feast on croissants that ooze butter. They eat creamy cheeses and fat-filled pastries. Breakfast is pain au chocolat, washed down with espresso. There is no oatmeal in sight. I suspect most have never heard of flaxseed. Yet the French have the lowest death rate from heart disease in the European Union, and when we compare this rate with North America's—well, there is no comparison. Our incidence of heart disease is double that of the French, who are also much slimmer than Canadians and Americans. How do we explain this situation, which has been dubbed the "French Paradox"? According to some researchers the secret is to be found in wine, particularly red wine. More specifically, they point a finger at resveratrol, an antioxidant compound in the polyphenol family.

The simplified argument goes like this. Most heart attacks occur when a blood clot forms in a coronary artery and chokes off the flow of blood, starving the heart of oxygen. Blood clots form when the endothelium, the inner lining of the artery, is damaged. Such damage is associated with the formation of deposits called plaque, which in turn are linked to the presence of excessive amounts of cholesterol in the blood. But cholesterol carries out its dirty work only when it undergoes a chemical change stimulated by the presence of oxidizing agents such as free radicals. Oxidized cholesterol, then, is the real culprit, and if its production can be curtailed, the risk of a heart attack can be reduced. Antioxidants can do this—at least in the test tube.

Resveratrol, as it turns out, is not only an effective antioxidant, it can also reduce the blood's clotting ability. Little wonder then that resveratrol pills have begun to appear in health food stores. The efficacy of these pills, however, is highly questionable, since isolated resveratrol is an unstable compound. Special care has to be taken to preserve it; for example, by packing it in airtight capsules under a nitrogen atmosphere. Such products do exist and have been shown to have antioxidant effects on human cells in cultures, but there is no evidence that they do anything in live animals, never mind in humans.

While I find the resveratrol research engaging, so far it hasn't convinced me to up my intake of red wine. The truth is that I'm just as happy to have a glass of water with my dinner, and it doesn't even have to be bottled water.

But I just may have to rethink my beverage preference in light of some interesting research coming out of Harvard Medical School. Although it doesn't exactly relate to the "French Paradox," it is still pertinent. Why? Because we would all like to live longer. Molecular biologist Dr. David Sinclair and his colleagues have found a way to increase lifespan—at least for yeasts—by feeding them red wine! All right, so yeasts aren't people—or even rodents. But what works for yeasts may work for humans, because it seems that we also have a version of the gene that allows yeasts to live longer when exposed to red wine.

Yeasts are excellent organisms to use to study aging because they are easy to work with in the laboratory and have relatively short life cycles. As early as 1991, researchers had discovered that some yeasts lived longer than others. Why, was the big question. That was answered by Dr. Leonard Guarente of the Massachusetts Institute of Technology, who found that the long-lived yeasts produced an enzyme called sirtuin, which had the ability to repair damaged DNA. Strangely, the gene that codes for this enzyme, termed SIR2 ("silent information regulator"), becomes more active when yeast cells are starved of nutrients. This is not totally surprising because evidence exists that not only yeasts, but also fruit flies, rodents and monkeys all live longer when put on a calorie-restricted diet. This characteristic is probably an evolutionary vestige: when food is in short supply, reproduction is difficult and organisms need to live longer so as to postpone breeding until conditions improve. Some research has shown that humans who eat roughly 30 percent fewer calories than generally recommended live longer than average.

Researchers' attention turned to possible ways to activate the gene that seems to code for the enzyme that plays a role in increased life span. They started systematically to examine chemicals that could possibly increase enzyme activity. It didn't take long to find one that aroused their interest. Resveratrol performed remarkably well, mimicking the effect of calorie restriction. And let's face it, drinking a glass of red wine every day is a lot more pleasant than reducing calorie consumption by 30 percent. According to the research, one glass (4 ounces) is all that is needed to increase life expectancy by 10 years, if indeed the effect on humans is similar to that on yeasts. There seems to be a sort of justice in this research. Yeasts convert grape juice into wine, and wine repays the favour by providing resveratrol to allow the yeasts to live longer.

When Dr. Sinclair progressed from yeasts to mice, he found an interesting result, one that certainly captured the imagination of journalists around the world. "Red wine substance appears to counter bad health in fat mice," screamed the headlines. Dr. Sinclair fed one group of mice a standard laboratory diet, another group an unhealthy diet with 60 percent of the calories coming from fat, and a third group the same unhealthy diet supplemented with regular doses of resveratrol. As expected, the mice in the second group became obese, showed signs of diabetes and heart disease, and died prematurely. The mice in the resveratrol group also became fat, but they remained healthy and lived as long as the animals that ate a normal diet and stayed thin. Before you reach for the corkscrew, note that the amount of resveratrol given the mice was roughly equivalent to that found in 100 bottles of red wine. By all means, though, if you have obese mice and want them to live a long time, feed them resveratrol supplements.

