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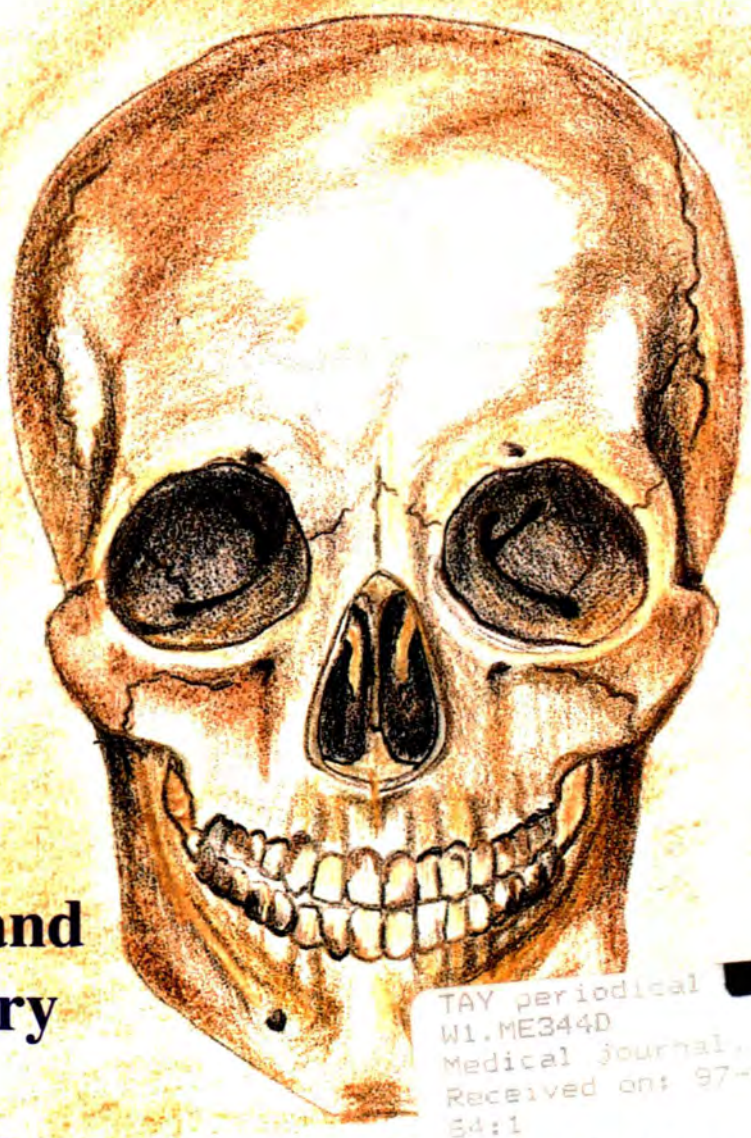


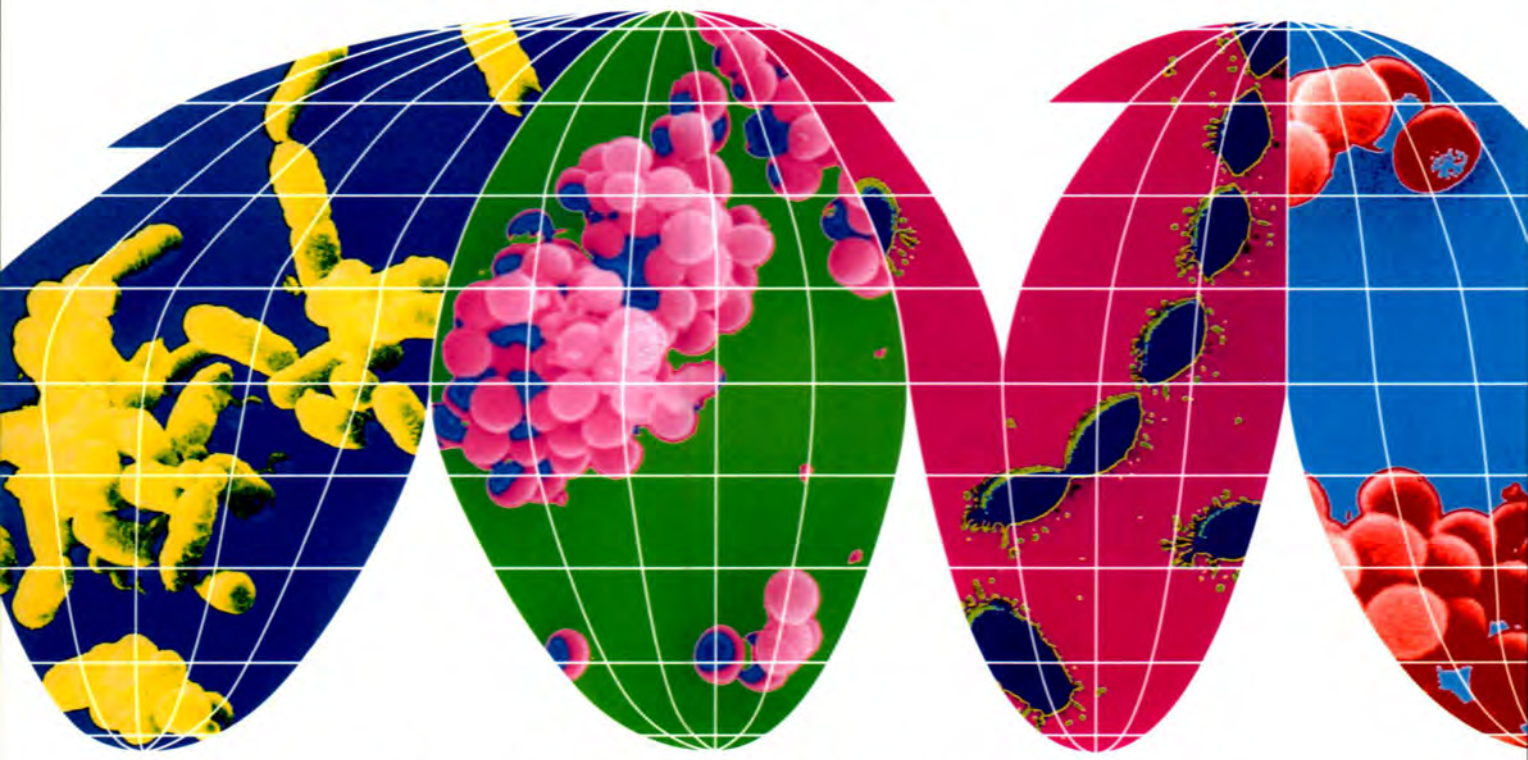
Neurology and Neurosurgery

INSIDE:

- The Decade of the Brain
- Charles Clifford Macklin, A Historical Overview
- Nutrition and Socioeconomic Status
- Management of Head Trauma
- Artificial Neural Networks
- Cochlear Implants
- Lumbar Disc Herniation
- Meds'94 Residency Assignments
- Class Reports, Alumni News, Stitches In Time, Medical Fax and more...

IN UPPER AND LOWER RTI

A CHANGING WORLD OF PATHOGENS...



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With its broad spectrum of activity, BIAXIN† effectively covers key pathogens causing today's respiratory tract infections – typical, atypical (including *Mycoplasma pneumoniae*), and β-lactamase-producing pathogens[1] – with bactericidal activity against *H. influenzae*.[2,3] And, BIAXIN has a tolerability comparable to amoxicillin and cephalosporins[4,5] without cross allergenicity.

BIAXIN*

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FIRST LINE FOR TODAY'S WORLD OF RTI

COMMITTED TO THE CONQUEST OF INFECTIOUS DISEASE

†BIAXIN is indicated for respiratory tract infections caused by *Haemophilus influenzae*, *Moraxella (Branhamella) catarrhalis*, *Streptococcus pneumoniae*, *Streptococcus pyogenes*, and *Mycoplasma pneumoniae*.

PAAB

EDITORIAL STAFF

THE NEXT ISSUE

EMERGENCY MEDICINE

SUBMISSION DEADLINES

ARTICLES .....*March 4, 1995*
ARTWORK.....April 1, 1995

COVER ART:

Mixed media drawing on paper of the anterior aspect of the adult human skull. The skull is a highly specialized bony and cartilaginous structure which houses four of the five senses exclusively, and contains the human brain - widely considered to be the most complex known structure.

Romy Croitoru, Meds '96

GUIDELINES TO AUTHORS

The purpose of the U.W.O. Medical Journal is to provide a single forum for original articles based on clinical or research medicine of topical or historical relevance. Since the readership of the Journal is interdisciplinary, articles published will attempt to reflect the wide range of medical disciplines. Informal peer review is required, i.e. non-specialist authors are encouraged to collaborate with or at minimum have their work reviewed by a specialist in the field. This individual, if not a co-author, is to be acknowledged at the end of the paper. Short biographical notes on the authors are to be included at the beginning of each paper. Affiliation with UWO is not a prerequisite for authorship.

Submissions are to include two paper copies double spaced and the full text on 3.5" computer diskette in Microsoft Word (Macintosh) or Word Perfect (IBM) format. Please include information about the authors. *Journal* staff artists are pleased to assist with pictorials, which are normally printed in black and white. Submissions and disks become the property of the *Journal*. *The Journal reserves the right to correct grammatical and stylistic errors.*

References are indicated numerically in the text[1] and listed as endnotes in order of appearance.[2] Punctuation comes before reference numbers and sentences are separated by one space only. Examples of *Journal re*ference format follow:

1. Douglas NJ, Thomas S, Jan MA. Clinical value of polysomnography. Lancet 1992; 339(2):347-50.

2. Dement WC, Carskadon MA, Richardson G. Excessive daytime sleepiness in the sleep apnea syndrome. In: Guilleminault C, Dement WC, eds. Sleep apnea syndromes. New York: Alan R Liss, 1978:23-46.

Please direct submissions, including return address, phone and fax number, to: *UWO Medical Journal, Health Sciences Blg., University of Western Ontario, London, Ontario N6A 5C1. Tel: (519) 661-2076 Fax: (519) 661-3797*
or Ross Mantle, Editor,
Fax. (519) 434 9199 Tel. (519) 434 2299

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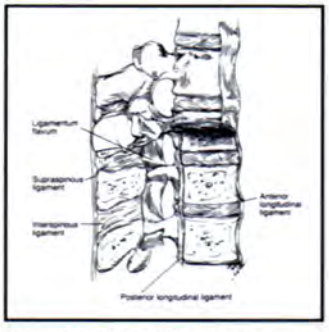

Lumbar Disk Herniation and Low Back Pain, A Neurosurgical Persective (P. 41)

Once-a-day

CARDIZEM® CD

Controlled Delivery diltiazem HCl/NORDIC

PRESCRIBING INFORMATION

"CARDIZEM" CD Once-a-day Controlled Delivery Capsules 120 mg, 180 mg, 240 mg and 300 mg.

THERAPEUTIC CLASSIFICATION

Antihypertensive and Antianginal agent.

INDICATIONS AND CLINICAL USE

Angina

1. CARDIZEM CD is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta-blockers and/or organic nitrates or who cannot tolerate those agents.
2. CARDIZEM CD may be tried in combination with beta-blockers in chronic stable angina patients with normal ventricular function. When such concomitant therapy is introduced, patients must be monitored closely (See WARNINGS).
3. Since the safety and efficacy of CD capsules in the management of unstable or vasospastic angina has not been substantiated, use of this formulation for these indications is not recommended.

Hypertension

CARDIZEM CD is indicated for the treatment of mild to moderate essential hypertension. CARDIZEM CD should normally be used in those patients in whom treatment with diuretics or beta-blockers has been ineffective, or has been associated with unacceptable adverse effects.

CARDIZEM CD can be tried as an initial agent in those patients in whom the use of diuretics and/or beta-blockers is contraindicated, or in patients with medical conditions in which these drugs frequently cause serious adverse effects.

Safety of concurrent use of CARDIZEM CD with other antihypertensive agents has not been established.

CONTRAINDICATIONS

Diltiazem HCl is contraindicated:

1. In patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker;
2. In patients with second or third degree AV block;
3. In patients with known hypersensitivity to diltiazem;
4. In patients with severe hypotension (less than 90 mm Hg systolic);
5. In myocardial infarction patients, who have left ventricular failure manifested by pulmonary congestion;
6. In pregnancy and in women of child-bearing potential.

WARNINGS

Cardiac Conduction

Diltiazem prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (6 of 1208 patients or 0.5%).

First degree AV block was observed in 5.8% of patients receiving CARDIZEM CD (see ADVERSE REACTIONS).

Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction.

Congestive Heart Failure

Because diltiazem has a negative inotropic effect *in vitro* and it affects cardiac conduction, the drug should only be used with caution and under careful medical supervision in patients with congestive cardiac failure (see also CONTRAINDICATIONS).

Use with Beta-blockers

The combination of diltiazem and beta-blockers warrants caution since in some patients additive effects on heart rate, AV conduction, blood pressure or left ventricular function have been observed. Close medical supervision is recommended.

Generally, diltiazem should not be given to patients with impaired left ventricular function while they receive beta-blockers. However, in exceptional cases when, in the opinion of the physician, concomitant use is considered essential, such use should be instituted gradually in a hospital setting. Diltiazem gives no protection against the dangers of abrupt beta-blocker withdrawal and such withdrawal should be done by the gradual reduction of the dose of beta-blocker.

Hypotension

Since diltiazem lowers peripheral vascular resistance, decreases in blood pressure may occasionally result in symptomatic hypotension. In patients with angina or arrhythmias using antihypertensive drugs, the additional hypotensive effect of diltiazem should be taken into consideration.

Acute Hepatic Injury

In rare instances, significant elevations in alkaline phosphatase, CPK, LDH, SGOT, SGPT and symptoms consistent with acute hepatic injury have been observed. These reactions have been reversible upon discontinuation of drug therapy. Although a causal relationship to diltiazem has not been established in all cases, a drug induced hypersensitivity reaction is suspected (see ADVERSE REACTIONS). As with any drug given over prolonged periods, laboratory parameters should be monitored at regular intervals.

PRECAUTIONS

Impaired Hepatic or Renal Function

Because diltiazem is extensively metabolized by the liver and excreted by the kidney and in bile, monitoring of laboratory parameters and cautious dosage titration are recommended in patients with impaired hepatic or renal function (see ADVERSE REACTIONS).

Pediatric Use

The safety of diltiazem in children has not yet been established.

Nursing Mothers

Diltiazem has been reported to be excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. Since diltiazem safety in newborns has not been established, it should not be given to nursing mothers.

Use in the Elderly

Administration of diltiazem to elderly patients (over or equal to 65 years of age) requires caution. The incidence of adverse reactions is approximately 13% higher in this group. Those adverse reactions which occur more frequently include: peripheral edema, bradycardia, palpitation, dizziness, rash and polyuria. Therefore, particular care in titration is advisable (see DOSAGE AND ADMINISTRATION).

Drug Interactions

Digitalis: Diltiazem and digitalis glycosides may have an additive effect in prolonging AV conduction. In clinical trials, concurrent administration of diltiazem and digoxin have resulted in increases in serum digoxin levels with prolongation of AV conduction. This increase may result from a decrease in renal clearance of digoxin. Patients on concomitant therapy, especially those with renal impairment, should be carefully monitored. The dose of digoxin may need downward adjustment.

Beta-blockers: The concomitant administration of diltiazem with beta adrenergic blocking drugs warrants caution and careful monitoring. Such an association may have an additive effect on heart rate, on AV conduction or on blood pressure. (See WARNINGS.) Appropriate dosage adjustments may be necessary. A study in five normal subjects showed that diltiazem increased propranolol bioavailability by approximately 50%.

Short and Long-acting Nitrates: Diltiazem may be safely co-administered with nitrates, but there have been few controlled studies to evaluate the antianginal effectiveness of this combination.

Other Calcium Antagonists: Limited clinical experience suggests that in certain severe conditions not responding adequately to verapamil or to nifedipine, using diltiazem in conjunction with either of these drugs may be beneficial.

ADVERSE REACTIONS

Angina

The safety of CARDIZEM CD, administered at doses up to 360 mg a day, was evaluated in 365 patients with chronic stable angina treated in controlled and open-label clinical trials. Adverse events were reported in 21.1% of patients, and required discontinuation in 2.2% of patients. The most common adverse effects reported were: first degree AV block (5.8%), dizziness (3.0%), headache (3.0%), asthenia (2.7%), bradycardia (2.5%), and angina pectoris (1.6%). The following percentage of adverse effects, divided by system, was reported:

Cardiovascular: First degree AV block (5.8%), bradycardia (2.5%), angina pectoris (1.6%), peripheral edema (1.4%), palpitations (1.1%), and ventricular extrasystoles (0.8%).

Central Nervous System: Dizziness (3.0%), headache (3.0%), asthenia (2.7%), insomnia (1.1%), nervousness (0.8%).

Gastrointestinal: Nausea (1.4%), diarrhea (0.5%).

Dermatological: Rash (0.8%).

Other: Amblyopia (0.5%).

The following additional adverse effects have occurred with an incidence of less than 0.5% in clinical trials: bundle branch block, ventricular tachycardia, ECG abnormality, supraventricular extrasystoles, chest pain, syncope, postural hypotension, paresthesia, tremor, depression, mental confusion, impotence, abdominal pain, constipation, GI disorder, epistaxis, nuchal rigidity, myalgia.

Hypertension

A safety evaluation was carried out in controlled studies in 378 hypertensive patients treated with CARDIZEM CD at doses up to 360 mg a day. Adverse effects were reported in 30.7% of patients and required discontinuation of therapy in 2.1%. The most common adverse effects were: headache (8.7%); edema (4.0%); bradycardia (3.7%); dizziness (3.4%), ECG abnormality (2.9%); asthenia (2.6%) and first degree AV block (2.1%). The following percentage of adverse effects, divided by system, was reported:

Cardiovascular: Edema peripheral (4.0%), bradycardia (3.7%), ECG abnormalities (2.9%), first degree AV block (2.1%), arrhythmia (1.6%), vasodilation (flushing) (1.6%), bundle branch block (0.8%), cardiomegaly (0.5%), hypotension (0.5%).

Central Nervous System: Headache (8.7%), dizziness (3.4%), asthenia (2.6%), somnolence (1.3%), nervousness (1.1%).

Gastrointestinal: Constipation (1.3%), dyspepsia (1.3%), diarrhea (0.6%).

Laboratory Tests: SGPT increase (0.8%).

Other: Leukopenia (1.1%), nocturia (0.5%).

The following additional adverse effects have occurred with an incidence of less than 0.5% in clinical trials: systolic murmur, supraventricular extrasystoles, migraine, tachycardia, increased appetite, increase in weight, albuminuria, bilirubinemia, hyperuricemia, thirst, insomnia, vertigo, nausea, pruritus, rash, increased perspiration, polyuria, amblyopia, tinnitus, and elevations in creatine kinase, alkaline phosphatase, and SGOT.

OVERALL CARDIZEM SAFETY PROFILE

In clinical trials of CARDIZEM tablets, CARDIZEM SR capsules and CARDIZEM CD capsules involving over 3300 patients, the most common adverse reactions were headache (4.6%), edema (4.6%), dizziness (3.5%), asthenia (2.7%), first-degree AV block (2.4%), bradycardia (1.7%), flushing (1.5%), nausea (1.4%), rash (1.2%), and dyspepsia (1.0%). In addition, the following events were reported with a frequency of less than 1.0%.

Cardiovascular: Angina, arrhythmia, bundle branch block, tachycardia, ventricular extrasystoles, congestive heart failure, syncope, palpitations, AV block (second- or third-degree), hypotension, ECG abnormalities.

Nervous System: Amnesia, depression, gait abnormality, nervousness, somnolence, hallucinations, paresthesia, personality change, tinnitus, tremor, abnormal dreams, insomnia.

Gastrointestinal: Anorexia, diarrhea, dysgeusia, mild elevations of SGOT, SGPT, LDH, and alkaline phosphatase (see WARNINGS), vomiting, weight increase, thirst, constipation.

Dermatological: Petechiae, pruritus, photosensitivity, urticaria.

Other: Amblyopia, CPK increase, dyspnea, epistaxis, eye irritation, hyperglycemia, sexual difficulties, nasal congestion, nocturia, osteoarticular pain, impotence, dry mouth, polyuria, hyperuricemia.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, erythema multiforme, exfoliative dermatitis, extrapyramidal symptoms, gingival hyperplasia, hemolytic anemia, detached retina, increased bleeding time, leukopenia, purpura, retinopathy, and thrombocytopenia. In addition, events such as myocardial infarction have been observed which are not readily distinguishable from the natural history of the disease in these patients. A number of well-documented cases of generalized rash, characterized as leukocytoclastic vasculitis, have been reported. However, a definitive cause and effect relationship between these events and CARDIZEM therapy is yet to be established.

SYMPTOMS AND TREATMENT OF OVERDOSAGE

Overdosage with oral diltiazem has been observed in 9 cases. Eight (8) patients recovered without sequelae over a few days. One patient who had ingested an unknown amount of diltiazem, tolazamide and alcohol experienced a fatal cardiac arrest. Doses ingested ranged from 1.8 to 10.8 grams. Bradycardia, AV block and hypotension were noted in most patients.

In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia

Administer atropine. If there is no response to vagal blockade, administer isoproterenol cautiously.

High Degree AV Block

Treat as for bradycardia above. Fixed high degree AV block should be treated with cardiac pacing.

Cardiac Failure

Administer inotropic agents (isoproterenol, dopamine or dobutamine) and diuretics.

Hypotension

Vasopressors (e.g., dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation.

DOSAGE AND ADMINISTRATION

Angina

Dosages for the treatment of angina should be adjusted to each patient's needs, starting with a dose of 120 mg to 180 mg once daily. Individual patients may respond to higher doses of up to 360 mg once daily. When necessary, titration should be carried out over a 7 to 14 day period. Patients controlled on diltiazem alone or in combination with other medications may be safely switched to CARDIZEM CD capsules at the nearest equivalent total daily dose. Subsequent titration to higher or lower doses may be necessary and should be initiated as clinically warranted. There is limited experience with doses above 360 mg, however, the incidence of adverse reactions increases as the dose increases with first degree AV block, dizziness, and sinus bradycardia bearing the strongest relationship to dose. Therefore, doses greater than 360 mg are not recommended.

Hypertension

Dosage should be individualized depending on patient's tolerance and responsiveness to CARDIZEM CD capsules. When used as monotherapy, usual starting doses are 180 to 240 mg once daily, although some patients may respond to 120 mg once daily. Maximum antihypertensive effect is usually observed after approximately 2 to 4 weeks of therapy; therefore, dosage adjustments should be scheduled accordingly. The usual dosage range studied in clinical trials was 240 to 360 mg once daily.

A maximum daily dose of 360 mg once daily should not be exceeded.

The dosage of CARDIZEM CD or concomitant antihypertensive agents may need to be adjusted when adding one to the other. See WARNINGS and PRECAUTIONS regarding use with beta-blockers.

Hypertensive patients controlled on CARDIZEM SR alone or in combination with other antihypertensive agents may be safely switched to CARDIZEM CD at the same total daily dose. Subsequent titration to higher or lower doses may be necessary and should be initiated as clinically warranted.

CARDIZEM CD capsules should not be chewed or crushed.

AVAILABILITY

CARDIZEM CD 120 mg capsules are supplied in bottles of 100. Each light turquoise blue capsule is imprinted with CARDIZEM CD 120 mg.

CARDIZEM CD 180 mg capsules are supplied in bottles of 100. Each light blue/light turquoise blue capsule is imprinted with CARDIZEM CD 180 mg.

CARDIZEM CD 240 mg capsules are supplied in bottles of 100. Each light blue/light blue capsule is imprinted with CARDIZEM CD 240 mg.

CARDIZEM CD 300 mg capsules are supplied in bottles of 100. Each light blue/light gray capsule is imprinted with CARDIZEM CD 300 mg.

Product Monograph available on request.

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The Canadian Neurosurgical Exodus, Crisis or Consequence?

Figures pertaining to the actual number of Canadian neurosurgeons and neurologists who have left Canada to practice in the United States are notoriously difficult to obtain. Nevertheless, over the past five years 50 to 90% of new graduates from Canadian neurosurgical residency programs accepted positions across the border. In 1992 some nine out of ten new graduates left. Simultaneously, at any given moment in Canada 15 to 25 neurosurgical positions remain unfilled.

In the year of the proclamation of the "Decade of the Brain" in the Canadian parliament (see Dean's Corner, this issue), it is ironic that this situation should prevail.

It seems clear that a number of disparate factors have conspired to produce in Canada conditions which favour the emigration of neurosurgical and other highly specialized medical professionals. As in many of the "socialized" European nations, Canadian Family Physicians enjoy freedom from excessive competition for patients and lifestyle, style of practice and remunerative options often unavailable to the very specialized, particularly in the surgical fields. Whereas the generalist enjoys opportunities to take on the desired workload in the desired location under a range of call conditions, the neurosurgeon must rely on the existence of an opening in close proximity to a fairly sophisticated operating room with a number of support services, such as a CT scanner and intensive care. Further, having located appropriate facilities, the neurosurgeon must obtain a reasonable number of hours of operating room time, and then fill those hours while providing on call coverage. If the neurosurgeon wishes to engage in research, laboratory space in a University must usually be obtained, pending availability of research grants, with a salaried arrangement on billings typically generous to the Faculty. Although exact salary figures are difficult to obtain in a society which prizes tact, the take-home of a neurosurgeon hired under recent conditions at Western would be in the two-hundred thousand dollar range. Well within reach of a moderately hard-working family practitioner. Not surprisingly, members of my own graduating class this year estimate that 70% of the class of 1995 wish to enter residency in Family Medicine.

While conducting informal research on this topic, I discovered that despite the obvious disparity in pay between Canadian and American neurosurgical practitioners (American neurosurgeons, in thousands per year, conservatively earn 400 $US, which is currently equivalent to 560 $CAN notwithstanding cost of living, taxation, and, in the other direction, malpractice insurance) the driving force behind the majority of expatriates is circumstantial. It appears to be much easier, not to mention possible, to obtain the desired practice conditions with research and support facilities in the larger American arena.

Consider epilepsy surgery. In this field, for which there currently exists a high and growing demand, the surgeon must have access to a dedicated neurologist/epileptologist, an epilepsy unit equipped with EEG and video monitoring, an MRI scanner and operating room for stereotactic electrode implantation, an anaesthetist trained in neuroanaesthesia, a laboratory in which to carry out neurostimulation experimentation, a nurse or nurse-clinician to perform routine patient care and co-ordinate follow up, and research grants to be able to enroll patients in studies of new anti-convulsant medications. The fingers of one hand would be quite sufficient to count the number of facilities equal to this task in Canada.

Farcically, government officials have so far responded to the problem by asking administrators in the neurosurgical division of the Royal College what *they* intend to do about it. And by officially proclaiming the "Decade of the Brain."

The underlying truth, however, may be that our system suffers from the same disadvantages consistently observed in all the socialized mechanisms of resource allocation in recent history: inability to respond to social and technological change, and to supply and demand forces. More ominously, socialized resource allocation by its nature is designed to produce a good, uniform standard, but not to exceed that standard. A sure recipe for decline in an advancing world. There is little doubt that Canadian neurology and neurosurgery possess the potential to fly to the moon and back. What remains to be found is the will to do so and the consequent enormous expenditure of resources.

Ross Mantle, Meds '95
Editor

Resurgence of Idealism in Medical Dramas

The beginning of the 1994-95 television season heralded the debut of three new dramas, all chronicling the lives of health care professionals. "ER" (NBC), "Chicago Hope" (ABC), and "Side Effects" (CBC) are the newest in a long history of medical television programs that began over four decades ago with "Medic".

In the early sixties, programmes such as "Dr. Kildaire" and "Ben Casey" gave us a new host of medical role models. Then in the seventies, we were entertained with medical dramas such as "Marcus Welby, M.D.", "Medical Center", and "M*A*S*H", and its spin-off, "Trapper John, M.D.". More recently, make-believe medicine has not been sufficient to satiate society's curiosities — every week we can watch a live operation, with commentary, on "The Operation".

The last time we were able to watch a serious medical drama was in 1988, when St. Elsewhere went off the air after six seasons. Now there are three dramas to chose from. The reasons for this resurgence of medical television are obvious. In the United States, the Clinton Administration's proposals for health care reform have put the medical profession in the spotlight. Issues such as physician salaries, accessibility of services, cost of expensive procedures and diagnostic investigations, and the inevitability of health care rationing have received incessant coverage by the media.

Society's fascination with physicians, however, goes beyond a simple interest in the provision of health care. Accordingly, "ER", "Chicago Hope", and "Side Effects" set their focus on the lives of the physicians themselves. By following every aspect of the characters' lives — both personal and professional — these programs continue the trend towards the demystification of the medical profession. They confront the inescapable paradox of fallible, flawed human beings with god-like expectations thrust upon them. Expectations that are impossible to live up to.

It has not always been like this. Forty years ago, "Medic" glorified the physician as "guardian of birth, healer of the sick, and comforter of the aged", reflecting a time when members of the medical profession were venerated. No other career choice garnered more respect and admiration; there was no greater joy than to have a son or daughter become a doctor (marrying a doctor was a close second). Times have changed. In the nineties, a career in medicine is simply a well-paying job. The aura and mystique surrounding the medical profession have all but disappeared. The new television programs reflect these new realities.

The news is not all bad, however. "ER", "Chicago Hope", and "Side Effects", for the most part, portray physicians as hardworking, empathetic, moral individuals who care more for their patients than they care for themselves. This portrayal is a refreshing change from the negative depiction of physicians that is ubiquitous in a media which emphasizes malpractice and sexual-harassment suits, physician-led protests against health-care reform, and lucrative "assembly-line" medical practices. Hopefully, these new dramas will continue to dramatize physician's positive attributes and promote the reemergence of the idea that medicine is a noble profession.

Jay Nathanson, Meds'96
Associate Editor

REFERENCES

Lacey L. The doctor is in. The Globe and Mail. Saturday, September , 1994. C1.

Marin R. Mixing speed and soap, angst and adranaline, 'ER' brings doctor shows back to life. Newsweek. October, 31 1994. 47-52.

LETTERS TO THE EDITOR

AUTHOR RESPONDS TO CRITIQUE

Dr. Paul Steinberg's letter to the Editor (UWO Med J 63(2):66) criticized my article, "Misadventures in Psychiatry," as offensive to clinical teachers and patients. I invite the reader to re-examine my article and then Dr. Steinberg's letter and decide which is more pejorative. Apparently, Dr. Steinberg is not aware that my columns are in the roman à clef tradition - persons and situations being all true but with names changed or omitted.

I would like to make it clear to Dr. Steinberg and to all Journal readers that he is not depicted in the article as a patient, nor as a psychiatrist.

Yours sincerely,
W. David Colby, MSc, MD, FRCP(C)
Chief, Division of Microbiology,
University Hospital.

NON-SEQUITUR DEBUNKED

I would like to make a few points with respect to the editorial comments inserted within the substance of a letter I had written as well as some comments directed towards me in the article entitled, "Naomi Wolf and Male Aesthetic Surgery." (UWO Med J 63(2))

Firstly, I would reiterate strongly that we made no attempt to censor the journal. The fact that four of the six articles in this issue were written without input from plastic surgery consultants (despite our offer of assistance) attests to the fact that we were superfluous. To use your words without sarcasm, there was "no censorship here, honest!"

Next, it is not my idea of aesthetic surgery that it treats people's neuroses with a scalpel but in fact that is the *stereotypical* idea of plastic surgery that much of the public has. Aesthetic surgery is a part of plastic surgery but my point was that it is not the whole.

Finally, the notion that somehow our objections arise from "paternalistic traditions" is an absurd *non-sequitur*. Male aesthetic surgery is routine and common. To suggest otherwise is misleading. Given that Webster defines "aesthetic" as "appreciative of the beautiful," it seems that penile lengthening is rooted more in a profound misunderstanding of the female sexual response than any attempt to improve the "aesthetics." Beauty may be in the eye of the (be)holder but Masters and Johnson may be of more help than the scalpel here.

I hope this serves to clarify some issues raised by your comments.

Sincerely,
Douglas C. Ross, MD, FRCSC
Dept. of Plastic Surgery,
St. Joseph's Health Centre. Ω

A NOTE TO OUR READERS

The Journal continues to grow in quality, variety and Faculty participation. A look at our new Editorial Staff for 1994-95 (page 3.) shows the high degree of involvement of first year medical students, a good sign for the future of the publication.

As medical students we continue to strive to produce a professional publication whose readership is primarily UWO Faculty. Equally important, though smaller in number are the medical students, residents, and clinical fellows. The latter two groups will once again be receiving a Journal free of charge, a state of affairs which has not existed for some years.

Though we are committed to provide two issues a year to all our target readership without charge, we request support from all Faculty participants in the form of a $17 voluntary subscription. This subscription aids the Journal financially and provides an opportunity for Journal supporters to be recognized on our list of friends and patrons, which is published in the fall issue. "Friends" of the Journal have paid the voluntary $17 subscription fee, while "Patrons" have contributed an additional, tax-deductible donation. Please send cheques to UWO Medical Journal, Health Sciences Bldg., University of Western Ontario, London, ON, N6A 5C1. Please include your return address so that we may provide your receipt.

We thank you, the readers, for your generous support in the past and look forward to the continuance of a relationship from which we can all derive learning and enjoyment.

Best regards,

Ross Mantle, Editor-In-Chief.
Ming-Ka Chan, Associate Editor
Jay Nathanson, Associate Editor.

Ω

DEAN'S CORNER

The Decade of the Brain

Dr. R.Y. McMurtry,
Dean UWO Faculty of Medicine

On Monday, November 14, 1994, Mrs. Sue Barnes rose in the house to speak to Bill C-39, an Act respecting the Decade of the Brain. In the address that followed Mrs. Barnes made a number of points including the following.

"There is no doubt the human brain is a remarkable feature that distinguishes us from other living creatures. A mere three pound mass of interwoven neurons control all of our activities. The brain is the most complex and mysterious wonder of creation. It has been called the seed of human intelligence, the interpreter of senses, the keeper of memory, in essence the sanctum of the soul. Ailments of the brain carry with them heart rending disabilities that can rob us of the very essence of personhood. A healthy mind is essential for living a full life with total liberty and independence."

This very eloquent speech underpins the tremendous importance of the brain and mind. In Canada we have an aging population. Increasingly, we need to seek alternatives to institutionalization of older Canadians. With the passage of time, age tends to be associated with problems afflicting "bones, bladder and the brain". In other words, there is an increasing disease burden relating to fractures, incontinence and dementia that will afflict our senior citizens and result in the need for admittance to hospital. The common denominator of all three is the need to retain cognitive function if there is to be hope of sustaining independence in people challenged in this way. The problem is monumental. It is estimated that it accounts for as much as 30% of hospital costs in Canada.

Clearly, an effective strategy for addressing this issue is necessary if we are to have any hope of keeping seniors functioning in the community.

At the other end of the age spectrum, there is increasing evidence that we are experiencing more and more problems regarding disadvantaged children. They live in poverty, and have problems of family dysfunction and maladjusted behaviour. There is also accumulating evidence that these problems are related to the first two years of existence, that is from the time of conception to the age of one to one and a half years. Normal brain development has been impaired in many of our children, especially those who are compromised socio-economically. The ill consequences of this arrested development have a long reach. There appears to be no effective remedy once these problems become established.

The basis of these developmental problems is just beginning to be understood but involves a complex interaction of the genetic make up of individuals and environmental influences such as nutrition and nurturing. This is indeed becoming a burning social issue that will have a major impact on Canada's future.

As yet unmentioned are the riveting problems of head injury, stroke, multiple sclerosis and central nervous system cancer. These are but a few of many afflictions that can strike the central nervous system. It is fair to state that these issues remain a public health problem in which the gap between what has been done and what could be done is very large.

It is a problem that has relevance to all of us engaged in the healing arts, as well as, the members of the community. Indeed, we should not rest until we can be more effective in giving comfort to those whose lives are compromised by central nervous system disorders.

Ω

CLASS REPORTS

Meds '95

by Jamie McNabb

So, how's everyone enjoying the middle ground on which we tread? Perhaps I should clarify. This installment is being written towards the end of our first elective block in fourth year - it's an interesting journey upon which we have embarked. Yes, yet another little jaunt down this endless road which we call medicine. (By the way, I've figured out this trip doesn't have a point of termination - sorry if I've been tardy in recognizing that fact.) Upon reviewing these last couple of sentences, I see how someone may interpret them as containing a sense of desperation, despair, perhaps even frustration. Well, I'm sure none of us feel this way, certainly not myself...well, maybe just a tad.

You see, I believe we're caught up in what you might call a state of suspended animation. First was the completion of clerkship - a modest sense of relief, if not something with which to whet our appetite for these fourth year electives. Yes, now we're knowledgeable, experienced, a full year of clerkship under our belts. (What's the dose for Ativan again?) Graduation and our first pay cheques mere months away. We're not just clerks, we're senior clerks - bow before us, knaves! Now, all that's left is to show just how keen we really are. Wait a minute. Didn't we just complete third year? But I digress. It's time to show those above us just how good we can be. (Sound like an ad for the military. Appropriate?)

So how do we prove ourselves even further, beyond levels we have already set? Must we compromise ourselves in such a way that we might be accused by our colleagues of "shmoozing?" I think not. By this stage, hard work and commitment are the keys to success. (Oh, my gosh, that was a good one Dr. Consultant! I'm in tears! I didn't even see it coming, not even after that "knock-knock" part. How do you have the time to be so funny and work so hard? I'm in hysterics - look, I'm slapping my knee. Do you see that, Dr. C. (as in Consultant) - I'M SLAPPING MY KNEE!) Yep, be focused. Do my job and do it well. (Oh, that's OK Dr. C., I was just washing your wife's car anyway. Yours will just take a minute longer.) Curiosity and initiative, and maybe just a dash of my own style. (You see, Dr. C., if I tie your shoelace just so, it won't come undone. Better yet, it won't tarnish the shine. Oops, wait a minute, I missed a spot. Let me get my shammy.) All right, perhaps a wee bit extreme, but you get the idea. Kind of like medicine's version of "The Name Game" - as yet an undetermined ratio of "what you know" to "who you know" - you pick the mix.

Back to that suspended animation idea. Doesn't it seem like there is a ton of stuff happening; an even bigger ton of stuff (that's for you, Dr. Cooper) that we actually seem to do each day, but no real sense of taking steps forward. I mean, here we are, concerned with performing well on our electives to help ourselves earn a residency position, and the CARMS bombardment never seems to cease. Additional letters of reference, call for this interview, wait for them to call you for that interview, look up that number on those little yellow cards that may or may not confirm that your file to date is complete. (That last part is particularly preposterous. I mean, I want to get into the program but I didn't realize it was a pre-requisite to memorize the CARMS code. A little haughty, aren't we? Mr. and Mrs. Carms? Put your bloody school's name on the damn thing! I've got better things to do. "Here you are, Dr. C., I thought some warm milk may help you sleep.")