There is also some intriguing preliminary evidence that drinking red wine may prevent Alzheimer's disease. "Preliminary" is the key word, but let's face it, all significant findings start out with preliminary research. Dr. Jun Wang at New York's Mount Sinai School of Medicine worked with mice that had been specially bred to produce high levels of a protein called beta-amyloid. This protein can accumulate in the brain and has been implicated in Alzheimer's disease. When Dr. Wang put such mice on a diet that included an amount of red wine equivalent to a couple of glasses a day for a human, he found something amazing. The mice were better able to solve mazes than a control group of animals that had consumed alcohol instead of wine. After the experiment, the brains of the mice were examined, and those in the wine group had significantly fewer deposits of beta-amyloid. Furthermore, Dr. Wang doused beta-amyloid protein with red wine in a test tube and discovered that the structure of the protein was altered in a fashion that prevented it from being deposited in the brain.

Research into resveratrol is clearly promising, but so far there is insufficient evidence to recommend that people who normally do not drink red wine take up the practice. And there are risks. Not much more than a couple of glasses a day has been associated with breast and oral cancers, and there are the social consequences of increased alcohol intake.

Returning to the "French Paradox," the answer to why the French are slimmer and are less likely to have heart disease may lie not in what they drink, but in what they eat—or rather what they don't eat. The French simply eat fewer calories than the majority of North Americans, and their obesity rate is only about 7 percent compared with about 33 percent for Americans.

In 2003, Dr. Paul Rozin of the University of Pennsylvania and his associates compared portion sizes in France and the United States, weighing servings in 11 comparable pairs of eateries in Paris and Philadelphia. These ran the gamut of pizzerias, fast food outlets and ethnic restaurants. The average portion size in the Paris restaurants was 277 grams as compared with 346 grams in Philly—a 25 percent difference. The American Chinese meals were a stunning 72 percent heftier than those served in the Parisian Chinese restaurants. Rozin also found that portions of packaged foods were larger in the United States. An American candy bar was 41 percent larger, a hot dog was 63 percent bigger and even single yogurt servings were much larger.

Then there was another finding. The French don't wolf down their meals, they take their time. Even at fast food joints like McDonald's, they take longer to eat their burgers and fries. Americans spend 14 minutes "enjoying" their fast food while the French linger for some 22 minutes. The French also don't eat at their desks and they don't eat on the run. In total, an average American spends an hour a day eating while a French person eats for some 100 minutes. It seems the French eat less and enjoy it more.

French wine producers prefer to credit red wine's antioxidants for producing the "French Paradox," and they have produced a white wine with similar properties. A team of wine researchers at Montpelier University have come up with a Chardonnay called "Paradoxe Blanc" that has almost the same antioxidant potential as red wine. They found

that if the grapes were macerated with the skins and seeds and the fermentation temperature increased, the polyphenol content of the wine increased dramatically.

Furthermore, these scientists managed to show that the Chardonnay really has an effect on the antioxidant capacity of the blood. They destroyed some of the insulin-producing cells in the pancreas of rats to make the animals diabetic, because diabetes is known to reduce the antioxidant capacity of the blood. Then they administered the new Chardonnay to the critters for six weeks and found that the antioxidant capacity was restored. So those drinkers who prefer white over red should track down some Paradoxe Blanc. Of course, the real paradox is why people just don't eat more fruits and vegetables, which have more antioxidants than red or white wine!

While the role of red wine in the French Paradox may be ambiguous, this alleged connection has spawned some other possibly fruitful lines of research. Dr. Joseph Anderson of the State University of New York at Stony Brook spends much of his time looking through a colonoscope searching for cancers and precancerous polyps in people's colons. Because alcohol consumption has been suspected as a contributing factor to colorectal cancer, Anderson decided to survey his patients about their alcohol habits. He found that beer or spirit consumers who drank more than one drink a day were significantly more prone to colorectal tumours than moderate drinkers or abstainers. But red wine drinkers, on the other hand, seemed to be protected from the disease. Only 3 percent of those who drank at least three glasses of red wine a week had either cancerous or precancerous lesions, as compared with 10 percent of those who drank no alcohol. White wine showed no benefit. Anderson thinks that resveratrol, which is found far more extensively in red grapes than in white, is responsible.