Perhaps after the interviews some sense of direction will overwhelm us, or maybe just leave us utterly destroyed. That should be interesting - I'm sorry, what was the question - I was distracted while drowning in the tidal wave of sweat coming from my brow. "Pardon? A tremor? No, no...that's just that darn old early onset Parkinson's. Yes, you're right, that would explain my facial expression. Can I go now?" Well, it probably won't go that poorly, but who knows? So what if it does? I hear PUC is hiring on part-time staff.

I don't wish to drone on, so I'll sum up. Clerkship is over, and all kidding aside, I do feel more knowledgeable; well, maybe comfortable is a better word. Suffice to say, I'd go toe to toe on double jeopardy with that security guard any day. But much more formidable tasks lie a head. It will require a great deal of hard work and some well timed repartée with those at higher levels, but I truly don't believe it's just a game. I would hope medicine is a much more worthy profession.

By this stage, at the very least we know what constitutes an honest effort and a job well done, and somewhere deep within the bureaucracy of medicine, those things should be recognized. As for the interviews, if you want it, you've worked hard towards it, and are honest about your intentions, then it will happen. If they don't select us, then it's probably for the best it didn't happen. Whatever this is we're doing - it's not just spinning our wheels. It does have a purpose.

And if that doesn't instill some sense of comfort, call home. It never hurts.

Meds '96

by Jay Nathanson

Sunday, October 30, 1994. Almost there... Almost there...

By the time you read this, we will have soared above the tedious and mundane world of the classroom and entered the realm of admissions, intubations, and low urine output that is clerkship.

The last summer of our studenthood was well spent by many in our class. Some stayed in London, some went back home, some did research, and some did absolutely nothing. A few lucky '96ers broadened their horizons through travel. Classmates ventured into distant and exotic lands such as the Czech Republic, Greece, Spain, Italy, Iceland (however briefly), and Quebec. As well, after a year's delay, the Med Outreach crew finally reached Africa and spent a valuable and enjoyable eight weeks in Tanzania.

This summer was not all fun and games, though. Especially if you're referring to baseball games. For the first time in the history of baseball, there was to be no Fall Classic. The players' strike ended the World Series aspirations of the best team in baseball, the Montreal Expos. This was especially distressing to yours truly, who was spending the summer in La Belle Province and would have had regular access to season tickets! The only remotely positive result of the strike is that the Blue Jays, despite their dismal performance in this truncated 1994-95 season, remain reigning World Series Champions for another year. Not to be outdone by their richer cousins, the NHL players also began a strike which continues to this time.

Much nuptial and prenuptial activity occurred in our class this summer. Steve Korkola, Lisa Miceli, Colleen Harvey, and Faizal Bawa are now engaged to be married, while Joe Kim, Cathy Longley, and Mike Horsey went the next step and tied the knot. Congratulations also go out to Ron Curtis, who became the proud father of a baby boy in the last weeks of Phase II. Finally, best of luck to William Tinmouth who is taking a brief leave of absence from medicine to pursue his rowing career. At least we know that there will be one person who will be getting up earlier than we are.

The school year began rather anticlimactically — ICC 4 not being exceedingly different from ICC 1, 2 or 3 (merely more of the same in considerably less time). The USLME, however, did manage to add some excitement to the lives of the courageous and diligent few who attempted the exam.

September also heralded the beginning of a new television season which included three series chronicling the lives of medical professionals: "ER", "Chicago Hope", and "Side Effects". The unofficial winner so far seems to be ER, with its plethora of medical jargon and interminable stream of multiple medical conditions. I suppose we simply don't get enough of it during the rest of the week.

The post-exam/pre-clerkship week was a well deserved calm before the storm. Our medical jurisprudence lectures forewarned us of such hospital hazards as litigious patients and poorly transcribed patient records. Our IV training sessions left us better prepared for drawing blood and starting IV lines, if not a little sore. The Halloween bash allowed us one final orgiastic release of tension before the abyss, and the trip to Stratford to see Burgess's adaptation of Rostand's "Cyrano de Bergerac" allowed for a few well-cultured classmates to proceed into the clerkship in a more cerebral fashion.

Well, that's all for now. By the time this is read, Phases I, II, and III will be nothing but bad memories. Clerkship awaits. Best of luck to us all.

Meds' 97

by Sue Huh and Sonny Bhalla

Fill in the blanks with the given choices:

Dear _____(mom, dad, great-aunt, bank loan officer),

How is life in _____(Toronto, Ottawa, Romania, other)? Med school is _____(great, morbid, old). I guess we haven't talked since last spring. My finals last year were a chance to _____(demonstrate my deep understanding of biochemistry, prove I could go two weeks without sleep, show that grown men/women

do cry). The summer was _____(short, relaxing, no different from the rest of the year, all of the above). I had the wonderful opportunity to _____(travel around Europe working on my tan, travel around campus working on my research project, drink beer at the cottage).

Second year so far has been _____(more interesting, more stressful, a bad case of deja vu). The year started off with quite a bang. I know I attended but I don't quite remember _____(the Pub Crawl, Fanshawe Park, Oktoberfest, Health Sciences Pub, all of the above). I had a chance to cultivate my creative side while I _____karaokeed, ballroom-danced, created my Halloween costume). Being involved in activities like _____(Med Outreach, the Healthy Responsible Sexuality Project, Med Students for Social Responsibility) has taught me that doctors not only have a lot to give to the community, but a lot to learn from the community. Oh yeah, some guy in our class revived the old Osler Society.

While not studying or in class (too much of the time), I've been involved in _____(softball, volleyball, ultimate, soccer) to try to stay in shape. But now I'm studying harder because exams are just around the corner. I'll see you during the holidays with _____(bags under my eyes, packed bags, the smile of relief). I can't wait _____(to see you, to get my gifts, until The Ridout on Dec. 5).

Love,

_____

(sign here)

Meds '98

by Rebecca Stulberg

Somebody pinch me, I have GOT to be dreaming! Is THIS medical school...? It can't be... this is actually FUN!

From the very first day, we knew we had a special group of students. We knew that MEDS 98' would kick butt with no problem. After several inspirational speeches by esteemed faculty members, we shared a Blue Light with the Dean, and kicked off our week of celebrating (please note that alcoholic beverages were available at all events, so you didn't HAVE to feel pressured to drink pop).

At the Gov'Nor we were a bit apprehensive, who WERE all of these first borns? Four years with THEM? Where do you insert a rectal thermometer?

At the PUB CRAWL, 'Tour of Duty', we warmed to each other. We laughed, we danced, Mike Rooney pierced his ear with a thumbtack, and we made it through 11 rounds. It is now apparent that we will be wonderful physicians AND alcoholics. Special thanks goes out to Anthony Cook and Mike Crouzat for the funny, funny trick they did with those plastic beer pitchers and their pyloric sphincters. You two are a bundle of laughs. Really.

By the time Fanshawe rolled

around, we realized that we really liked one another, albeit some pairs more than others (no specific mentions here—I don't gossip). Fanshawe was a night of eating, dancing, drinking and SWIMMING. What was that about the E. Coli count? Most memorable was the boat race at the end of the night. The 98's were great, the 97's CHEATED, and the 96's unimpressive. It's obvious from this bitterness that we came second. We'll win next year, and THAT is an official challenge to YOU, MEDS 97'.

Special thanks goes to Steve Herr and Mike Crouzat who were the most awsome orientation leaders we could have wanted. You made our induction into medical education a pleasure. We love you. Are you twins?

The start of classes marked the beginning of several new experiences. Seven AM awakenings (oh my god), comprehension at 8 AM (good luck), handwriting, and coffee (isn't there an EXTRA jumbo size?). We also met our big brothers and sisters, who carefully navigated us through the first days of school and the titilating experience of buying books. Next, we bought instruments (mine's green, what colour is yours?), for which OSAP will be paying dearly.

Then there were the classes... Physiology, Anatomy, Biochemistry, Embryology...the first day of Anatomy Lab.... We were forced to admit that the real reason we were in medical school was to LEARN (no kidding?). I think we all agree that the professors are great, some we could say are entertaining— what with Gamal and Haines taking off their clothes in front of the class... Exhibitionist tendencies, must be. Seriously though, the pace is slowly picking up.... anyone else notice?

And then there was the Homecoming float.... which was seemingly thrown together with BORROWED hospital equipment, cardboard, toilet paper and string. Actually, it looked really good! The premise was that it was the EMERG, UofT player's entrance. For those of you who failed to wake up at six the Saturday of Homecoming weekend, we had injured football players in wheelchairs, surgeons, defibrillations, blood and guts. Special thanks to Andy and Chuck for organizing and excecuting the whole job, and to all that showed up to brave the SUB ZERO temperatures Friday night and Saturday morning. (We had a pretty good way of warming up, though, didn't we?)

Last but not least were the class elections. Better late than never, they provided the class with many tough decisions to make. Elected were: Chuck Su: President, Andrew Thompson: Vice-President, Anita Kadikar: Secretary/Treasurer, Tracey Andrews and Dave McKinnon: Social convenors. Best of luck to the 98' executive, work hard for us and we may let you live.

And so now it's now... exams are approaching... seems like the class attitude is (or was when this was written) pretty relaxed. Let's stay lucid, guys, the year will be a long one, especially with no hockey (no offence, Craig).

So, now we have the gold-plated key to the door leading to fulfillment, happiness and complete social acceptance (at least we think). We are content, we know where we came from and where we are going. We are satisfied. One burning question plagues us though — what the hell is a PREEMITIV STRIK? Ω

ALUMNI NEWS

The first edition of MEDS, the official UWO Faculty of Medicine Alumni newsletter, is expected to be out before Christmas. The newsletter is a project of Martha Powell, the Alumni Development Officer within the Dean's Office.This issue of MEDS includes a discussion on Faculty Development, a message from our Dean as well as information about the Health Intelligence Unit (HIU). The HIU is a liason network that encourages communication between faculty, health units and the community in an effort to share resources and information. Dr. Evelyn Vingilis is the Director of the Health Intelligence Unit.

Homecoming at Western, held every October, is a popular event especially for the Faculty of Medicine. During this weekend event, the Faculty holds its alumni reunion going back every five years. In October 1995, the graduates of Meds '90, '85, '80... will reconvene at Western to reminisce, receive awards of recognition and participate in a Continuing Medical Education conference. Traditionally, the fiftieth anniversary alumni and recent graduates are invited to a Senior Meds Luncheon held by Western's President and Dean of Medicine.This luncheon is an opportunity for the University to officially pay tribute to our longstanding alumni. Specific Alumni reunion information is available through Western's Alumni Gazette.

The graduating class of Meds '94 are well into their respective residency programs.A compiled list of the new homes of our most recent graduates is included for your information. Ω

University of Western Ontario

Anaesthesia
Sanjiv Mathur

Diagnostic Radiology
Justin Hugo Amann
Daniel W. Struk

Family Medicine
David John Bricker
Kyle W. Brydon
Paula Ann Dozzi
Marc A. Fischer
Renata J. Frankovich
Sean L. Gartner
Trevor N. Glazman
Diane B. Humphrey
Julie Ann Jenner
David Kim
Stephen I. Lai
Jeffrey Scott Main
Raymond G. Marier
Hans Roy Marquardt
Simone Meikleham
Gary R. Neumann
Julia Edna Wilhelm
Maria Chin Yun Yu
Norman B. Yu
Alexander Zilberman

General Pathology
Kenneth W. Alanen

General Surgery
Margherita Cadeddu
Glen D. Hooker
Santosh Pudupakkam

University of Western Ontario - cont. . .

Internal Medicine
Karen E. A. Burns
George K. Dresser
Dana Cecelia Jerome
Albert Kadri
Edward J. Lawson
Thomas J. Nolan
William R. Plaxton
Gregory Tsoi
Sue A. Quan
Lisa Y. Yip

Neurosurgery
Balraj Singh Jhawar

Obstetrics and Gynaecology
Denise L. Campeau
Deborah A. Penava

Orthopaedic Reseach
Faisal Mirza
Zane Zarzour

Orthopaedic Surgery
Christopher Lindsay
Ian King Y. Lo

Paediatrics
J. Burke Baird
Alexander C. Barron

Plastic Surgery
Abhay Gupta

Psychiatry
Lisa Anne Bogue
Paul L. Eshleman

Radiation Oncology
Ingrid M. Tiessen

Urology
Jonathan I. Izawa
Jonathan V. B. Riddell

University of Toronto

Anaesthesia
Naseer Baig
Robert Walter Mohl

Emergency Medicine
Ivy Shu-Shan Cheng
J. Douglas Chisholm

Family Medicine
Adilson Araujo
John Depaoli
N. Scott Gledhill
Tammy G. Hermant
Aaron Ay-Lunn Low
Ara S. Makdessian
Eena Sachdev
Karyn Ruth Sheldon
Alan Larry Shievitz

Neurosurgery
Michael David Taylor

Obstetrics and Gynaecology
Lisa Ann De Vries
Shobhana J. Patel

Orthopaedic Surgery
Vaselios Manolopoulos

Plastic Surgery
Leila Kasrai

University of Ottawa

Family Medicine/Civic Hospital
Gillian L. Buckley
Caroline B. Ehrat
Catherine M. Motz
Amit Pravin Shah

Family Medicine/General Hospital
Lesley Ann Horton

University of Ottawa - cont. . .

Family Medicine/Sudbury
Mark S. Mensour
Theordore Mitchell

General Surgery
Daniela G. Filippone
Kamyar Kazemi

Obstetrics and Gynaecology
Liisa Lyn Honey

Ophthalmology
Thomas Johnson

Psychiatry
Brian Neil Talarico

McGill University

Anaesthesia
Michael A. Breden

Diagnostic Radiology
Dennis Zander

Family Medicine
Paul Alan Druzin

Internal Medicine
David N. Fisman

Otolaryngology
Geoffrey Mark Fine

University of British Columbia

Anaesthesia
G. Michael A. Solylo

Diagnostic Radiology
Anne Margaret Silas

Family Medicine/Urban
Donna Jean McClure

Family Practice - Community Base, Family Practice/Rural
R. Stephen J. Burnett

Neurology
Jeffrey Mark Politsky

Obstetrics and Gynaecology
Harold Gordon Hunt

University of Manitoba

Family Medicine
Robin Gray

Neurology
Zoe Arvanitakis

Queen's University

Family Medicine
Ellen Hon-May Choi
Shawn Ross Edwards
John C. MacDonald

McMaster University

Diagnostic Radiology
Katherine A. Klein

Internal Medicine
Caroline A. Lohrisch

Paediatrics
Ramsay G. MacNay

Dalhousie University

Obstetrics and Gynaecology
M. Christine Hatfield

Family Medicine
Nina Makkar

University of Alberta

Internal Medicine
Sheryl Lynn McCuaig

Obstetrics and Gynaecology
Gulnaz Jiwa

University of Calgary

Internal Medicine
Gregory B. Schnell

Cleveland Clinic Foundation

Internal Medicine
Peter Jon Mazzone

Charles Clifford Macklin (1883-1959), A Historical Overview.

by Ming-Ka Chan, Med'95

Charles Clifford Macklin, pioneer Canadian pulmonologist, was born on October 23, 1883 in the village of Todmorden, near Toronto. Dr. Macklin's research in lung structure and function extended over 35 years, much of this time spent at the University of Western Ontario Faculty of Medicine. His most renowned work was his wartime investigations of alveolar size among mammalian species. The following is a historical overview of Dr. Macklin's work based on research by Dr. Norman Staub, MD, physiologist at the University of California San Francisco.[1]

Dr. Macklin's educational background was at the Central Business College of Toronto before entering the University of Toronto Medical School. He graduated in 1914 with honours and won the first James H. Richardson Fellowship for research in anatomy. The publication of his description of the skull of a 40 mm human fetus in 1914 prompted Franklin P. Mall, Chairman of the Anatomy Department at Johns Hopkins Medical School, to offer him a position as an instructor in anatomy.

After moving to Baltimore, Dr. Macklin met his wife, Madge DeGrofft Thurlow. She was then a freshman medical student. They were married in 1917 and had three daughters. Dr. Madge Macklin became internationally recognized for her work on the hereditary and genetic basis of human disease. She was one of the first investigators to establish a genetic factor in breast cancer and later became President of the American Society of Genetics.

During the first World War, Dr. Macklin was refused when he tried to enlist in the Canadian army because of a minor congenital foot deformity. He did serve as a contract surgeon for the US Army. He worked in the neurosurgical laboratory at John Hopkins university under Professor Louis Weed with a focus on brain trauma and repair.

In 1921, Dr. Macklin was recruited to Western University by the Paul McKibben, the Dean of the Faculty of Medicine, as Professor of Histology and Embryology. Interestingly, his recruitment was reportedly an attempt to improve Western's quality as it had been recommended for closure in the 1910 Flexner Report on Medical Education. With the recruitment of Dr. Macklin and others, Western's Medical Program became recognized as a first class facility after the Second World War.

Dr. Madge Macklin also came to Western, becoming well known as an exceptional instructor in Embryology and Histology. Charles Macklin unfortunately was an ineffective teacher with a reputation for difficult exams and a belief in self-directed learning.

It was also in 1921 that Dr. Macklin switched to lung research from his original investigations of brain injury, cellular division and bone development and repair. His first lung paper was entitled "Elastic membrane of the bronchial tree and its functional significance." He followed this with an extensive review, "The musculature of the bronchi and lungs." This review is the keystone to the modern understanding of the structure and function of airways and of lung expansion during breathing.

In the mid 1930's, Dr. Macklin published his research on the effects of lung overinflation and continued this renowned research on interstitial emphysema until 1944.

In 1938, Sir Frederick Banting toured Canadian universities to determine their preparedness for war related research. He found Charles and Madge Macklin's laboratory one of the few that were adequately prepared. However Dr. C. Macklin's grant application for research on alveolar epithelial damage was initially rejected. In 1940, Dr. Banting awarded $5000 to Dr. Macklin, one of the largest grants given by the National Research Council of Canada at that time.

Charles and Madge Macklin's lab consisted only of themselves, Stan Hartcroft (Dr. Macklin's only research fellow), Charles Jarvis (histology technician) and Edna Cunningham (a typist). The focus of their wartime research was on the quantification of alveolar size in various mammalian species which was later published as a series of short articles in *Transactions of the Royal Society of Canada*. Part of their findings included a correlation between alveolar dimensions and body size. Dr. Macklin felt this correlation reflected the exercise performance of different species. This hypothesis later became useful in his work on alveolar surface tension and surfactant.

By the mid 1920's, the Macklins began to have difficulties with the administration at UWO. These difficulties were intensified when George Hall became Dean and Madge Macklin's services were terminated. She then moved on to Ohio State University. Several months later, Charles Macklin was replaced as head of Histology by Dr. Murray Barr (best known for his discovery of the Barr body) and made Research Professor of Experimental Histology. Despite these conflicts, Dr. Macklin's never accepted a position elsewhere, and his research continued to flourish. He discovered alveolar and extra-alveolar vessels. He also realized the link between breathing and blood circulation, calling the lungs an "accessory heart," a concept which was to become crucial in the modern application of cardiopulmonary resuscitation. In 1950, he summarized 15 years of research in a landmark review, "The alveoli of the mammalian lung."