There appears to be some theoretical justification for this possibility. Prostaglandins are compounds produced in the body that serve a multitude of functions, but some can suppress immunity and even stimulate tumour cell growth. Resveratrol has been shown to block an enzyme, cyclooxygenase-2, which catalyzes the conversion of arachidonic acid (a dietary component) into the problematic prostaglandin. In separate experiments, resveratrol has been shown to be a potent scavenger of potentially harmful free radicals. Still, the resveratrol connection may be overly simplistic, given that there are many other polyphenols in red wine that may contribute to the overall antioxidant effect.

Dr. Janet Stanford of the Fred Hutchinson Cancer Research Center in Seattle shares the view that resveratrol may be the key component. She studied alcohol consumption in 750 men with recently diagnosed prostate cancer and in a similar group of healthy men. Drinking at least four glasses of red wine a week was associated with a 50 percent lower risk. Stanford hypothesizes that resveratrol's ability to rid the body of free radicals, its anti-inflammatory effect and its tendency to hold down cell growth all play a part in its protective role.

Since free radicals have also been implicated in the neurological damage that follows a stroke, Dr. Sylvain Doré and colleagues at Johns Hopkins University investigated resveratrol's potential to prevent such damage. Oral pretreatment of mice with resveratrol resulted in a 40 percent decrease in the area of the brain damaged by the induced stroke. Doré even managed to tease out the specific mechanism involved in the protection, namely an increased level of heme oxygenase, an enzyme known to shield nerve cells against free-radical damage. Based on his mice experiments, Doré thinks that a couple of glasses of red wine a day could produce a prophylactic effect against stroke damage in humans. But that's just a guess—just like almost everything else about red wine.

Now let's get back to the "French Paradox." Actually, there may not even be one. Some researchers argue that the French use different criteria in ascribing causes of death and that some cases that would be described in North America as "cardiac" would not necessarily be described this way in France. In any case, while the extent of a reduced risk of heart disease in France is debatable, there is one thing we do know from reliable statistics: the French life expectancy is roughly the same as it is in North America. They don't live any longer; they just exit by a different route.

Notes

Wednesday, Nov. 25 – Afternoon Plenary

15:45 - 16:15 Dermatology Quiz

Wayne Carey MD, FRCP
Associate Professor , Department of of Dermatology,
Royal Victoria Hospital – MUHC
Director, Dermatology Surgery, McGill University

Notes

Abdelnour, Miriam
Montreal, QC

Abikhzer, Victor
Montreal, QC

Abrahams, Heather
Montreal, QC

Acre, Yael
Westmount, QC

Adams, Robert
Alexandria, ON

Ahmed, Sabrina
Brossard, QC

Aina, Judy
LaSalle, QC

Akriotis, Van
Scarborough, ON

Albert, Andrew
Burks Falls, ON

Albert, Deborah
Burks Falls, ON

Alcius, Michaelle
Toronto, ON

Alexopoulos, Karen
Guelph, ON

Alizadehfar, Reza
Montreal, QC

Almasi, Julie
Montreal, QC

Alper, Deborah
Montreal, QC

Andonatos, Stella
Montreal, QC

Arora, Harpreet
Cambridge, ON

Arora, Shefali
Cambridge, ON

Arsenault, Lyne
Hawkesbury, ON

Aspler, Aviva
Côte-St-Luc, QC

Assayag, Yan Raphael
Montreal, QC

Babakifard, Katayoun
Montreal, QC

Backler, John
Westmount, QC

Bah, Abdoulame
Montreal, QC

Bailey, Robert
Montreal, QC

Bakker, Gerry
Sudbury, ON

Bartlett-Esquilant, Gillian
Montreal, QC

Bashala, Roger
Campbellton, NB

Baylis, Penny-Jane
Montreal, QC

Beauchesne, Christian
Granby, QC

Ben Haddad, Abdeltif
Dorval, QC

Benaroch, Thierry E.
Montreal, QC

Berringer, Ross
Ottawa, ON

Birss, John
Cambridge, ON

Blach, Peter
Cornwall, ON

Blondeau, Hélène
Québec, QC

Boersma, Robert
Arnprior, ON

Boillat, Miriam E.
Montreal, QC

Bosse, Natasha
Montreal, QC

Bouchard, Jacques
La Malbaie, QC

Boulay, John
Montreal, QC

Brooks, Douglas
Sault Ste Marie, ON

Brousseau, Martine
Outremont, QC

Brown, Bernard
Candiac, QC

Bruemmer, Aurel
Montreal, QC

Brunsdon, Peta
Saint John, NB

Buchanan, Gordon Stuart
St Andre Avellin, QC

Bui, Yen Giang
Longueuil, QC

Busuioc, Ruxandra
Montreal, QC

Caldareri, Carmelo
Montreal, QC

Cameron, Clare
Cambridge, ON

Cameron, David
Waterloo, ON

Carey, Wayne
Westmount, QC

Carpentier, Véronique
St-Rémi, QC

Carrasco, Julian
Mistissini, QC

Carroll, Paul
Montreal, QC

Cartwright, Pierre
Étang-du-Nord, QC

Cavallé-Garrido, Tiscar
Montreal, QC

Cecere, Assunta
Montreal, QC

Chan, Grace
Ottawa, ON

Chan, Peter
Montreal, QC

Chana, Karam
Victoria, BC

Chaput, Gen
Lasalle, QC

Charghi, Parissa
Montreal, QC

Chen, Marie-Luce
Mount-Royal, QC

Chettiar, Ramen
Grand Falls, NB

Ciccone, Mario V.
Timmins, ON

Ciuntu, Ioana
Montreal, QC

Coelho, Ramona
Montreal, QC

Cohen Taussky, Tamia
Montreal, QC

Cohen, Martin
Pointe-Claire, QC

Conde, Jean Joseph
Val d'Or, QC

Cordeau, Karyne
Québec, QC

Cosman, Catherine
Knowlton, QC

Cossette, Louis
Senneterre, QC

Courchesne, Donna
Shawville, QC

Couture, Denis
Otterburn Park, QC

Crocker, Percy
Torbay, NL

Cruz, Javier
Montreal, QC

da Costa, Derek
Dollard-des-Ormeaux, QC

Damyanova, Anastasiya
Longueuil, QC

Dannenbaum, David
Montreal, QC

Dardashti, Marzieh
Beaconsfield, QC

Dawes, Martin
Montreal, QC

De Ladurantaye, Alain
Duhamel-Ouest, QC

Delaney, J. Scott
Montreal, QC

Desmarais, Maryse
Ste-Adèle, QC

Desmeules, Jean
Cowansville, QC

Desmond, Gerard
Swan Lake, MB

Dobrowolski, Marek
Ottawa, ON

Doucet, Lionel E.
St-Jean, QC

Dove, Marion
Montreal, QC

Dowdall, Mary
Montreal, QC

Drummond, Robert
Montreal, QC

Dubois-Roy, Monique
Montreal, QC

Duret, Pascale
Montreal, QC

Dworkind, Michael A.
Montreal, QC

Dysart, Allison
Sackville, NB

Eaton, David
Wheatly, ON

Élie, Michel
Montreal, QC

Eliev, Sonia
Gracefield, QC

Elliott, Bonney
Ottawa, ON

Engo, Michael
LaSalle, QC

Eniojukan, Rachael
Chateauguay, QC

Erb, John
Lansdowne, ON

Estevez, Wendy