Dr. Macklin retired in 1953 but still continued his research, publishing three major papers on the function of the type II pneumocyte. His last publication involved the preferred sites of broncogenic carcinoma in relation to the airway concentration of particles during clearance.

In 1959, Charles Macklin was found slumped over in his seat by the driver of a bus in which he was a passenger. He was rushed to Victoria Hospital and pronounced dead. He was buried at Ebenezer Church near his childhood home. In 1962, Madge Macklin died and was buried beside him

REFERENCE

1. Staub, NC.. Charles Clifford Macklin (1883-1959): Pioneer Canadian Pulmonologist. Can Respir J. Fall 1994; 1(3):199-207.

Ω

The Use of Non-Medical Criteria in the Allocation of Scarce Medical Resources

by Jay A. Nathanson

Canadians take equal access to health care for granted. We believe that persons have a moral right to health care, based on principles of equality and justice.[1] However, it is becoming increasingly obvious that the government cannot provide for all the health care needs of all Canadians.[2] The cost of health care continues to rise dramatically. In 1965, health care made up 6.1 percent of the Canadian GDP, in 1980, 7.4 percent, and in 1986, 8.5 percent.[3] Methods for improving the efficiency of the health care system, such as better management, elimination of redundancies, abandonment of useless procedures, and reduced fees will only lead to temporary improvement.[3] Rationing of health care may be inevitable.

Health care rationing is "a system of deliberate choices about the sharing of health care resources among persons (i.e. who gets what care, and in what order of priority) on grounds that go beyond an individual patient's clinically defined needs; the criteria specifically include both comparative medical need and social equity."[4] Rationing in health care is necessary because of the scarcity of natural resources (e.g. donor organs) or due to financial constraints on the health care budget.[5]

The spectre of rationing is met with apprehension and consternation at all levels of the health-care system.[5] Politicians are forced to decide between controlling spiralling spending on health care and the public's vocal demands for high-calibre health care. Physicians resent the infringement into their relationships with their patients by a third party who demands that the best interests of their patients be compromised in order to reduce costs. Most importantly, patients worry that essential medical services will be denied.

The main ethical question for all those involved is: On what basis do we decide who is given access to certain medical resources and what specific resources should be made available? In some cases we may even have to decide who lives and who dies.

There are many methods by which we may select patients for access to scarce medical resources (SMR). Many are based on medical criteria alone, and take into account such factors as risk of imminent death, likelihood of benefit, length of benefit, and quality of benefit. When making life and death decisions, medical concerns are certainly important. However, health care does not occur in a vacuum and there are more intangible criteria that may also merit consideration.

This essay examines three prominent non-medical rationing criteria: social worth, extent of personal responsibility for the illness, and the age of the patient. I also examine the possible use of random selection (i.e. no substantive criteria at all) to ration access to SMR. Finally, I discuss each method's moral acceptability and relevance to Canadian society and present my conclusions on what will be required of both physicians and patients if fair, effective, and efficient rationing is to succeed.

ABOUT THE AUTHOR:

Jay Nathanson is a third year medical student at the University of Western Ontario with an interest in medical ethics. He has authored several papers on the subject in previous issues. Mr. Nathanson's career plans at the present time include neurology, neurosurgery, or internal medicine.

Rationing based on "social worth"

This criterion for health care rationing takes into account patients' social responsibilities as well as their societal contributions. Despite the extreme difficulty involved in evaluating a patient's "social worth", this method merits some consideration. Most other methods of rationing look at the patient's life exclusively from the perspective of the patient. This method acknowledges the importance of a person's life to those around him or her - family, friends, associates, neighbours, colleagues, society at large, and so on.[6] It also recognizes and takes into consideration the effect, both emotional and financial, that an illness or death will have on the patient's spouse, children, or parents. Other things being equal, this method would give treatment priority to a cleric over a convict, a member of state over a homeless vagrant, a mother and wife over a childless bachelor.

Since society is paying for patients' medical treatment, their potential for societal contributions are important factors when determining access to SMR. Those who will make the greatest possible contributions are most likely to benefit. In other words, the future "return" is what determines how the "investment" is made.[6] In addition, fairness obligates society to recognize and "reward" those who have made contributions in the past. Through the potential for greater future access to SMR, this method encourages individuals to contribute to society to the best of their ability.[6]

I do not believe that social worth is a morally acceptable criterion for allocating SMR. Such decisions violate patients' autonomous status as persons by making them means in the realization of a greater social good rather than ends in themselves. As well, the physician-patient relationship would be irreconcilably damaged if physicians chose patients for SMR on the basis of 'social worth'.[7] To maintain the crucial level of trust inherent in the relationship, physicians must act as absolute advocates for their patients. To declare any patient socially inadequate or socially inferior to another patient would be an obscene violation of that trust.

Rationing based on unhealthy lifestyle choices

Coronary artery disease (CAD) and alcoholic liver disease (ALD) are two common examples of illnesses linked to or exacerbated by voluntary lifestyle choices (smoking and excessive alcohol consumption, respectively) despite vast public information about the health risks of these behaviours. Therefore, rationing could be accomplished by giving patients who require SMR (e.g. coronary artery bypass grafting, liver transplantation, etc.) for such "avoidable" conditions lower priority than patients who require the SMR through no fault of their own.

There are several reasons why this method may be morally acceptable. One of the most integral presumptions of the basic right of equal access to health care is the indiscriminate nature of illness and well being.[1] Our genetic makeup, which determines our susceptibility to disease, the performance of our organs, and how we react to foods, chemicals, allergens, and other external agents is beyond our control. In addition, we are, for the most part, powerless to avoid accidents that require medical treatment. However, when patients choose to lead unhealthy lifestyles, the resultant morbidity is avoidable and the aforementioned presumption is no longer applicable. Therefore, these patients, by voluntary choice, abrogate their right to the same health care as everyone else. To not take this factor into account violates the principle of equality and justice.[1]

Rationing based on unhealthy lifestyle choices can also be justified if health care is regarded as a three-way contract between society (who pays for the cost of health care), the individual (who benefits from health care), and the physician (who provides the health care). The ultimate goal of this contract is to work towards the patient's best interests without putting an excessive financial strain on the rest of society. Subsequent unhealthy behaviour constitutes a breach of that contract because the patient chooses to behave in a manner that will add an avoidable burden to health care resources.

Further support for rationing based on personal responsibility for health can be found in three principles of distributive justice: Distributive justice requires (1) that each person receive an equal chance to reach a desired goal, (2) similar treatment for similar cases, and (3) treatment according to individual effort.[8] Consequently, from the first principle, a child suffering from biliary atresia should be given an opportunity for a first liver before a patient with ALD is given a chance at a second one.[9] According to he second principle, patients whose illnesses are preventable are not equal to others, and it is acceptable to treat them differently.[9] Finally, the third principle holds the individual personally responsible for maximizing his or her own health and averting the complications of the unhealthy behaviour.[9]

I have significant difficulty accepting unhealthy behaviour as a rationing criterion for SMR. If smokers and alcoholics are punished for their behaviours because they are 'preventable', would the same be true for obese people who eat to excess and lead a sedentary lifestyle, promiscuous patients with AIDS, pregnant women who didn't use contraception seeking abortions, accident victims who weren't wearing seat-belts, athletes with sport-related injuries, victims of skin cancer who spent too much time at the beach or tanning salon, attempted suicides, and so on? Although it may seem an attractive method of rationing at first glance, this *reductio ad absurdum* highlights the futility of attempting to ration based on unhealthy behaviour.

Age-based rationing

The elderly population of the world is growing at an extraordinary rate. At the beginning of this century, the elderly constituted only four percent of all Canadians. Today, twelve percent of the population is over sixty-five, and this number is expected to reach twenty-five percent by 2035.[10] As well, the aged consume a disproportionate amount of health care resources compared to younger members of society. The U.S. federal government spends six times as much on health care for those over 65 as for those under 18, with one percent of the GNP spent on health care for elderly persons in their last year of life.[11] Therefore, the cost of health care can be significantly reduced through the use of age as a criterion for the allocation of SMR. That is, the older a patient is, the more likely he or she is to be denied access to SMR.

Can discrimination based on natural factors beyond one's control ever be justified as a means of allocating SMR? I believe that it can. A crucial difference between ageism and other forms of discrimination, such as racism and sexism, is that while people eventually change from young to old, race and sex (almost invariably) remain constant. In other words, "young birth cohorts age and are transformed into older age-groups".[12]

With this in mind, we should consider the health care that patients consume throughout their lives, rather than at a particular moment in time. When considering what distribution of health care they would want over all stages of life, most people would choose to have the greatest access to SMR in their earlier years in order to improve the chances of reaching a normal lifespan. When this same reasoning is applied to the consumption of health care by all members of society, the result is a form of age-based rationing. As a result of this "lifetime health care package",[13] when a certain age is reached, the elderly, along with their families and physicians, would accept the limits on access to SMR of age-based rationing.

Rationing by random selection

This method is a reaction to all other rationing criteria. The premise of this method is that no one should have the right to 'play God' and that all selection criteria are therefore unethical. Consequently, the only just way to select who benefits from SMR is through randomness or chance — a 'first come, first served' basis or a lottery. The advantage of such a system is that it "preserves a significant degree of personal dignity by providing equality of opportunity".[7] Random selection is free of the value-laden judgements inherent in decisions based on other criteria - it is the ultimate equalizer.

From the perspective of the patient, being denied access to SMR through random selection is less psychologically traumatic than being rejected due to subjective judgements.[6] Patients believe that they have the same 'right' to SMR as everybody else. Therefore, if rationing is to occur, they would want to have the same 'chance' as everybody else. If they lost the 'SMR lottery', they would be unlucky, but would not feel that they had been unfairly treated.

The main weakness with this method of selection is that it is too equal. It makes absolutely no distinctions between patients where, morally, distinctions should be made. Rationing by random selection allows those who must make the decisions to make no decision at all. Relevant medical and ethical criteria such as probability of survival and quality of life after receiving the SMR are ignored.

Discussion

I find rationing based on social worth, unhealthy lifestyle choices, or random selection morally deficient criteria for rationing health care. Rationing based on social worth would lead to irreparable harm to the physician-patient relationship. More importantly, it reduces patients to means to an end for the benefit of society — a philosophy taken to the extreme in the racial hygiene programme of Nazi Germany. Rationing based on unhealthy lifestyle choices is inadequate because, upon close consideration, it requires that health care be denied to a considerable portion of the population. Finally, rationing based on blind chance is faulty in that it unjustly fails to consider any difference between patients, even in cases where differences are relevant.

Of the four criteria mentioned above, only age-based rationing is, to me, morally acceptable. This method, although discriminatory towards those in their late years, treats all patients equally when health care needs are considered for an entire lifespan, rather than at a specific point in time. Acceptance of this method of rationing will not come easily to Canadians. Important changes will have to be made with respect to how physicians and patients view the goals of medicine. Most importantly, we must reconsider our attitudes and outlook with respect to old age and dying.

Society currently thinks of old age as a disease, to be confronted and overcome through modern medicine and technology. The goal of medicine in the care of the elderly and the terminally ill should be the improvement of quality of life, rather than merely the

extension of life. We would return to the idea that there is a "natural life span", independent of medical technology and skill, and work to enhance life, not to prolong it indefinitely at the end. Thus, medicine would be concerned with maximizing the chances of reaching the "natural life span" and with minimizing suffering thereafter.[11]

The road to an equitable and efficient health care system will be a long and difficult one. Health care providers and administrators must achieve a greater understanding of disease and health care needs.[14] Physicians must abandon their hopes for infinite medical progress and put an end to the modern myth that "every disease should be cured, every disability rehabilitated, every health need met, and every evidence of mortality, especially aging, vigorously challenged".[15] Society must come to accept that unlimited access to health care can never be a reality, and that some may have to go without. Unpleasant ethical, economical, political, social, and organisational choices will have to be made, but these are the essence of rationing.

REFERENCES

1. *Kluge EH. Drawing the ethical line between organ transplantation and lifestyle abuse. Can Med Assoc J 1994; 150 (5): 745-6.*
2. *Lowy FH. Restructuring health care: rationing and compromise. Humane Medicine 1992; 8 (4): 263-7.*
3. *Aaron H, Schwartz WB. Rationing health care: the choice before us. Science 1990; 247: 418-22.*
4. *Reagan MD. Health care rationing. What Does it mean? New Engl J Med 1988; 319(17): 1149-51.*
5. *Singer PA, Lowy FH. Rationing, patient preferences and cost of care at the end of life. Arch Intern Med 1992; 152: 478-80.*
6. *Rescher N. The allocation of exotic medical lifesaving therapy. In: Mappes TA, Zembaty JS, editors. Biomedical Ethics. 2nd ed. Toronto: McGraw-Hill Book Company, 1986: 601-11.*
7. *Childress JF. Who shall live when not all can live? In: Mappes TA, Zembaty JS, editors. Biomedical Ethics. 2nd ed. Toronto: McGraw-Hill Book Company, 1986: 611-20.*
8. *Beauchamp TL, Walters L, editors. Contemporary Issues in Bioethics. 3rd edition. Belmont: Wadsworth Publishing Company, 1989: 33.*
9. *Moss AH, Siegler M. Should alcoholics compete equally for liver transplantation? JAMA 1991; 265 (10): 1295-8.*
10. *Katz S. Growing number of elderly patients will mean more ethical issues for MDs, conference told. Can Med Assoc J 1992; 147 (8): 1239-41*
11. *Callahan D. Aging and the ends of medicine. In: Arras JD, Rhoden NK, editors. Ethical Issues in Modern Medicine. 3rd ed. Mountain View: Mayfield Publishing Company, 1989: 533-9.*
12. *Daniels N. Am I my parents' keeper? In: The President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research. Securing Access to Health Care, 1983; Vol. 2, Appendix K: 265-91.*
13. *Childress JF. Ensuring care, respect, and fairness for the elderly. In: Arras JD, Rhoden NK, editors. Ethical Issues in Modern Medicine. 3rd ed. Mountain View, CA: Mayfield Publishing Company, 1989: 526-32.*
14. *Heginbotham C. Rationing. BMJ 1992; 304: 496-9.*
15. *Callahan D. The way to affordable health care. New Engl J Med 1990; 322 (25): 1810-3.*

Ω

Nutrition and Socioeconomic Status: Results from the Ontario Health Survey

by Christine Hatfield, BScH, MD (Meds '94)

INTRODUCTION

The Nutrition Canada National Survey (1970-72) [1] was the first nationwide survey to examine the nutritional status of Canadians. Since then, other national surveys have included questions on nutrition in their design (Canada Health Survey 1979 [2], Canada Fitness Survey 1981 [3]), but not in as much detail as the Nutrition Canada National Survey. In 1990, the Ontario Ministry of Health conducted the Ontario Health Survey (OHS) to collect information on the health status and health-related behaviours of Ontario residents. An extensive nutrition section provides us with the most detailed overview of nutritional status of this population since the 1970's.

Using data from the OHS, we examined the dietary practices of Ontario residents, asking three questions: (1) how closely does dietary intake follow the nutrition recommendations for the four food groups, fibre and fat; (2) are there variations observed with socioeconomic status (SES) and (3) which SES factors, if any, have the greatest impact on nutrition.

MATERIALS AND METHODS

The OHS used a sample of households from selected geographic clusters across Ontario (multi-stage, stratified cluster design) [4]. The target population consisted of residents of private dwellings, excluding residents of Native Reserves and remote areas, and inmates of institutions.

The survey consisted of two parts: (1) a personal interview with one member of each household who provided information for all household members and (2) a self-administered questionnaire completed by all household members over the age of 12.

Dietary intake was measured with a food frequency questionnaire in the self-completed section. It asked if and how often specific foods were eaten in the past month, and average serving sizes.

We divided the sample into four age groups: adolescents (12-19), young adults (20-39), adults (40-64) and elderly (65+). We also separated the sample into men, non-pregnant women and pregnant women. Three measures of socioeconomic status were used in the analysis: *income* adjusted for household size and stratum (urban vs. rural), *education* and *source of income* (Table I).

Table I
Definition of SES variables

Variable	Definition
Income	
Low Income	0-11,999 or 12,000-19,999 and household size>=2, urban or 20,000-29,999 and household size>=4, urban or 12,000-19,999 and household size>=3, rural or 20,000-29,999 and household size>=7, rural;
High Income	all other individuals.
Source of Income	
Wages	income from wages and salaries
Welfare	income from family benefits or "welfare"
Other	income from inheritance, stocks, bonds, interest
Education	
None	no formal education
Primary	elementary school
Secondary	high school
Trade	community college, CGEP, nursing, trade school
University	university

The statistics package SAS was used to calculate the proportion of individuals: (1) consuming at least the minimum daily recommended number of servings of each of the four food groups (from *Canada's Food Guide* [5]), (2) meeting the recommendations for intake of dietary fibre and (3) whose fat intake was at or below the suggested level (as outlined in the *Nutrition Recommendations for*

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Table II
Nutrition Recommendations

Nutrition Recommendations
1. Follow the recommendations of Canada's Food Guide for daily intake of the four food groups
i) milk and milk products - adults, 2 servings - adolescents/pregnant, 3 servings
ii) fruits and vegetables - 4 servings
iii) meats and meat alternates - 2 servings
iv) breads and cereals - 3 servings
2. Consume at least 30g of dietary fibre per day
3. Limit fat intake to no more than 30% of total calories

Canadians [6]). These recommendations are shown in Table II. These values were compared across the three indicators for SES, gender/ parity groups and four age groups using the chi-squared test.

RESULTS

A total of 61,216 individuals participated in the OHS. Response rates were 88% and 76% for the interview and self-completed portions of the survey respectively.

The sample size of the pregnant group was small (n=486). As a result, analysis of the dietary habits of the pregnant group was only performed in relation to income.

Not all residents of Ontario met dietary recommendations. Only about 2/3 of the population consumed the recommended number of servings of any one of the four food groups. Less than 30% of any age/sex group consumed the minimum level of 30g of dietary fibre per day and with the exception of women aged 65+, the proportion of individuals consuming 30% or fewer of their total calories from fat never exceeded 20%.

FRUITS AND VEGETABLES

The proportion of individuals consuming the recommended number of servings of fruits and vegetables was consistently higher in the high income group, with the exception of pregnant women. Not including those in the elderly group, individuals receiving family benefits met the recommendation less often than those whose income was derived from wages or some other source. Meeting the fruit and vegetable recommendation was positively associated with level of education, except among adolescents (Figure I).

MEATS AND MEAT ALTERNATES

Individuals in the low income group and those receiving welfare benefits were more likely to meet the recommendation for this food group. The reverse was true for adolescent males and the elderly. Level of education varied inversely with the likelihood of eating the daily recommended number of servings of meats among men 20-39 and 40-64 and women 12-19 and 20-39 (Figure II).

BREADS AND CEREALS

Apart from the elderly, those in the low income group were more likely to meet the recommendation for breads and cereals. Those on welfare consumed the recommended number of servings more often than the wages and salaries group, except for adolescent boys and women 20-39. There was no obvious effect of education on bread and cereal intake.

MILK AND MILK PRODUCTS

High income was associated with a greater proportion of individuals consuming the recommended number of servings of dairy products within the youngest and oldest age groups for each sex (12-19 and 65+) (Figure III). The reverse was found for young adults and adults, with the low income group meeting the milk recommendation most often. With the exception of men 65+ and women 20-39, milk consumption was lower among those receiving family benefits. Increasing level of education was associated with a greater proportion of individuals meeting the recommendation for milk, except among adolescent girls, who demonstrated an inverse relationship with level of education (Figure IV).