Dollard-des-Ormeaux, QC

Falls, Elizabeth
St. Bruno, QC

Farah, Rita
Montreal, QC

Favreault, Luce
Amos, QC

Febbraro, Mario
Sault Ste Marie, ON

Fegelman, Alan
Toronto, ON

Fernandez, Carmen
Montreal, QC

Figueira, Sabina
Montreal, QC

Fine, Barry
Chisasibi, QC

Finkelberg, Susan
Dollard-des-Ormeaux, QC

Fitzcharles, Mary-Ann
Montreal, QC

Folkerson, Curtis S.
Ste-Cassis de Masham, QC

Fortin, Marquis
Montreal, QC

Fournier, Marcel
Montreal, QC

Frechette, Claude
Laval, QC

Friedman, Gad
Montreal, QC

Fruth, Irmgard
Baie d'Urfé, QC

Fuks, Maria
Montreal, QC

Gagnon, Claude-François
Chambly, QC

Gallant, Marc
Acton Vale, QC

Garant, Dominique
Amos, QC

Gardner, Marieke
Côte-St-Luc, QC

Gauthier, Gilles
Gaspé, QC

Gavsie, Adam
Westmount, QC

Geagea, Khalil
Montreal, QC

Genest Jr., Jacques
Montreal, QC

Genge, Angela
Montreal, QC

Geukjian, S. K. Gregory
Ormstown, QC

Ghazigian, Taline
Dollard-des-Ormeaux, QC

Giordano, Isabelle
Gatineau, QC

Glaser, Stuart R.
Town of Mount Royal, QC

Goehring, Lawanda N.
Hatboro, PA, United States

Golberg, Deborah
Montreal, QC

Goldstein, Howard
Côte-St-Luc, QC

Golgoon, Michael
Pointe-Claire, QC

Gordon, Benjamin
Dollard-des-Ormeaux, QC

Gordon, Earl
Woodlawn, ON

Gore, Brian
Westmount, QC

Goulard, Jean-Francois
Bathurst, NB

Grad, Roland
Montreal, QC

Gray, Susan
Saint John, NB

Grunbaum, Beatrice
Côte-St-Luc, QC

Guerra Escobio, Ana Maria
Anjou, QC

Hackett, Charles
Atlanta, GA, United States

Harvey, Pierre Claude
Sept-Îles, QC

Hazell, Paul
Port Hope, ON

Hechtman, Lily
Montreal, QC

Heyding, Robert
Toronto, ON

Honos, George N.
Montreal, QC

Houde, Jean
Rouyn-Noranda, QC

Huang, Sarah
Montreal, QC

Iancu, Andreea
Hudson, QC

Ince-Cushman, Daniel
Montreal, QC

Ionescu, Loretta-Vivianne
Montreal, QC

Iqbal, Sameena
Montreal, QC

Iskandar, Hani
Verdun, QC

Ith, Bun Hor
Laval, QC

Jagan, Sarva
St-Lambert, QC

James, Chris
Victoria, BC

Jast, Zygmunt
Montreal, QC

Jilwan, José
Saint-Laurent, QC

Jimenez, Vania
Montreal, QC

Jobin, Nicolas
Baie-Comeau, QC

Kader, Tina
Montreal, QC

Karayan, Lina
Montreal, QC

Kassab, François
St-Jean-sur-Richelieu, QC

Kawerninski, Michael
Smithers, BC

Kealy, Walter
Sudbury, ON

Kehler, Faye
Dryden, ON

Keyserlingk, John R.
Montreal, QC

Khadilkar, Madhu
Montreal, QC

Khakee, Sam
Montreal, QC

Khatib, Ahmad
Campbellton, NB

Khazandar, Fatimah
NDG, QC

Klein, Benjamin
Montreal, QC

Klein, Jack
Kirkland, QC

Klincewicz, Stephen
Ambler, PA, United States

Korin, Tamara
Westmount, QC

Kovacina, Nebojsa
Montreal, QC

Kovitch, Ingrid
Westmount, QC

Kremer, Bernardo
Montreal, QC

L'Heureux, Christian
Ville-Marie, QC

La Barre, Marc
Papineauville, QC

Labarias, Jose Luis
Montreal, QC

Labelle, Céline
Montreal, QC

Lacroix, Chantal
Ottawa, ON

Lacroix, Daniel
Moose Creek, ON

Ladores, Mina
Montreal, QC

Ladouceur, Roger
Montreal, QC

Lafrenière, Celine
Ste-Julie, QC

Lajzerowicz, Michelle
Wakefield, QC

Lalla, Daniel E.
Montreal, QC

Lalla, Leonora
Montreal, QC

Lam, Loan
Town of Mount Royal, QC

Lamarche, Maurice
Shawville, QC

Lamarre, Martin
Gaspé, QC

Landry, Elaine
Shediac, NB

Landry, Esther
Gaspé, QC

Lang, Eddy
Montreal, Qc

Laplante, Louisette
Sherbrooke, QC

Laplante, Patrice
Sherbrooke, QC

Laplante, Severine
Montreal, QC

LaRue, Frank J.
Gatineau, QC

Lau, William
Laval, QC

Laurin, Carroll H.
Town of Mount Royal, QC

Le Clair, Marie
Hudson, QC

Leahy, James R.
Windsor, NS

LeBel, Tania
Ottawa, ON

Leblanc, Isabelle
Montreal, QC

Lee, Dennis
Toronto, ON

Levin, Richard I.
Montreal, QC

Libman, Michael D.
Montreal, QC

Liebich, Anne-Marie
Montreal, QC

Lu, Paul
Winnipeg, MB

Luconi, Francesca
Montreal, QC

Luger, Sherry
Montreal, QC

Luna, Alberto
Dollard-des-Ormeaux, QC

Lysy, Paul G.
Westmount, QC

Ma, Grace
Montreal, QC

Ma, Sandy
Mukilteo, WA, United States

Macek, Adrian
Montreal, QC

MacGeachy, Fiona
Dollard-des-Ormeaux, QC

Macleod, Carol
Montreal, QC

Magnan, Johanne
Lachine, QC

Mahood, Robert
Montreal, QC

Main, Jeff
waterloo, ON

Mallet, Louise
Montreal, QC

Malus, Michael
Montreal, QC

Mamen, Julie
Hudson, QC

Manoli, Sabrine
Dollard-des-Ormeaux, QC

Maranda, Julie
St-Lazare, QC

Marc, Regimbal
Gatineau, QC

Marchand, Pierre
Sherbrooke, QC

Martin, Colette
Kirkland, QC

Massey, Ephraim
Westmount, QC

Mazzarelli, Mark
Beaconsfield, QC

Mehta, Jagdish
Dollard-des-Ormeaux, QC

Meisels, Monica
Québec, QC

Ménard, Jacques
Gatineau, QC

Michaud, Julie
Chicoutimi, QC

Minasian, Vicken
Laval, QC

Minz, Gabriel
Dorval, QC

Mitchell, Gregor
Lachute, QC

Mitnick, Howard
Montreal, QC

Mitrica, Mirela
Beaconsfield, QC

Moini, John
Brossard, QC

Monahan, Barbara
Montreal, QC

Morin, Carl
Ottawa, ON

Morin, Suzanne
Montreal, QC

Morris, Randi
Westmount, QC

Morris, Sandra
Montreal, QC

Morrison, Cindy
Arnprior, ON

Mout, Julie
West Brome, QC

Nadkarni, Ashok
Cornwall, ON

Narasiah, Lavanya
Montreal, QC

Nazerali, Najmi
Montreal, QC

Nelson, Lawrence
Pickle Lake, ON

Nemeth, Joe
Montreal, QC

Neylon, Norah
Montreal, QC

Ng, Sheau Chian
Corner Brook, NL

Nguyen Duong , Y Nhu
Town of Mount Royal, QC

Nica-Danes, Doina
Montreal, QC

Nimigan, Wayne
Ottawa, ON

O'Shaughnessy, Gael
Montreal, QC

Ocasiones, Carmencita
Laval, QC

Oommen, Ashok
Montreal, QC

Pantazopoulos, Efrosini
Hudson, QC

Paquet, Christine
Fatima, QC

Parayre, Michel
Rouyn-Noranda, QC

Parent, Lorne
Ottawa, ON

Parent, Marc-Antoine
L'Étang-du-Nord, QC

Parent, Roger
Québec, QC

Parsons, Trent
Corner Brook, NL

Partlova, Hana
Baie d'Urfé, QC

Pavilanis, Alan
Montreal, QC

Peacock, Ingrid
Carbonear, NL

Pearson, Margaret
Pointe-Claire, QC

Perley, Michael
Woodstock, NB

Perrotta, Rosalba
Montreal, QC

Peterson, George
Saint-Basile-le-Grand, QC

Pinard St-Pierre, Vanessa
Montreal, QC

Poirier, Eric
Sept-Iles, QC

Poitevin, David
Sault Ste Marie, ON

Polson, George
Ste-Anne-de-Bellevue, QC

Poray-Wybranowski, Jerzy
Longueuil, QC

Préfontaine, Odette
St-Eustache, QC

Prossin, Albert
Verdun, QC

Quao, Nii T.
Montreal, QC

Quesada, Michel
Le Creusot, Bourgogne, France

Radhakrishna, Mohan
Montreal, QC

Ratner, Jack
Montreal, QC

Reid, Shelley
Ottawa, ON

Rezaeifar, Parand
Montreal, QC

Riche, Cyril
St. John's, NL

Richter, Anne-Katrin
Montreal, QC

Rideout, Gary
Mount Pearl, NL

Rif, Maria
Montreal, QC

Rivilis, Jeffrey
Montreal, QC

Rivington, Jennifer
Arnprior, ON

Robinson, Elizabeth
Montreal, QC

Rodger, Linda
Fossambault, QC

Rohan, Ivan
Montreal, QC

Rohan, Peter
Montreal, QC

Rosengarten, Michael David
Montreal, QC

Rotman, Laurie
Scarborough, ON

Routh, John
Port Hope, ON

Roy, Christine
Drummondville, QC

Roy, Nadine