DIETARY FIBRE

The low income group consistently consumed the suggested amount of dietary fibre more often than those with a higher income, except among elderly men. Welfare recipients were also more likely to meet the recommendation than those with wages and salaries with the exception of men over the age of 40. Education was not a good predictor of fibre intake.

FAT

The low income groups and welfare recipients (except adolescent males) uniformly consumed fat at or below the recommended level more frequently. The same was true among those in the lowest and highest education groups apart from men 40-64, whose fat intake better met the recommendations with increasing

education, and women 65+, who had a generally consistent fat intake across all levels of education.

DISCUSSION

Defining Socioeconomic Status

The conceptualization and measurement of socioeconomic status is a controversial area in epidemiology. For simplicity, three indicators of SES were used in this study: an income measure adjusted for family size and area of residence; a source of income variable that contrasts those receiving income assistance, those receiving employment income, and those with mainly investment income; and level of formal education. There is some overlap among these variables. For example, individuals reporting a low household income will more likely receive welfare benefits. Likewise, a higher education might be associated with better employment opportunities and hence a higher income. Our choice of indicators was an attempt to measure comprehensively, within the constraints imposed by secondary data analysis.

How did dietary intake vary with SES in the OHS sample?

Numerous nutrition surveys have been performed over the past two decades and the results suggest that there is no clear relationship between SES and nutrition. Some studies have found that members of the lower socioeconomic classes consume a diet higher in fat and sugar, lacking in variety from the four basic food groups and low in essential nutrients and vitamins. Others have found no effect of SES on nutrition, suggesting that dietary patterns are constant across socioeconomic strata. Looking at the three indicators of SES used in this study (income, source of income and education), it becomes evident that there are deviations from the recommendations for each of the four food groups and fibre and fat, but the pattern is not consistent.

Beginning with fruits and vegetables, we see that fewer individuals consumed the recommended number of servings if they were in a low socioeconomic group (low income and/or low education and/or receiving welfare benefits). Reports of a similar relationship between fruit and vegetable consumption and SES have appeared repeatedly in the literature [7,8,9,10]. Lower SES groups are at risk of vitamin deficiencies, especially vitamins C, A, B12 and iron.

Using data from the early 1970's, Kreidler et al [11] observed that the purchase of dairy foods was higher in households with a college-educated head and/or high income. Other reports of the effects of SES factors on nutrition agree that milk consumption increases with rising socioeconomic class [7,8,9]. In contrast, we found an inverse relationship between dairy product consumption and rising SES in the *Ontario Health Survey* but it was not consistent, varying with age and sex. Income effects were

seen only among adolescents and the elderly while education was a strong factor for everyone except adolescents. The decreasing likelihood that adolescent females will consume the recommended number of servings of milk and milk products as education increases clearly demonstrates the age/sex specificity of SES effects.

Past nutrition studies have demonstrated a relatively constant consumption of the breads and cereals and meat and meat alternates groups across levels of SES [7,8]. However, the OHS data show a direct relationship between low SES and the likelihood of meeting the recommendation for each of these food groups (although the effects of education on bread consumption were not as evident).

Apart from adolescent males and the elderly, those in the higher SES groups were all less inclined to eat the suggested number of servings of meat and meat alternates (eggs, legumes, nuts and seeds). Nutrition experts have advised the public to decrease its consumption of saturated fats and cholesterol. Foods in the meat and meat alternates group (especially red meats and eggs) have been targeted as the primary sources of these substances in the diet. It is possible that the higher SES groups are responding to this advice by reducing their consumption of these foods.

From this information, one might expect the higher SES groups to consume a diet significantly lower in fat. However, the OHS data show that the lower SES groups were more likely to consume fewer than 30% of their total calories from fat. In reviewing recent literature, data concerning fat intake with SES are conflicting. Read *et al.* [12] demonstrated that both education and income were positively related to the intake of dietary fat while Jeffery *et al.* [13] reported a higher fat diet in the low SES groups. Hulshof and his co-workers [10] showed no difference in total fat consumption between different classes. Obviously, more work is needed to determine the true effects of SES on fat intake.

The suggested daily intake of dietary fibre has been set at 30g. However, fewer than 30% of the population met this recommendation. High fibre was positively associated with a low SES, which may be related to the slightly higher intake of breads and cereals seen within this group.

CONCLUSIONS

No one food group can provide all the essential nutrients needed for health and growth, so it is recommended that Canadians select from all four food groups daily. The results of the OHS indicate that not all

residents of Ontario are eating a diet that follows these recommendations. For each of the four basic food groups, no more than 2/3 of the population consumed the recommended daily number of servings, placing at least 1/3 of the population at risk for nutritional deficiencies. This is especially true for the lower SES groups who were found to consume fewer fruits and vegetables and less milk.

Residents of Ontario are consuming too much fat, especially those in the higher SES groups. A high fat diet has been found to be associated with coronary heart disease, hypertension, stroke and non-insulin dependant diabetes mellitus. Effective programs to reduce dietary fat are essential if we want to reduce the morbidity and mortality from these diseases which continue to be among the leading causes of death in Canada (despite substantial declines from earlier periods).

The recommendations for fibre intake were set to help reduce the risk of colon cancer. Ontario residents, especially those in the higher SES groups, are not eating the recommended amounts of fibre.

Socioeconomic factors were found to play a role in the proportion of individuals meeting the nutrition recommendations. The lower SES groups were least likely to meet the nutrition recommendations set for dairy products and fruits and vegetables while they met the recommendations more frequently for meat and meat alternates and breads and cereals. Those at lower SES levels were also found to consume fibre and fat at levels closest to the recommendations.

The effects of SES on nutrition in the Ontario population are complex, often limited to specific age/sex groups and showing no consistency between different food groups or nutrients. Specific attention should be paid to the adverse effects of SES on nutrition, especially the lower consumption of milk, fruits and vegetables by the lower SES groups and the high fat diet of the higher SES groups. These problems should be targeted in nutrition programs.

Although there was considerable overlap between the different SES variables, education was perhaps the strongest correlate of the adequacy of dietary intake. Since education is presumably related to nutrition knowledge, improving public nutrition education might reduce the disparity seen between the socioeconomic classes and result in a more even distribution of healthy eating.

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REFERENCES

1. *Health and Welfare Canada. 1973. Nutrition - a National Priority. Ottawa: Minister of Supply and Services.*
2. *Health and Welfare Canada. 1981. The Health of Canadians -Report of the Canada Health Survey. Ottawa: Minister of Supply and Services.*
3. *Health and Welfare Canada. 1989. The Active Health Report on Fitness, Nutrition and Safety. Ottawa: Minister of Supply and Services.*
4. *Ministry of Health. 1990. The 1990 Ontario Health Survey Documentation Report.*
5. *Health and Welfare Canada. 1982. Canada's Food Guide Handbook (Revised). Ottawa: Minister of Supply and Services.*
6. *Health and Welfare Canada. 1989. Nutrition Recommendations for Canadians. Ottawa: Minister of Supply and Services.*
7. *Cronin FJ, Krebs-Smith SM, Wyse BW and Light L. Characterizing food usage by demographic variables. J Am Diet Assoc 81:661-673. 1982.*
8. *Myres AW and Kroetsch D. The influence of family income on food consumption patterns and nutrient intake in Canada. Can J Public Health 69:208-221. 1978.*
9. *Slesinger D, McDivitt M and O'Donnell F. Food patterns in an urban population: age and sociodemographic correlates. J Gerontol 35:432-441. 1980.*
10. *Hulshof K, Lowik M, Kok F, Wedel M, Brants H, Hermus R and ten Hoor F. Diet and other life-style factors in high and low socio-economic groups (Dutch Nutrition Surveillance System). Eur J Clin Nutr 45:441-450. 1991.*
11. *Kriedler PL, Boehm WT, Lentner MN and Driskell JA. Dairy product purchases by southern households. J Am Diet Assoc 77:41-45. 1980.*
12. *Read M, Fisher K, Bendel R, Bhalla V, Bock A, Harrill I, Mitchell M, Schutz H, Sheehan E and Standal B. Dietary fat intake: demographics. J Am Diet Assoc 89:830-831. 1989.*
13. *Jeffery R, French S, Forster J and Spry V. Socioeconomic status differences in health behaviours related to obesity: the Healthy Workers Project. Int J Obesity 15:689-696. 1991.* Ω

FEATURE SECTION

This issue features neurology and neurological surgery (neurosurgery). Reviews of the neurosurgical management of head trauma and low back pain, two very common areas of neurosurgical practice, are presented together with thought-provoking reviews of cochlear implants and the potential of artificial neural networks.

Management of Head Trauma

by Ross Mantle, Meds'95

The following is a brief discription of the epidemiology, diagnosis and management of head trauma from a neurosurgical perspective.

EPIDEMIOLOGY

MORBIDITY AND MORTALITY OF HEAD INJURY

Trauma is the leading cause of years of life lost in the developed world. Of these deaths, mortality attributable to head trauma accounts for approximately 50%. In the United States, the incidence of head injury with loss of consciousness is 0.2-0.3% per capita per year, while the head injury mortality rate is 25 per 100 000. The major causes of head injury in that country are: 1) motor vehicle accidents, 2) gunshot wounds to the head, 3) falls (including suicidal) and non-firearm assaults. Males in their second decade are most likely to be affected. Males sustain head injuries at twice the rate of females, and fatal head injuries at four times the rate of females. Two thirds of head injury deaths occur before the patient reaches hospital.[1]

Of particular interest to the neurosurgeon, approximately half of head injured patients who are in coma on arrival to hospital have an intracranial hematoma visible on CT scan. [2]

The severity of head trauma may be quantified using the Glasgow Coma Scale (GCS) (Table 1), which has been extensively used in research and closely correlated with prognosis (Fig. 1). The severity of head injury can be broadly classified based on initial GCS as mild (GCS 13-15), moderate (GCS 9-12) or severe (GCS <8).

Fig. 1 relates GCS to mortality based on a study of 1248 patients where the observed GCS was due to head injury alone, i.e. without a metabolic or drug-induced cause for coma.[3] Mortality due to head injury is generally higher in older age groups, a fact which is not taken into account in Fig. 1.

Table 1. Glasgow Coma Scale (GCS)

points	best eye opening response	best verbal response	best motor response
1	none	none	none
2	to pain	incomprehensible	decerebrate posturing*
3	to speech	inappropriate	decorticate posturing**
4	spontaneous	confused	withdraws to pain
5	—	orientedx3***	localizes pain
6	—	—	obeys commands

The GCS score is the sum of the points obtained in each of the three categories, maximum score: 15, minimum score: 3. eg. a patient who opens the eyes to speech, verbalizes inappropriately, and withdraws to pain would have a GCS of 10.

**Decerebrate posturing: extension of the limbs and neck with internal rotation and flexion of the hands and feet. This is due to injury below the cortical level affecting the vestibulospinal tract and pontine reticular formation of the midbrain.*

***Decorticate posturing (primate "antigravity" response): flexion in all joints of the upper limbs with extension of the lower limbs and internal rotation and plantarflexion of the feet. This is due to injury of the corticospinal pathways above the midbrain.*

****Orientedx3 implies that the patient correctly identifies self, year/month, and location.*

Those who survive head injury are subject to post-traumatic seizure disorders, hemiparesis, ataxias, post-traumatic headaches, and prolonged or permanent neurobehavioural sequelae including slowed information processing with interrelated attentional disturbance and memory impairment. Left pre-frontal lesions are associated with disorganized language and impoverished narrative, while right pre-frontal damage results in socially inappropriate and tangential communication. Childish behaviour, diminished insight, poor impulse control, motivational problems, irritability, anxiety and psychomotor retardation are also common in addition to specific deficits, such as anosmia, related to brain injury at functional foci.[4] Rimel and colleagues have shown that three months after injury, one third of mild head injury survivors, two-thirds of moderate, and almost all severely head injured patients have not returned to work.[5]

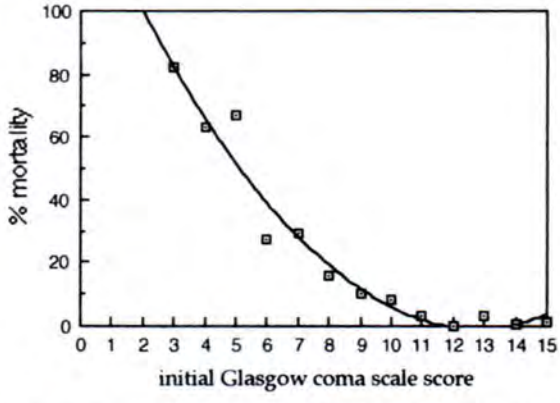

Fig. 1 Correlation of Glasgow Coma Scale with Mortality

Created by author based on data from a series of 1248 patients (Jane JA, Rimel RW. Prognosis in head injury. Clin Neurosurg 29:346, 1982.). The solid line represents a parabolic regression (r=.98) when % mortality = 142 - 22(GCS) + 0.9(GCS)^2.

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Table 2.
Diagnostic implications of several ocular abnormalities.

abnormality	implication
lateral gaze preference	destructive lesion of ipsilateral frontal aversive eye field (Brodman's area 8) or contralateral pons, or seizure in contralateral frontal aversive eye field.
one eye immobile with fixed and dilated pupil and ptosis	ipsilateral IIIrd nerve palsy
vertical diplopia with downward gaze, improved by head tilt to the contralateral side	ipsilateral IVth nerve palsy
one eye unable to rotate laterally	ipsilateral VIth nerve palsy
inability to close one eyelid with ipsilateral facial muscle paresis	ipsilateral VIIth nerve palsy
isolated inability to rotate eye(s) medially	internuclear ophthalmoplegia: disruption of medial longitudial fasciculus, may be bilateral
downward gaze preference (rare)	acute compression of tectal plate as with posterior thalamic hemorrhage or sudden hydrocephalus
progressive inability to gaze upward	compression of the tectal plate, seen in bilateral subdural hematoma or hydrocephalus
skew deviation: one globe rotated upward	unknown brainstem lesion most likely on the side of the hypotonic eye
afferent pupillary defect (pupil reactive to light only when shone into the other eye)	ipsilateral IInd nerve palsy, or retinal or preretinal damage.
hippus: rhythmic bilateral pupil dilation and contraction which may correspond to respiratory pattern	disinhibition of dampening of autonomic tone fluctuations due to acute cerebral insults, associated with altered consciousness
bilateral pinpoint (small) pupils	hyperparasympathetic tone due to opiates, metabolic encephalopathy, or destructive bilateral lesion of pons
unilateral miotic (small) pupil with ptosis and loss of sweating on ipsilateral face	Horner's syndrome: interruption of ipsilateral sympathetic signals anywhere along path from hypothalamus to thoracic cord to carotid sheath
corectopic pupil: size and shape (round to oval) of pupil varies, migration of pupil about the iris	asynchronous contraction of iris muscle fibers due to midbrain disorder
bilaterally fixed and dilated pupils	reversible inadequate cerebral perfusion, or late stage uncal herniation.
transient cortical blindness	focal concussion of occipital lobes

NB. Palsies of the cranial nerves serving the eye are either immediate or may be delayed several days after the trauma due to dural swelling over a fracture of the clivus.

DIAGNOSIS

Head Trauma History and Physical Exam

Evaluation of head trauma is secondary to initial consideration of airway, breathing and circulation. Four to five percent of patients rendered unconscious due to head trauma have associated spine fractures, most commonly at the C5-6 level. Therefore, the spine must be included in the evaluation. Approximately 60% of severely head injured patients have sustained injury to one or more non-neural organ systems. The possibility that loss of consciousness due to a mechanism other than trauma may have preceded or resulted in the trauma such as ruptured aneurysm or seizure should be considered.

The neurologic assessment of head injured patients must be carried out sufficiently aggressively to ascertain the true best level of function. A vigorous effort to determine arousability and ability to follow commands must be made. Patients who are intubated or unable to speak for other reasons may be able to answer questions non-verbally. Periorbital bruising or ecchymoses with swelling may prevent eye opening, though manual retraction of the lids may reveal good visual tracking and the ability to move the eyes in response to commands. Poorly responsive patients are typically stimulated by rubbing over the sternum with the knuckle, pinching of the trapezius muscle, or pressing with the thumb against the inferior margin of the supraorbital ridge. Withdrawal to pain is ascertained by applying pressure to the proximal nail bed with a hard object, or rubbing the plantar surface of the foot with the handle of a reflex hammer. The degree of withdrawal is often graded out of 5.

ESSENTIAL HISTORY

1. Mechanism, nature, time of injury. Presence of restraints, helmet.
2. Occurrence, duration of unconsciousness.
3. Degree and duration of amnesia, confusion.
4. Presence of neck pain, back pain, radicular symptoms, numbness.
5. Visual symptoms.
6. Presence of headache, photophobia, nuchal rigidity, vomitting.

PHYSICAL EXAM

1. Mental status: evaluation of orientation, Glasgow Coma score.
2. Inspection. Scalp ecchymosis, abrasions, lacerations. Signs of basal skull fracture: racoon's eyes (periorbital ecchymosis), Battle's sign (perimastoid ecchymosis), hemotympanum, laceration of external auditory canal. Signs of breach of dural integrity: CSF otorrhea or rhinorrhea. Obvious signs of spine trauma.
3. Palpation of the face for LeFort or orbital rim fractures.
4. Auscultation for bruits over the carotids, which may be due to traumatic dissection, and over the globe of the eye, which may indicate traumatic rupture of the carotid into the cavernous sinus.
5. Examination of the cranial nerves including funduscopy for retinal hemorrhage or retinal detachment. This includes assessment of facial movement and tone, conjugate gaze, gaze preference (right, left, upward, downward), pupil shape, size and reactivity, glabellar, corneal and gag reflexes. Table 2. describes the diagnostic implications of several abnormal eye findings.[6]

6. Motor exam: deltoids, biceps, hand intrinsics, ilipsoas, quadriceps, hamstrings, plantarflexion and dorsiflexion tested and graded as 5 normal strength, 4 decreased strength, 3 antigravity, 2 not antigravity, 1 flicker, 0 no movement. Check withdrawal to pain if uncooperative, note spontaneous movement and rectal tone with anal wink and bulbocavernosus reflex for assessment of spinal function.
7. Sensory exam in cooperative patient: pinprick on trunk and limbs to detect sensory levels, and position sense in toes for posterior column function.
8. Other reflexes: deep tendon, plantars, presence of clonus.

CHILD ABUSE

Retinal hemorrhage seen on fundoscopy is considered virtually pathognomonic of child abuse, and occurs in Shaken Baby syndrome. Retinal hemorrhages may less commonly appear as a result of benign subdural effusion sometimes seen over the frontal lobes in term infants, or acute high altitude cerebral edema. Also, multiple skull fractures, bilateral skull fractures, and fractures that cross suture lines are more likely due to child abuse than accidental trauma.

INDICATIONS FOR OBSERVATION AND DISCHARGE ONLY

1. No change in level of consciousness or amnesia.
2. No progressive headache.
3. No intoxication.
4. No post-traumatic seizure.
5. No vomiting.
6. No signs of basilar skull fracture, depressed skull fracture or penetrating skull injury.
7. No focal neurological findings, sensory level or weakness.
8. No tenderness on palpation of the dorsal spines of the vertebrae.