Dieppe, NB

Rubin, Alexandra
Montreal, QC

Russek, Richard
Cambridge, ON

Sader, John
Town of Mount-Royal, QC

Salazar-Oldrich, Trinidad
Beaconsfield, QC

Sami, Magdi Hanna
Montreal, QC

Sanche, Gilbert
Laval, QC

Satenstein, Gary
Wakefield, QC

Saul, Mark
Masham, QC

Scheim, Alyssa
Dorval, QC

Schnare, Ted
Ottawa, ON

Schoel, Gerald
Montreal, QC

Schulz, Jan
Montreal, QC

Schwarcz, Joseph A.
Montreal, QC

Schweitzer, Morris
Montreal, Qc

Sheftel, Raisa
Verdun, QC

Sheppard, Richard
Montreal, QC

Shiff, Dori
Montreal, QC

Shulman, H. Mitchell
Montreal, QC

Sims, Louise
Cambridge, ON

Skanes, Susan
Moncton, NB

Smeja, Christina
Montreal, QC

Smolinski, Walter
Saint John, NB

Solymoss, Susan
Montreal, QC

Son, Florina
Ile-Bizard, QC

St-Cyr, Julie
Montreal, QC

Stanciu, Adela
Montreal, QC

Stanley, Donald E.
Nobleboro, ME, United States

Starr, Michael R.
Montreal, QC

Steg, Doris
Montreal, QC

Steibelt, Roslyn
Montreal, QC

Stein, Michael
Montreal, QC

Steinman, Robert
Montreal, QC

Stiharu, Simona
Pierrefonds, QC

Sun, Kathryn
Montreal, QC

Superstein, Rosanne
Montreal, QC

Ta, Diana
Val d'Or, QC

Taylor, John
Chapleau, ON

Tector, Suzie
Ottawa, ON

Teodorescu, Cristina
Lasalle, QC

Tesfaye, Yoseph
Montreal, QC

Tessier, Dominique
Montreal, QC

Tewfik, Yvette-Nelly
Montreal, QC

Thériault, Marie-Noël
Ville-Marie, QC

Thériault, Pierre
Carleton, QC

Touzel, Liz
Napanee, ON

Touzel, Tom
Napanee, ON

Tozer, Nancy
Pointe-Claire, QC

Trattner, Raquel
Pointe-Claire, QC

Tremblay, Eric
Montreal, QC

Tremblay, Florence
Montreal, QC

Tremblay, Jacques
Montreal, QC

Tremblay, Louise
Pointe-Claire, QC

Tremblay, Marino
Rawdon, QC

Tremblay, Roger
Cornwall, ON

Tsiodras, Athanasios
Montreal, QC

Tulandi, Tati
Beaconsfield, QC

Turcotte, Jean
Valcourt, QC

Tzouannis, Nicholas
Sherbrooke, QC

Van Sterthem, Marie-Josée
Pincourt, QC

Vernec, Alan
Ottawa, ON

Versteeg, Elmyre
Toronto, ON

Walker, Angela
Carignan, QC

Ward, Brian J.
Montreal, QC

Wassmann, Sven
Montreal, QC

Weber, Marie
Montreal, QC

Weech, Philip
Montreal, Qc

Wein, Theodore
Montreal, QC

Welik, Leonard
Hudson, QC

Whiteman, Avi
Montreal, QC

Wierzchoslawski, Krzysztof
Miramichi, NB

Wise, Audrey
Montreal, QC

Younes, Layla
Montreal, QC

Zaklos, Mayer
Montreal, QC

Zavotsky, Diane
Salmon, ID, United States

Ziegler, Cleve
Montreal, QC

Zigman, Michael
Montreal, QC

Zylberszac, Bernard
Laval, QC

Régie de l'assurance maladie Québec

DEMANDE DE REMBOURSEMENT

Programme de formation continue
Annexe XIX - FMOQ

PROFESSIONNEL

NOM	PRÉNOM	Nº DU PROFESSIONNEL

FORMATION CONTINUE

JOUR	01	02	03	04	05	06	07	PÉRIODE DU ANNÉE MOIS JOUR
QUANTIÈME								
DURÉE (1 jour ou 0,5 jour)								AU ANNÉE MOIS JOUR
ALLOCATION FORFAITAIRE (Montant réclamé par jour)								MONTANT TOTAL DE L'ALLOCATION FORFAITAIRE

IMPORTANT : La formation doit avoir lieu un jour ouvrable (les samedis, dimanches et jours fériés sont exclus).

RENSEIGNEMENTS COMPLÉMENTAIRES

PIÈCE JUSTIFICATIVE :
L'attestation liée à la formation, dûment signée par le responsable officiel du cours de formation, doit être jointe. Cette attestation doit préciser le nom de l'organisme responsable du cours de formation, la ou les dates de l'activité, la durée de l'activité de formation ainsi que la catégorie de crédits de formation attribués.