If all of the above criteria are satisfied, the likelihood of intracranial injury is less than 1 in 1176, even if headache, dizziness, scalp injuries, or linear skull fracture are present.[7] In this case a short period of observation only followed by discharge into the care of a responsible person who can carry out precautions against the possibility of later subdural hemorrhage is recommended. Further investigations are not recommended. Subdural precautions consist of checks every two hours over a twelve hour period for orientation and arousability. Lack of tenderness of the dorsal vertebral spines indicates probable absence of traumatic injury likely to compromise the spinal cord.

INDICATIONS FOR LABORATORY INVESTIGATIONS

Research demonstrates in animals that mild hypoxemic and concussional injuries from which complete recovery within 24 hours is expected can result in severe brain damage and death when both insults occur within a short time of one another. Unfortunately, as

many as half of head injured patients present to hospital with one or more of: arterial hypoxemia (pO_2<60 mmHg), arterial hypotension (systolic <90 mmHg), anemia (Hct. <30%), and arterial hypercarbia (pCO_2 >45 mmHg).[8]

Continuous oxygen saturation and arterial blood gas sampling are used to enable aggressive correction of hypoxia and hypercarbia. Blood type and cross, and close attention to blood pressure and hemoglobin/hematocrit are required for use of transfusion in patients with significant blood loss or anemia. Screening for the presence of alcohol, drugs and for blood glucose level, and BUN/creatinine is usually indicated to rule out metabolic causes of cerebral toxicity or encephalopathy. CBC, electrolytes, BUN/creatinine, serum osmolality and clinical exam indicate level of hydration. Serum PT/PTT/INR and platelet levels are obtained prior to surgery to prevent excessive bleeding due to surgery in the presence of coagulopathy.

VITAL SIGN DISTURBANCES IN HEAD INJURY.

Blood pressure is a critical consideration in severe head injury because cerebrovascular autoregulation is usually impaired, and intracranial pressure (ICP) is usually increased. Since cerebral perfusion pressure is equal to the systemic mean arterial pressure minus ICP, cerebral blood flow is compromised when ICP is high. To maintain appropriate cerebral blood flow, systemic pressures must be maintained within the normal range when ICP is normal, and at slightly higher levels if ICP is high or when cerebral vasospasm is present. Cerebral vasospasm typically occurs approximately seven days after subarachnoid hemorrhage related to ruptured aneurysm, but may occur as a result of subarachnoid hemorrhage due to trauma (trauma being the most common cause of subarachnoid hemorrhage). Definitive diagnosis and monitoring of cerebral vasospasm is accomplished using trans-cranial doppler.

Low blood pressure is almost never caused by central nervous system injury except in terminal stages with dysfunction of the medulla. Hypotension is more likely due to blood loss. Cushing's response is a result of near fatal medullary compression. It immediately precedes death, and is characterized by rising blood pressure with decreasing heart rate. Cushing's triad of 1) hypertension, 2) bradycardia, and 3) respiratory irregularity, is a classic finding in cases of raised ICP, but actually occurs in only about 33% of cases.[7]

Several abnormal respiratory patterns indicate damage at various anatomical levels. Bihemispheric or diencephalic dysfunction cause the crescendo - descrescendo ventilatory pattern known as Cheyenne-Stokes respirations. Lower midbrain or upper pontine lesions can cause sustained hyperventilation, while low pontine lesions result in apneusis, a respiratory pattern in which the inspiratory phase is prolonged. Sighing, yawning and hiccoughs can be signs of disinhibition of brainstem reflexes indicating impending central cephalic herniation due to high ICP. Irregular or ataxic respirations indicate medullary dysfunction.

Injury to the upper brainstem may result in centrally mediated fever.

INDICATIONS FOR CT HEAD

1. head injury with decreased level of consciousness at any time or amnesia
2. focal neurological signs or deterioration in neurologic exam
3. intoxication
4. possible depressed skull fracture or penetrating injury
5. inadequate history or pre-ictal event such as seizure
6. absence of acute herniation or uncontrolled abdominal or other bleeding requiring immediate operative procedure

CT scans of the head are repeated if there is deterioration in neurological status or increase in ICP to 20 mmHg or more, and at 3-5 and 10-14 days after a severe head injury.

FEATURES OF DIAGNOSTIC IMPORTANCE ON HEAD CT

The most common CT finding in head trauma is intracranial blood, which appears hyperdense with respect to brain parenchyma when fresh, becoming isodense at about 21 days and hypodense thereafter as the collected blood undergoes changes related to coagulation, cell decomposition, osmotic migration of fluids, and metabolism by surrounding tissues. Because of the large number of factors affecting this process, dating of intracranial blood by its appearance on CT is extremely unreliable. In addition, fresh blood may appear less dense on CT in individuals with significant anemia or where blood is mixed with CSF. A subdural collection of fluid of similar density to CSF may indicate a traumatic hygroma. This is a collection of CSF caused by rupture of the arachnoid, releasing CSF into the subdural space.

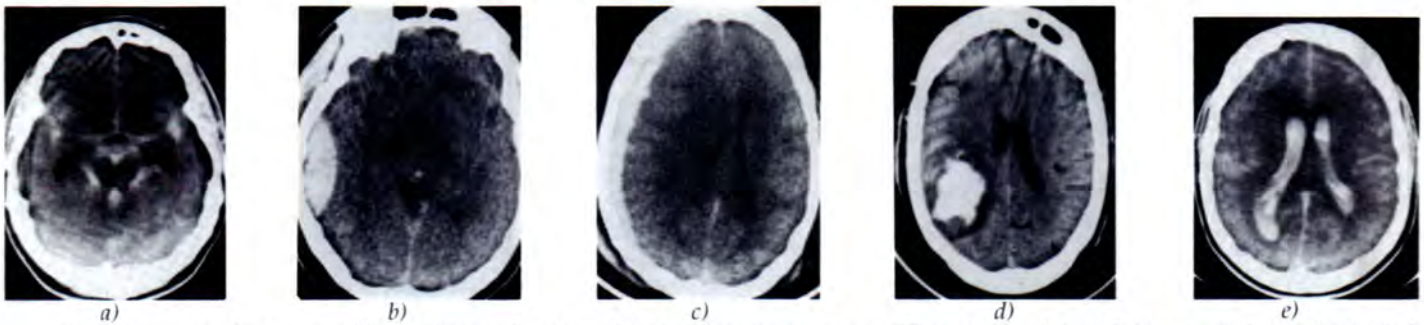
Determination of the location of hemorrhage on CT allows diagnosis of: subarachnoid hemorrhage (Fig. 2a), epidural hematoma (Fig. 2b), subdural hematoma (Fig. 2c), intraparenchymal hemorrhage (hemorrhagic contusion) (Fig. 2d), intraventricular hemorrhage (Fig. 2e).

Enlargement of the ventricles indicates hydrocephalus, obliteration of the basal cisterns and ventricular compression indicate cerebral swelling, and the presence of air intracranially (pneumocephalus) may reveal an open skull fracture. Cerebral anoxia causes loss of the gray-white matter interface on CT and may be accompanied by signs of swelling. Skull fractures, including depressed and linear fractures of the calvarium as well as basal fractures and facial fractures may also be seen.

INDICATIONS FOR CEREBRAL ANGIOGRAPHY

1. Suspicion of sub-arachnoid hemorrhage, i.e. pre-ictal event
2. Suspicion of traumatic cavernous carotid fistula (pulsating exophthalmos on exam)
3. Suspicion of carotid dissection

The indications for cerebral angiography are limited when CT is available, but the technique can provide valuable information about vascular injury, or the presence of space-occupying lesions which do not appear on CT but do cause displacement of major vessels.



a) b) c) d) e)

Fig. 2 Diagnosis of intracranial hemorrhage or hematoma based on location as seen on CT scan of head. 2. a) Subarachnoid hemorrhage. High signal attenuation due to blood collection evident in basal cisterns encircling the brain stem and other CSF spaces including the Sylvian fissure, interhemispheric fissures and sulci. 2b) Epidural hematoma. Blood collection outside the dura evident over right parietal region with typical biconvex appearance as dura is peeled from its adhesion to the inner table of the skull. 2c) Subdural hematoma. Extensive extra-axial (peripheral) blood over right hemisphere with flat or concave inner margin. 1-2 cm midline shift away from the lesion is seen. 2 d) Intraparenchyma hemorrhage (hemorrhagic contusion). Obvious well demarcated high density lesion in right parietal region with surrounding low-density edema. 2.e) Intraventricular hemorrhage. High density material within the ventricles indicates the presence of blood.

INDICATIONS FOR SKULL X-RAYS

Skull X-rays are rarely useful and tend to be overused. Indications should be limited to evidence of depressed or non-linear skull fracture. May be useful in some cases to help in the precise localization of an intracranial foreign body.

INDICATIONS FOR MRI HEAD

Generally not useful in acute trauma because of relatively poor sensitivity to intracranial hemorrhage, long acquisition time and the incompatibility of the strong magnetic field with metal in ventilators and IV pumps. Used when the patient's neurologic findings on CT are not sufficient to account for the neurologic status.

INDICATIONS FOR C-SPINE X-RAYS

1. Patient has suffered significant trauma of any kind.
2. Trauma patient with loss of consciousness.
3. Minor trauma with neck or back pain, neurological signs in the extremities, abdominal breathing, or priapism.

STEPS TO CLEAR THE C-SPINE.

1. Portable lateral C-spine X-rays while in collar showing intact vertebrae C1 to T1 with absence of significant pre-vertebral edema and absence of tenderness on palpation of vertebral spines - may remove collar to complete step 2. (Neck pain may prevent step 2.)
2. AP, lateral and odontoid views. May proceed to step 3 if there is no subluxation greater than 3.5 mm and the patient is neurologically intact.
3. Flexion and extension views showing absence of any increased subluxation.

Note: oblique views are rarely helpful, and expose the patient to unnecessary risks due to neck movement. Patients who were thrown from a motor vehicle, or experienced a fall >1.8 m (6 ft.) or who have back pain or isolated neurologic findings in the lower extremities should have thoracic and lumbar X-rays.

INDICATIONS FOR ICP MONITORING

1. head injury with insufficient neurological exam (GCS <7) to monitor neurological deterioration.

ICP is monitored most commonly with the connection of an intraventricular catheter to a pressure monitor, using a subarachnoid bolt, or intraparenchymal monitor. Some centres do not use the technique because of a lack of evidence for benefit in terms of outcome.

CLINICAL EVIDENCE OF RAISED ICP

1. non-localized, unremitting headache. Worse in the morning, made worse by Valsalva maneuver, bending, coughing.
2. nausea and vomitting, worse in morning
3. papilloedema. Often only appears after several days, may not be present if raised ICP is acute.
4. progressively decreasing level of consciousnesss

CLINICAL EVIDENCE OF UNCAL HERNIATION

1. progressively dilating (mydriasis), sluggish to non-reactive pupil with progressive ptosis. This results from loss of parasympathetic signals due to compression of oculomotor nerve by the uncal gyrus with preservation of sympathetic signals originating in the hypothalamus and passing through the thoracic spinal cord.
3. rapidly decreasing level of consciousness progressing to decorticate, then decerebrate posturing on painful stimulation.
4. contralateral hemiparesis. Ipsilateral hemiparesis may rarely occur (Kernohan's notch phenomenon) due to compression of the contralateral uncus against the tentorium.
5. loss of oculocephalic (doll's eyes) and oculovestibular (caloric stimulation) reflexes
6. Abnormalities in vital signs including abnormal respiratory patterns, increasing blood pressure and decreasing heart rate.

MANAGEMENT

After stabilization and appropriate diagnostic procedures as discussed above, the need for immediate vs. delayed surgery is determined. The patient is then managed medically to provide the optimum conditions for healing and recovery, often in the intensive care setting. Commencement of IV or enteral alimentation as soon as is feasible may prevent depression of the immune system and is associated with better outcome. A neurosurgery consultation from the emergency department is recommended for any patient with a GCS <14, a history of loss of consciousness, altered mentation, post-traumatic seizures, or neurologic deficit including pupillary signs.[9]

INDICATIONS FOR INTUBATION

1. Level of consciousness incompatible with protection of airway
2. GCS<7 (require hyperventilation to manage high ICP)
3. Severe facial trauma with possibility of loss of airway patency
4. Need for pharmacologic paralysis for management, such as surgery.

Nasotracheal intubation is to be avoided in patients with possible skull base fractures as in such cases it is possible to pass the tube intracranially through the cribiform plate. The patient's ability to verbalize should be noted prior to intubation as this important diagnostic feature is then lost. Early intubation is crucial in severe head injury and should be accomplished in the field or before transfer to a neurosurgical facility.

MANAGEMENT OF RAISED ICP

Possibly 90% of severe head injuries are accompanied by ICP >20mmHg (normal adult ICP <10-15 mmHg, lower in children). ICP >25-30 mmHg can be fatal because of compromise of cerebral blood flow and uncal herniation, the most common brain herniation syndrome. Uncal herniation is fatal because of rupture and detachment of arterial perforators supplying the brainstem. This detachment occurs as the brainstem moves downward through the foramen magnum while the basilar artery remains fixed to the skull base.

Current understanding of intracranial pressure rests on the Munroe-Kellie doctrine, which states that the sum of the volume occupying components (blood, brain, CSF) within the cranium is a constant. Any increase in volume in any component must, therefore, be offset by a decrease in that of the other components, or an exponential rise in ICP will develop. High ICP occurs as result of: 1) brain swelling (edema), 2) dilation of blood vessels in response to elevated pCO_2, traumatic loss of vasomotor control or systemic hypertension, 3) presence of intracranial hemorrhage or hematoma, 4) hydrocephalus, 5) obstruction of venous outflow due to venous sinus thrombosis or high intrathoracic pressure due to seizures or agitation. A delayed rise in ICP may occur with delayed hematoma formation, cerebral vasospasm causing hypoxic brain swelling, and hyponatremia - most commonly due to inaproppriate anti-diuretic hormone secretion common in brain injury.

MEASURES TO CONTROL HIGH ICP:

1. IV mannitol 1-2 g/kg bolus of 20% solution q6h, may alternate with IV Lasix 20 mg q6h in between mannitol doses.
2. Hyperventilation to pCO_2 of 25 mmHg
3. Sedation, seizure and pain control
4. Maintain high serum osmolality (320 Osm) using Lasix diuresis, normal saline IV and fluid restriction
5. Control fever (fever stimulates increased cerebral blood flow and a 5%/C° increase in metabolic rate)
6. Control hypertension
7. Control hyperglycemia
8. Ventricular drainage of CSF via ventriculostomy catheter
9. Surgical evacuation of space-occupying lesion
10. Elevation of head of bed to 30% (produces minimum ICP with maximum blood flow)[12]

Elevation of the head of the bed has not been shown to improve outcome in some studies.[10] Induction of barbiturate coma using pentobarbital 100 mg slow IV also lowers ICP, but is used generally as a last resort.

Mannitol is a relatively inert sugar which is thought to decrease the volume of the brain parenchyma by drawing fluid from the interstitium osmotically. Indications for immediate mannitol include evidence of raised ICP or herniation as described above, a focal neurologic deficit such as hemiparesis, which could be caused by a mass effect, sudden deterioration in neurologic status, or CT evidence of a lesion likely to result in raised ICP or mass effect. The maximum decrease in cerebral volume is achieved after 20 minutes. Mannitol causes an initial increase in intravascular volume followed by diuresis, therefore patients with congestive heart failure may require prior treatment with Lasix. Mannitol is contraindicated in the face of hypotension because of its diuretic effect.

Hyperglycemia is generally present to some degree in head injury, and is associated with persistently high levels of lactate in the CSF. Control of hyperglycemia is recommended since it is associated with exacerbation of cerebral edema.

Induction of coma using pentobarbital clearly lowers ICP, and has a number of theoretical benefits including decreased metabolic demand and free radical scavenging, but has not been proven to improve outcome. Similarly, steroids reduce vasogenic cerebral edema, such as around brain tumours, but are less effective in cytotoxic cerebral edema, which prevails in trauma. Their regular use has fallen out of favour because of side effects.

Antacids or H2-antagonists should be used because of the significant risk of formation of Cushing's stress ulcers due to the hypergastrinemia associated with raised ICP.

Prolonged hyperventilation may have deleterious effects including changes in cerebral blood flow and acidosis. It is therefore often used intermittently for acute elevation of ICP.

MANAGEMENT OF SKULL FRACTURES

Depressed skull fractures are elevated if they are depressed beyond the thickness of the surrounding skull or are associated with dural laceration and CSF leak. Contaminated fractures may necessitate removal of the involved bone to prevent infection. Damage to underlying dural sinuses may require saphenous vein grafting.

Most basal skull fractures do not require treatment. Associated traumatic aneurysms caused by damage to the arterial wall, carotid-cavernous fistulae, and persistent CSF fistulae may require treatment. Use of prophylactic antibiotics to prevent meningitis in the presence of dural breach with CSF leakage is controversial and not widely practised except in the case of fractures through the nasal sinuses, which are considered contaminated.

Temporal bone fractures are also usually not treated except where the fracture has caused a facial nerve palsy. Steroids are used to attempt to decrease swelling-related nerve dysfunction. Failing this, surgical decompression may be attempted.

Facial fractures disrupting both the anterior and posterior walls of the frontal sinus are treated surgically to prevent intracranial infection. The posterior wall of the sinus is removed and the dura repaired with a periosteal or fascia lata graft. Pneumocephalus may occur as a result of a fracture through an air sinus or over the convexity of the skull. The patient may become symptomatic if the dural defect is such that air is allowed in but not out, creating tension pneumocephalus. Decompression of the gas produces dramatic improvement.

MANAGEMENT OF INTRACRANIAL HEMORRHAGE/HEMATOMA AND HYGROMA

As a rule of thumb, surgical removal of a collection of intracranial blood will be beneficial in cases where CT indicates at least a 5 mm midline shift due to the lesion. Surgery is also recommended in cases of bilateral lesions more than 5 mm from the inner table of the skull, as these are likely to produce significant elevations in ICP.[2] Time is of the essence for most lesions, with significantly improved outcomes related to rapid evacuation. In a study of 82 patients with acute subdural hematoma, for example, those evacuated within four hours of injury had a 30% mortality, compared to a mortality of 90% if surgery was delayed.[11]

EXPLORATORY BURR HOLES

In the face of the ominous triad of 1) altered mental status, 2) unilateral fixed and dilated pupil, and 3) contralateral hemiparesis in the emergency room (see other signs of herniation, above), the patient's chances of satisfactory recovery are at best 20% with immediate surgical treatment. Since the responsible lesion is most likely an extra-axial hematoma on the side ipsilateral to the affected pupil, immediate placement of exploratory burr holes without CT evidence is defensible, though rarely attempted unless an extreme triage situation exists in the emergency department. Placement of the first burr hole in the temporal bone just above the zygoma maximizes the chances of access to a hematoma. The dura is opened if it has a bluish discoloration suggestive of subdural hematoma.

OPERATIVE TREATMENT OF INTRACRANIAL HEMATOMA OR HYGROMA

Breach of the blood brain barrier in head trauma exposes the systemic circulation to brain thromboplastins, which may lead to activation of coagulation cascades and depletion of coagulation factors (disseminated intravascular coagulation or DIC). The presence of DIC, as determined by serum PT/PTT/INR, D-dimer concentration, and fibrinogen and fibrinogen degradation products, precludes surgery unless adequate coagulation can be restored through the use of replacement products. In an emergent situation, the PT/PTT/INR should at minimum be obtained prior to surgery.

Hemorrhagic contusions, epidural hematomas, and acute subdurals are evacuated via craniotomy, while chronic subdurals are drained through burr holes, unless persistent recurrence necessitates craniotomy to remove subdural membranes. Traumatic subdural hygromas are drained through burr holes as with chronic subdural hematomas, although hygromas tend to recur and may require craniotomy to attempt to locate the source of the arachnoid breach or subdural-peritoneal shunt in cases refractory to other management.[7]

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REFERENCES

1 *Frankowski RJ, Annegers JF, Whitman S. Epidemiological and Descriptive Studies Part I: The descriptive epidemiology of head trauma in the United States. in Becker DP, and Povlishock JT, eds. Central Nervous System Trauma Status Report - 1985. NIH 1985:34.*

2 *Narayan RK. Emergency room management of the head-injured patient. In Becker DP, Gudeman SK (eds) Textbook of Head Injury. Philadelphia:Saunders 1989:23-61.*

3 *Jane JA, Rimel RW. Prognosis in head injury. Clin Neurosurg 29:346, 1982.*

4 *Levin HS, and Eisenberg HM. Neurobehavioural Outcome. in Eisenberg HM and Aldrich EF eds. Management of Head Injury. Philadelphia:Saunders 1991:457-472.*

5 *Rimel RW, Giordani B, Barth JT, Boll TJ, and Jane JA. Disability caused by minor head injury. Neurosurgery 9(3):221-228, 1981.*

6 *Selhorst JB. Neurological examination of head-injured patients. In Becker DP, Gudeman SK (eds) Textbook of Head Injury. Philadelphia:Saunders 1989:2-4.*

7 *Greenberg MS. Handbook of Neurosurgery. 3rd ed. Lakeland:Greenberg Graphics 1994:523-48.*

8 *Becker DP. Common Themes in Head Injury. In Becker DP, Gudeman SK (eds) Textbook of Head Injury. Philadelphia:Saunders 1989:2-4.*

9 *Trauma service department, Henry Ford Hospital Trauma Center. Trauma Treatment Protocols, Detroit, MI, 1st ed. 1994, p. 10.*

10 *Rosner MJ, Coley IB. Cerebral perfusion pressure, intracranial pressure, and head elevation. J Neurosurg 1986; 65:636-41.*

11 *Seelig JM, Becker DP, Miller JD, et al. Traumatic acute subdural hematoma: Major mortality reduction in comatose patients treated wihtin four hours. N Engl J Med 1981; 304:1511-8.*

12 *Quentin JD, Del Maestro RF, Sibbald WJ. Cerebral and cardiovascular responses to changes in head elevation in patients with intracranial hypertension. J Neurosurg 1983; 59:938-944.*

Ω

ARTIFICIAL NEURAL NETWORKS AND CLINICAL MEDICINE: A GLIMPSE INTO THE FUTURE.

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ABSTRACT

Recently, there has been an increased interest in Artificial Neural Networks and their use in clinical decision making. The focus of this article is to evaluate their use in clinical trials. The basic function of these systems and the ethical issues surrounding ANNs are also discussed.

INTRODUCTION

Modern computers represent an attempt to better understand and duplicate the complex processes that result in "intelligence." Until very recently computers had largely failed to emulate human intelligence because they failed at creativity, complex pattern recognition and the ability to learn from past experience. An Artificial Neural Network (ANN) is a computer program modeled after the biologic nervous system. The basic processing unit of an ANN, analogous to a biologic neuron, receives numeric input variables, transforms this information, and produces an output. The output then "synapses" with one or more similar processors and produces a final numeric output as the result (*Figure 1*). Unlike traditional computer programs, ANNs appear to be very effective at recognizing complex patterns in data based on past experience.[1] ANNs are "trained" by presenting sets of input patterns called the "training set" together with the corresponding outputs that the trainer wishes the network to learn. The behaviour of the neural network is determined by the weights of the interconnections between neurons that result from the training process.[1] ANN learning can then be evaluated by inputting similar but previously unseen example data patterns called the "validation set." Medical decision making depends upon complex subjective and objective data patterns. It is in complex pattern recognition that ANN technology is most likely to have a significant impact in clinical medicine.

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EVIDENCE

Artificial neural network technology has been used successfully in a number of studies to predict outcomes and to compare the accuracies of computer predictions with expert physician predictions. For example, Baxt has used an ANN to diagnose myocardial infarction in patients presenting to an emergency department with anterior chest pain.[2] Initially, he trained his network using retrospective data from a large number of patients who had presented to the ER with anterior chest pain and who had subsequently been shown to have had or not had an acute MI. The trained ANN was then prospectively tested on 331 consecutive patients presenting with anterior chest pain. Input variables, including patient history, physical examination findings and results of laboratory investigations were provided by the attending physician. The physician was also asked to predict whether or not the patient had had a myocardial infarction. ANN predictions had a sensitivity of 97% and specificity of 96% compared with a sensitivity of 78% and a specificity of 85% for the physicians' predictions. The author concluded that ANN may someday be a valuable diagnostic aid for ER physicians in evaluating chest pain and is certainly worthy of further investigation.

More recently the use of ANNs has been extended to the intensive care unit setting. Danter et al. used an ANN to compare computer outcome predictions with predictions made by ICU consultants regarding patient survival or mortality as well as length of stay after admission to a mixed medical-surgical intensive care unit.[3] Once the ANN was trained with retrospective data from 167 patients, learning was evaluated on prospective data from 110 patients admitted to the ICU. As in the Baxt study, the intensivists were asked to predict outcome based on the same information that was presented to the ANN. The accuracy of the ANN predictions of survival or death was 99.1% compared with 94.1% for the physician predictions ($p=0.06$). ANN predictions of length of stay after ICU admission were 90.9% accurate while physician predictions were 82.7% accurate (NS). However, ANN predictions were far more accurate than those of physicians in predicting which patients would stay 8 or more days in the ICU (82.4% vs 41.2%, $p=0.02$). The ability to predict early those patients who were at high risk of dying could lead to improved outcomes in some patients. Accurate length of stay predictions could also result in more appropriate allocation of limited ICU resources.[3] In this study, ANN predictions appeared to be at least as accurate as those of expert physicians and further study with promising new technology was recommended.

In another study in the Intensive Care Unit setting, Chang et al. used daily APACHE II scores to compare physician and nurse "outcome unknown or will die" evaluations with computer predictions in 227 ICU patients.[4] The predictions made by physicians and nurses had a false-positive rate of between 7.7% and 16.7% that the patient would die. Significant numbers of deaths correctly predicted by both doctors and nurses were missed by the computer model and there were deaths predicted by the computer that were missed by the doctors and nurses. Predictions made during this study were not acted upon and while the group felt that their results warranted further evaluation in their own ICU, the computer model was not ready for general use.

In addition to the applications of the ANN outlined above, the complex pattern recognition capability of this technology have been demonstrated in other areas. ANNs have been used in analyzing electrocardiograms,[5] electromyograms,[6] and to assist in image processing in computed tomography and ultrasonography.[7] In addition, studies have demonstrated the usefulness of neural networks in diagnosing pulmonary embolism,[8] evaluating hepatic masses,[9] and diagnosing psychiatric disturbances.[10] ANNs have also been able to accurately predict such outcomes as (i) death or survival, (ii) length of stay, and (iii) the possibility of early discharge in patients admitted to hospital with pneumonia.[11]

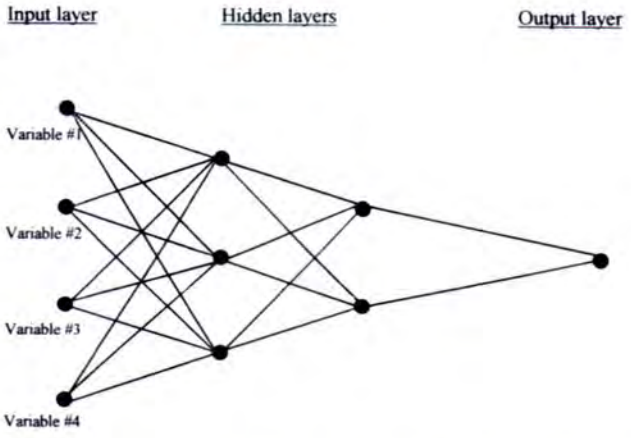

Figure 1- Example of a simple Artificial Neural Network with four input variables, two hidden layers and one output.

PROS AND CONS

The major impact of ANN technology is likely to be improved predictive accuracy resulting from better evaluation of patient data patterns that may be too complex for even expert physicians. The Baxt study indicates that improved predictive sensitivity could result in more accurate diagnosis of acute myocardial infarction.[2] Improved specificity, on the other hand, could result in significant cost saving by reducing unnecessary interventions and admissions to the hospital in patients without myocardial infarction. Baxt provided evidence that a 3% improvement in diagnostic specificity in patients presenting with anterior chest pain would lead to an annual saving of $85 million dollars in the U.S.[12] In today's cost conscious medical environment, improved predictive accuracy would almost certainly lead to more appropriate utilization of scarce resources.

ANN predictive accuracy can improve over time if the ANN is periodically retrained with more appropriate and up to date patient data. Another advantage of computer predictions is that they should be more consistent since they are unaffected by emotions, variability in physician expertise or fatigue although there is still potential for physician bias at the time of data input.[1] Furthermore, ANN technology offers the traditional benefits of computer systems which can evaluate vast amounts of data with impressive speed. With advances in hardware miniaturization and voice recognition software, future trained and validated ANN systems could be embedded in inexpensive hand held, voice activated computers. ANNs are currently available for virtually any personal computer.

Unfortunately ANN technology is young, expensive and computer intensive. In addition to the costs of data collection and preparation for training an ANN, commercially available systems range in price from around $1,000 to more than $30,000 in U.S. funds.[3] The iterative process of ANN training , evaluation, modification, and retraining may take weeks to complete, even for simple systems of a few hundred "neurons" with less than 20 input variables and only 2 or 3 output categories. The major stumbling block to wide spread acceptance of this new technology remains the "Black Box" nature of ANN predictions.[13] Present generation ANNs are not able to explain how they arrived at their predictions. These steps are known as "hidden layers" (*Figure 1*). The recent development of more advanced ANNs capable of reporting interneuron connection weights represents the first major step towards at least a limited capacity for explanation.

ETHICAL ISSUES

As with most new technologies, critics have identified a number of important ethical issues surrounding the use of ANNs. Although ANNs have produced some exciting results, many physicians and patients remain concerned about the increasing dehumanization of modern medicine. They argue that this technology lacks the human factors essential to the "art" of clinical medicine. Understandably, many physicians will not place their trust in a process which remains impenetrable and inexplicable. To counter this argument, Guerriere and Detsky point out that the history of science and medicine is filled with landmark empirical observations without a complete understanding of the process. They use the example of the discovery of penicillin to emphasize this point.[1] Accountability is another important issue. Who will make the final clinical decision? Especially when there is difference of opinion between computer and physician.

Most proponents of ANNs have emphasized that the physician must continue to make the patient care decisions and that this technology provides a tool to help physicians to make better informed decisions. Strict guidelines for the use of ANNs will have to be developed in order to confront issues like equality and appropriateness.[3] The introduction of ANNs will also force the medical community to ensure that abuses of the new technology do not occur.

CONCLUSION

Research involving ANNs in clinical medicine has produced some promising results and raised several ethical questions. However, we are still years away from the widespread introduction and acceptance of ANNs into the day to day practice of medicine. Initially, at least, ANNs are more likely to be used as complex pattern recognition tools to enhance the prediction and decision making capabilities of physicians. It is crucial to recognize ANNs as an emerging technology which requires rigorous evaluation with regard to its validity, reproducibility, safety, and utility. Continued development of ANN technology will be an important element of clinical research that propels modern medicine into the 21st century and beyond.

REFERENCES:

1. *Guerriere R.J., Detsky A.S. Neural Networks: What are they? Ann. of Int. Med. 115:906-907 (1991)*
2. *Baxt W.G. Use of an Artificial Neural Network for the Diagnosis of Myocardial Infarction. Ann. of Int. Med. 115:843-848 (1991)*
3. *Danter W.R., Wood T., Morrison N.J., Whately G., Ngan J. A pilot study of Artificial Neural Network predication of outcome for patients admitted to a mixed medical-surgical intensive care unit submitted to Critical Care Medicine Sept. 1994*
4. *Chang R. et al. Accuracy of decisions to withdraw therapy in critically ill patients: Clinical judgment versus a computer model Crit. Care Med. 17:1091-1097 (1989)*
5. *Edenbrandt L., Devine B. Classification of electrocardiographic ST-T segments: Human expert vs Artificial Neural Network Eur.Heart J. 14:464-468 (1993)*
6. *Spitzer A. Signal decomposition and diagnostic classification of the electromyogram using a novel neural network technique. Proceedings of the 14th Annual Symposium on Computer Application in Medical Care 552-556 (1990)*
7. *Boone J., Sigillito V., Shaber G. Neural Networks in radiology: an introduction and evaluation in a signal detection task Med. Phys. 17:234-241 (1990)*
8. *Tourassi C.,Floyd C. Acute pulmonary embolism: Artificial Neural Network approach for diagnosis Radiology 189:555-558 (1993)*
9. *Maclin P., Dempsey J. Using an artificial neural network to diagnose hepatic masses J. Med. Syst. 16:215-225 (1992)*
10. *Modal I., Stoler M. Clinical decisions for psychiatric inpatients and their evaluation by a trained neural network. Meth. Inf. Med. 32:396-399 (1993)*
11. *Wood T., Danter W.R. A neural network expert system for predicting death or survival for patients admitted to hospital with pneumonia submitted to J.A.M.A Sept. 1994*
12. *Baxt W.G. Use of an Artificial Neural Network for data analysis in clinical decision making: the diagnosis of acute coronary occlusion Neural Comp. 2:480-489 (1991)*
13. *Hart A., Wyatt J. Evaluating black boxes as medical decision aids: issues arising from a study of neural networks Med. Inf. Lond. 15:229-236 (1990)*

Ω

BREAKING THE SOUND BARRIER WITH COCHLEAR IMPLANTS

by Priya Chopra, Meds '95

The ability of the cochlear implant to dramatically change the life of a profoundly hearing-impaired person is unsurpassed by any other sensory aid. Use of the multichannel cochlear implant has been described as the first instance in which a lost sense can be restored to a degree of useful function with a prosthetic device. This remarkable technology is designed to replace a non-functioning inner ear with a direct electrical stimulus to the auditory nerve, thus bypassing the hair cells. It must be emphasized, however, that the machine is only as effective as the accompanying auditory and speech production training. This is especially true in children.

To qualify for the procedure, the candidate must have bilateral profound or total sensorineural hearing loss, must obtain no useful benefit from hearing aids and have no medical contraindications to implantation such as ossification or congenital absence of the cochlea as determined by CT scan. Children must have suitable intelligence to allow for conditioning and implant programming, and both adults and children (and their families) must be highly motivated and hold realistic expectations of the surgery. The implants are generally offered to pre- and post-lingually deaf children (i.e. onset of deafness before and after learning to speak respectively) but only to post-lingually deaf adults. Children often suffer sensorineural deafness secondary to a bout with bacterial meningitis causing breakdown of the hair cells. Other causes of deafness potentially treatable with the implants include congenital hearing disorders, ototoxic drug use, head injury with perilymph fistula formation, labyrinthitis, trauma or tumour.

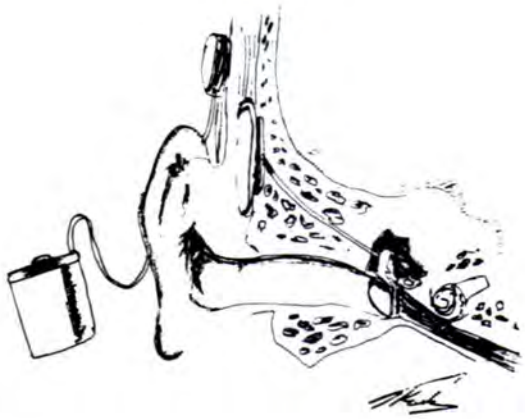

Figure 1. Coronal cross-section through the mastoid bone showing external and internal components of cochlear implant device.

The implant consists of internal and external components. (Fig. 1) The internal components are surgically placed and consist of a receiver/stimulator and a 22-channel electrode array. The receiver/stimulator is positioned under the skin in a shallow recess made in the mastoid bone and connected to the electrode array. The array is passed through a facial recess approach to the middle ear, through the round window and advanced 2-3 cm into the cochlea. (Fig. 2) The external components are worn by the individual and incorporate a speech processor similar in appearance to a calculator, a headset comprised of a directional microphone worn like a behind-the-ear hearing aid, a transmitting coil held magnetically over the receiver/stimulator, and two wires connecting the processor, microphone and transmitter.

The microphone receives and transduces ambient sound and sends the corresponding signals to the speech processor. These electrical waveforms are then amplified, filtered and digitized by the processor. The information is then sent via the transmitter across the skin to the implanted receiver/stimulator as a FM radio signal. The receiver/stimulator then delivers appropriate amounts of electrical energy to the correct electrode among the 22 electrode array, stimulating auditory nerve fibers in the cochlea which, in turn, transmit auditory information to the brain. These events take place on a millisecond time scale, allowing sounds to be perceived by the user without noticeable delay.

The speech processor is the mastermind behind this incredible machine. It contains a microchip implementing a unique coding strategy called SPEAK, named after the "spectral peak" phenomenon. This is based on the observation that unwanted complex sound waves resulting in noise are homogeneous when considered over time, while meaningful sounds such as speech show intermittent amplitude peaks. The processor divides sounds into twenty programmable pitch bands which are each assigned to one of the 22 electrodes arranged tonotopically on the cochlea. Higher frequency sounds, which would in the normal ear cause resonance vibration of the basilar membrane in an area where it is appropriately narrow (near the base of the cochlea), are converted to electrical signals which stimulate an electrode located over that approximate area. Pulsed stimulation of the electrodes varies inversely with the complexity of the sounds picked up such that there is a low stimulation rate for complex sounds such as background noise and an increased stimulation rate for simpler sounds such as speech. The SPEAK strategy therefore attempts to maximize the sounds of speech while minimizing the effects of noise.

The evaluation of a candidate pre- and post-

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operatively involves a large team of professionals. The audiologist provides pre-and post-implant assessments and counselling, speech processor programming and consultation with educators. The surgeon ensures that the candidate meets the medical criteria for implantation and performs the surgery, of which the most critical aspect is how far into the cochlea the electrode can be advanced. The auditory-verbal therapist both monitors and develops programs to aid the child's listening and language skills. The child-life specialist supports the family and child during their hospital stay. The psychologist helps in counselling the patient and family regarding expectations of surgery and may do pre-and post-op psychological evaluations. The social worker assesses the social supports in place and addresses financial concerns. The education program for children is of critical importance as they require hours of daily auditory-oral stimuli to "learn how to hear". Post-lingually deaf adults generally do not require formal rehabilitation to successfully use the implant. Both children and adults begin "hearing" with the activation of the speech processor 6 weeks post-operatively. This phase involves the initial programming of the processor over two days based on the user's perception of a series of test pitches. Re-programming is scheduled for 2, 6, 12, 24, and 52 weeks post activation and then once a year for five years. The follow-up re-programming is particularly important in children as they are better able to tell the audiologist what they hear as they grow older.