SIGNATURE DU PROFESSIONNEL

Ce formulaire doit être signé à la main et daté par le médecin (afin de faciliter la vérification, ne pas utiliser un stylo noir). Les photocopies et les tampons ne sont pas acceptés.

J'atteste que les renseignements inscrits sur la présente demande sont exacts.	SIGNATURE	ANNÉE MOIS JOUR

L'original de la demande de remboursement doit être envoyé à :

Régie de l'assurance maladie du Québec
Case postale 500
Québec (Québec) G1K 7B4

McGill Faculty of Medicine

Certificate of Attendance

This is to certify that the undersigned has attended the continuing medical education activity entitled:
60th Annual Refresher Course for Family Physicians on November 23, 2009
Bonaventure Hilton Montréal, Montréal, Québec

Participant's Name (printed):

Participant's Signature:

Study credit hours: ____ (for a maximum number of 8 credits for the day)

This event is an accredited group learning activity (Section 1) as defined by the Maintenance of Certification program of the Royal College of Physicians and Surgeons of Canada (24.5 hours). This program meets the accreditation criteria of the College of Family Physicians of Canada and has been accredited for (24.5) MAINPRO-M1 credits. The Centre for CCHPE, Faculty of Medicine, McGill University designates this educational activity for a maximum of (24.5) Category 1 credits towards the AMA Physicians Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The McGill Centre for CCHPE is accredited by the Committee on Accreditation of Canadian Medical Schools (CACMS) as an accrediting body for continuing medical education activities for physicians. Participants eligible to receive AMA Physicians Recognition Award credits can claim the AMA PRA Category 1 credits through a reciprocal agreement. Participants eligible to receive Prescribed Credit Hours from the AAFP can claim the credits through a reciprocal agreement.

Ivan Rohan, MD, CCFP
Course Director

McGill Faculty of Medicine

Certificate of Attendance

This is to certify that the undersigned has attended the continuing medical education activity entitled:
60th Annual Refresher Course for Family Physicians on November 24, 2009
Bonaventure Hilton Montréal, Montréal, Québec

Participant's Name (printed):

Participant's Signature:

Study credit hours: ____ (for a maximum number of 8.75 credits for the day)

This event is an accredited group learning activity (Section 1) as defined by the Maintenance of Certification program of the Royal College of Physicians and Surgeons of Canada (24.5 hours). This program meets the accreditation criteria of the College of Family Physicians of Canada and has been accredited for (24.5) MAINPRO-M1 credits. The Centre for CCHPE, Faculty of Medicine, McGill University designates this educational activity for a maximum of (24.5) Category 1 credits towards the AMA Physicians Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The McGill Centre for CCHPE is accredited by the Committee on Accreditation of Canadian Medical Schools (CACMS) as an accrediting body for continuing medical education activities for physicians. Participants eligible to receive AMA Physicians Recognition Award credits can claim the AMA PRA Category 1 credits through a reciprocal agreement. Participants eligible to receive Prescribed Credit Hours from the AAFP can claim the credits through a reciprocal agreement.

Ivan Rohan, MD, CCFP
Course Director

McGill Faculty of Medicine

Certificate of Attendance

This is to certify that the undersigned has attended the continuing medical education activity entitled:
60th Annual Refresher Course for Family Physicians on November 25, 2009
Bonaventure Hilton Montréal, Montréal, Québec

Participant's Name (printed):

Participant's Signature:

Study credit hours: ____ (for a maximum number of 8.0 credits for the day)

This event is an accredited group learning activity (Section 1) as defined by the Maintenance of Certification program of the Royal College of Physicians and Surgeons of Canada (24.5 hours). This program meets the accreditation criteria of the College of Family Physicians of Canada and has been accredited for (24.5) MAINPRO-M1 credits. The Centre for CCHPE, Faculty of Medicine, McGill University designates this educational activity for a maximum of (24.5) Category 1 credits towards the AMA Physicians Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The McGill Centre for CCHPE is accredited by the Committee on Accreditation of Canadian Medical Schools (CACMS) as an accrediting body for continuing medical education activities for physicians. Participants eligible to receive AMA Physicians Recognition Award credits can claim the AMA PRA Category 1 credits through a reciprocal agreement. Participants eligible to receive Prescribed Credit Hours from the AAFP can claim the credits through a reciprocal agreement.

Ivan Rohan, MD, CCFP
Course Director