Evaluation of the effectiveness of the implants subjectively through a questionnaire showed the most significant benefits to be in areas of one-to-one communication, feelings of self-confidence, sense of safety, reduced stress level and ability to socialize. A study from the Central Institute for the Deaf compared children with implants versus conventional hearing or tactile aids. Preliminary results show that the implanted children were better able to perceive vowels and consonants after one to three years, and their ability to lip-read was improved 25% as compared to 16% two years after implantation. With respect to speech production at two years, differences in overall intelligibility were not significant nor was vocabulary acquisition. However, the implanted children were producing more varied speech sounds and longer more complex sentences. Although it is impossible to predict how successful a child will be following implantation it is believed that the earlier a child is implanted the greater the potential for progress.

Limitations are an inherent part of all man-made electronic systems attempt-ing to substitute for a complex human sense. Factors affecting the degree of benefit achieved from an implant include the degree of auditory memory (in the post-lingually deaf), number and location of healthy nerve fibres remaining in the cochlea, motivation and commitment to developing listening skills, and the educational programs in which the implantee is enrolled. There are always standard surgical risks and also those specific to inner ear surgery to be considered. These include injury or stimulation of the facial nerve, taste disturbance, dizziness, increased tinnitus, neck pain, and perilymph fluid leak causing meningitis.

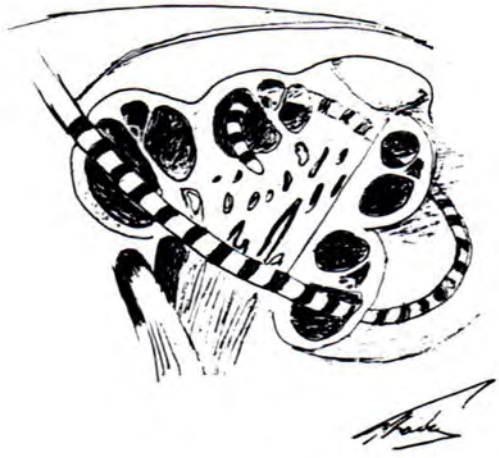


Figure 2: Cut-away view of cochlea with electrode array.

According to the manufacturer of the device, the cost of the machine and pre- and post-implant care ranges around $25 000 US (or $15 600 per quality-adjusted-life-year) which puts it slightly below the cost-effectiveness of triple-vessel coronary artery bypass grafting. Implantation is covered under OHIP and some insurance plans in the United States. The system also allows for future flexibility; should a better implant become available, the electrode array can be removed and replaced or a new implant can be placed in the other ear.

Nevertheless, some individuals and groups do not view the implants positively. Across Canada in mid-May of 1994 a group of deaf protesters held demonstrations against the use of cochlear implants and the availability of government funding for this program. Preferring to see themselves as different rather than handicapped, demonstrators opposed to implant funding argued that the implants deprive hearing-impaired children of the richness of deaf culture and substitute it with a crude form of hearing.

REFERENCES

Hall C, Treasure S. Deaf Deserve Choices. Toronto Star 04/05/94

Charbonneau L. Early ear implant showing outstanding results. The Medical Post 14/05/94

Radcliffe D. Window to the world of hearing: cochlear implants in children. The Hearing Journal 1994; 47(6):13

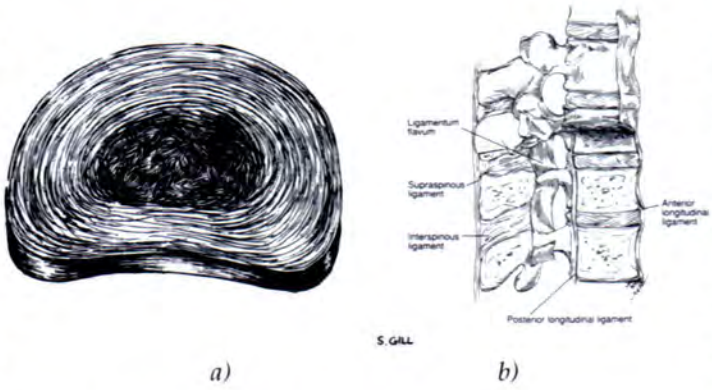
Cochlear Corporation promotional material: "Issues and answers", 1994.

Ω

LUMBAR DISC HERNIATION AND LOW BACK PAIN A NEUROSURGICAL PERSPECTIVE

by Andrew E. Jones, Meds '97

Low back pain is one of the most prevalent problems facing physicians in the industrialized world today. It will affect approximately 80% of North Americans at some point in their lives,[1] and is the leading cause of employee absenteeism.[2] Fortunately, only 7% of sufferers will continue to have symptoms 6 months later. This small fraction, however, will consume up to 90% of the money spent on the treatment and compensation of back pain,[1] which has been estimated at upwards of 24 billion dollars annually in the United States alone.[3] These chronic patients are also subject to a relapse rate of 75%, and will repeatedly return for treatment. It is therefore important for physicians to have a good understanding of the causes of low back pain and any treatment regimens which may be available to them.



a) *b)*

Figure 1. The anatomy of the intervertebral disc. a) Isolated, b) in situ.

ETIOLOGY AND DIFFERENTIAL DIAGNOSIS

Low back pain is merely a symptom whose cause may lie in any one of many neighbouring or distant structures. Although almost 95% of low back pain is mechanical, the physician must first rule out other more serious organic causes of low back pain. Several researchers have recently been studying structures which may be responsible for low back pain.[4] One such study involved stimulating structures in low back pain sufferers during surgery to determine which structure was most responsible for the generation of the patient's pain. The study identified the annulus fibrosus and posterior longitudinal ligament as the two most common structures responsible for pain. Surprisingly, it was also discovered that muscular tissue was rarely the cause. Other researchers have intensely studied the role of the myofascial pain syndrome as it relates to low back pain,[5] while still others have stressed the role of the lumbar disc, proposing that most low back pain originates therein.[3] Several risk factors have also been identified which may predispose an individual to low back pain (Table 1).

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Although the purpose of this article is to discuss low back pain in the context of lumbar disc herniation, a brief discussion of the differential diagnosis of low back pain is required. The diagnosis and subsequent treatment of low back pain rely, as with any condition, on a well taken history and a thorough physical examination. Table 2 contains a list of the possible causes of low back pain. Most conditions listed present with symptoms other than low back pain and hence are usually easily distinguished from discogenic pain; however, this may not always be the case. Special maneuvers may be required, such as palpation of the relevant trigger point in cases of myofascial pain,[5] or special investigations such as a bone scan, to reveal possible neoplasms.

THE LUMBAR SPINE : ANATOMICAL CONSIDERATIONS

The lumbar intervertebral disc is a connective tissue element that serves to absorb the stress and shock to which the spine is subjected in addition to relieving the shear and compressive forces sustained during spinal motion.[6] It is composed of three distinct elements: the nucleus pulposus, the annulus fibrosus, and the cartilaginous endplates (Fig. 1). The nucleus pulposus is a mucopolysaccharide containing gel located in the centre of the disc. It is surrounded by the alternating tissue fibres of the annulus fibrosus. Attachments of the fibres are made centrally to the cartilaginous endplates and laterally to the vertebral body. The endplates themselves are fixed to the superior and inferior aspects of their respective vertebrae via calcified cartilagenous attachments. The fibres also make attachments to the anterior and posterior longitudinal spinal ligaments. It is significant to note that the posterior ligament is markedly narrower than the corresponding anterior ligament. As a result, the disc is not attached to a ligament at its posterolateral aspect, the most frequent site of rupture.

Table 1
Risk factors associated with low back pain.*

Increasing age up to age 55
White race
Smoking
Prolonged driving of a motor vehicle
Hard physical labour
Psychologic stress
Job dissatisfaction
History of low back pain
Existing osteoporosis

**adapted from Loesser & Volinn in Fager, C.A. Lumbar discectomy. In: C.B. Wilson, (ed). Neurosurgical Procedures : personal approaches to classic operations. Williams & Wilkins, Baltimore, MD (1992). and Margo in Vanderburgh, D.F. and Kelly, W.M. Radiographic assessment of discogenic disease of the spine. Neurosurgery Clinics of North America, 4:13-33 (1992).*

Since the spinal cord normally terminates at approximately the L1/L2 level, the more caudal nerve roots must travel further down the canal to reach their point of exit under the pedicle of the corresponding vertebral body. It is therefore possible for a single disc protrusion to encroach upon more than one root as illustrated in Figure 2. A large central protrusion and/or sequestration may lead to a cauda equina syndrome whereby all passing roots are affected and the patient presents with back and bilateral leg pain in addition to bowel, bladder, and sexual dysfunction.

LUMBAR DISC HERNIATION

It is important to realize that the intervertebral disc undergoes a normal degenerative process over its lifetime. The posterior annulus fragments and its attachment to the posterior ligament weakens. The water content of the nucleus pulposus slowly decreases, which results in narrowing of the disc space. In addition, the general elasticity of the disc decreases as elastic fibres are replaced by more fibrous ones. These factors can lead to bulging of the disc into the spinal canal.[6] This process, known as degenerative disc disease, occurs in all healthy people beginning as early as age 30 and can be accelerated by certain activities such as repetitive bending and heavy lifting. Disc degeneration, however, does not equal disc herniation, a concept which must be made clear to patients.

True disc herniation is a result of weakening of the annular fibres and subsequent bulging of the nuclear material into and/or through the annulus. The extent of nuclear material released classifies the prolapse into: i) a simple protrusion, ii) an extrusion, or iii) complete sequestration (see figure 3). The magnitude of the herniation does not always correlate with the clinical findings or the patient's symptoms, as even the smallest protrusion may cause excruciating pain, often for unknown reasons.[7]

The pain produced due to a herniated disc generally has two separate components: i) the spinal and ii) the radicular. Spinal pain may simply be a result of a torn annulus, which itself is richly innervated by a combination of branches of the sinuvertebral nerves, ventral rami and the grey rami communicantes.[8] The pain may also be due to stretching and/or rupture of the posterior ligament, dural inflammation or "protective" muscle spasms.[6,7] In contrast, radicular pain is a result of the compression of the root itself. Ectopic stimulation of the nerve via the disc or a sequestrated fragment results in symptoms of pain, paresthesias and numbness.[9]

The nature of the herniation will govern which roots, if any, are involved. Typically, the disc herniates just lateral to the posterior ligament, i.e., "posterolateral" herniation, compressing the root which is leaving the dural sac (and will exit under the pedicle of the immediately inferior vertebral body). A large, central protrusion may compress the entire cauda equina whereas the so-called "far lateral" disc protrusion may

Table 2.
Causes of low back pain.*

Mechanical	Segmental instability, trauma, congenital deformity, spinal stenosis, leg length inequality, iliac crest syndrome, facet syndrome, herniated lumbar disc
Peripheral nerve	Piriformis syndrome, proximal neuralgia, femoral/obturator/sciatic neuropathy
Neoplasms	Osteoid osteoma, multiple myeloma, skeletal metastases, benign cysts of the spinal canal/retroperitoneum, intradural tumors (especially of the cauda equina)
Rheumatologic disorders	Polymyalgia rheumatica, fibrositis, ankylosing spondylitis, Reiter's syndrome, psoriatic/enteropathic/osteoarthritis
Metabolic disorders	Paget's disease, gout, hemoglobinopathies, osteogenesis imperfecta, hyperparathyroidism, diabetic amyotrophy, bursitis, osteoporosis, adhesive arachidonitis, diffuse idiopathic skeletal hyperostosis
Inflammatory conditions	Vertebral osteomyelitis, discitis, epidural abcess
Referred pain	Gastrointestinal, vascular, renal, gynecological/obstetrical, hip/knee/sacroiliac joint problems, coccygodynia
Miscellaneous	Idiopathic, Charcot's spine, Scheuermann's syndrome, radiation induced, spondylosis, spondylolisthesis, psychogenic

**adapted from Watson & Benson in Jennett, B., and Lindsay K.W. An Introduction to Neurosurgery. Butterworth-Heinmann, Oxford, UK (1994). and Margo in Vanderburgh, D.F. and Kelly, W.M. Radiographic assessment of discogenic disease of the spine. Neurosurgery Clinics of North America, 4:13-33 (1992).*

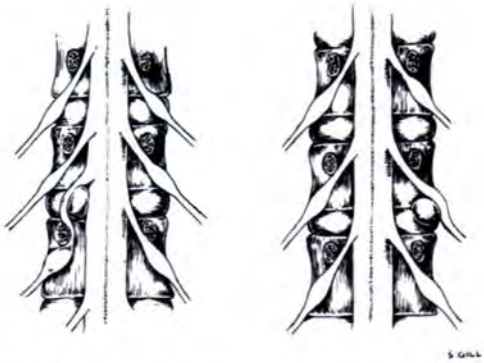


Figure 2. Herniated lumbar disc. a) Posterolateral protrusions will affect the root exiting under the pedicle of the immediately inferior vertebral body; b) Far lateral protrusions usually affect the nerve root exiting above the disc.

pinch the root which has left the dural sac at the level above as it passes beneath the pedicle and past the lateral part of the disc. Therefore an L4/L5 herniation more commonly compresses L5, but can theoretically compress the L4 root as well. Sequestration of nuclear material further complicates matters as these elements may migrate up or down the canal to produce symptoms characteristic of other discs.[7] The size and shape of the canal also can affect the result of the herniation. Herniation into a small, narrow canal will cause more significant symptomatology than one into a large, wide canal.[10]

The most commonly affected discs are the L4/L5 and L5/S1 discs, each accounting for 45% or more of lumbar disc herniations. 5-10% are primarily at the L3/L4 level.[9]

The etiology of the herniation can be somewhat of a mystery. Some patients do report a single traumatic event which precipitated the initial episode of pain, although most patients cannot recall a specific event.[10] While it is obvious that trauma may play a role, a variety of other components interact to result in disc herniation. Despite the prevalence of this condition, the pathophysiology of the herniation is poorly understood.

HISTORY

As with all other medical conditions, an accurate history is vital in obtaining a clear picture of the problem. Patients will often have a history of several years of intermittent back pain with or without associated leg pain.[3] It is important to rule out non-mechanical causes of the pain, such as rheumatologic, inflammatory, or neoplastic conditions. It is possible that the patient will have only low back pain as a symptom of their condition, but most often other signs and symptoms will point to something non-mechanical. Table 3 lists several questions which are valuable in distinguishing organic from mechanical back pain.

Questions regarding the onset, quality, quantity, and aggravating and relieving factors are all important in the diagnosis and treatment of discogenic pain. Back pain from disc related problems may often be distinguished on the basis of associated leg pain. Back pain without leg pain is almost never due to a disc.[1,7] In fact, only 10% of people who present with back pain have evidence of true sciatica,

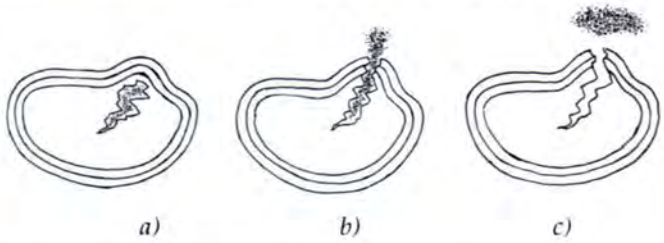


Figure 3. Lumbar disc prolapse.
a) protrusion; b) extrusion; c) sequestration.

and therefore the majority of low back pain is not due to a prolapsed disc.

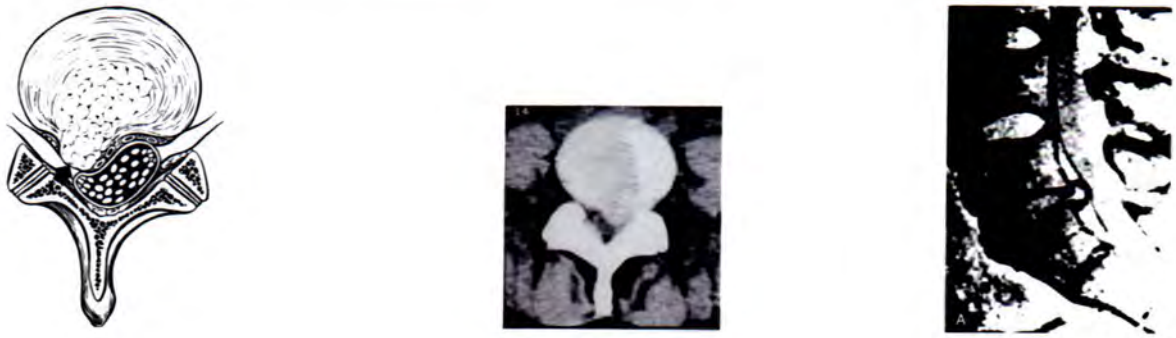
The exact nature of the leg pain is also important in separating discogenic pain from other mechanical and organic causes. Referred pain from organic conditions or myofascial pain can often radiate into the leg as far as the knee, however rarely in a clear dermatomal pattern. Mechanical joint pain can also produce similar symptoms. Fortunately, lumbar radiculopathies present with relatively well recognized signs.

Patients often report waking up with an aching or pulling sensation in their back. Similarly described leg pain usually follows soon after, following the general path of the sciatic nerve. The pain may also be associated with numbness, parathesias, and weakness radiating in dermatomal and myotomal patterns corresponding to the nerve root affected. The pain may be intermittent for several years, but usually has become constant at the time of presentation. It may be relieved by reduced physical activity and rest, but rarely does it ever disappear entirely.[11] Pain is exacerbated by excessive lifting, bending, and rotating and also by coughing, sneezing and straining (e.g., Valsalva maneuver). The latter occurs due to an increase in intraspinal pressure.[9] Patients with a large sequestrated disc often report an initial onset of back pain which subsequently subsides, only to be replaced by unrelenting leg pain radiating in a typical sciatic distribution. In either case, patients most often describe the radicular pain to be much worse than the back pain.[7] If the reverse is true, then other conditions should be considered. Patients presenting with typical pain accompanied by sphincter dysfunction should be suspected of having a large central protrusion compressing the cauda equina.[1]

All patients with disc herniations will not necessarily present in exactly this manner, however large deviations from such a history may point to other causes.

PHYSICAL EXAMINATION

A complete neurological exam of the lower extremities is necessary in order to help localize the site of herniation.[3,9] Testing should include muscle strength and tone in all major muscle groups, sensory assessment in each dermatome by pin prick, and deep tendon reflexes (knee, hamstring and ankle jerks). For example, an S1 root compression may result in any combination of decreased ankle jerk, plantar flexion weakness, pain in the distribution of the sciatic nerve and sensory loss in the region of the S1 dermatome. Dermatomes vary widely and so sensory loss has less reliable localizing value than other findings such as decreased reflexes or muscular weakness.



(a) (b) (c)

FIGURE 4. Radiographic investigations of the lumbar spine, a) Diagrammatic representation of a posterolateral herniation; b) Axial CT of a posterolateral L4/L5 disc herniation; c) Sagittal MRI of a large L5/S1 disc hemiation. (courtesy Dr. D. Pelz, University Hospital, London, ON)

Other special maneuvers may be performed in order to better ascertain the nature of the problem. Straight leg raising may reproduce pain in the affected leg as it stretches the sciatic nerve and hence the nerve roots. Some people experience hamstring discomfort before the nerve is adequately stretched. In such cases, the knee and hip may be flexed and the ankle then sharply dorsiflexed. This results in stretching of the tibial nerve and may reproduce the patient's pain. Palpation of the spine and associated musculature to identify areas of tenderness and spasm, respectively, can also aid in the diagnosis. Patients who complain of pain along the entire length of the spine should be approached with some skepticism as discogenic pain does not refer in such a wide distribution. At this time, spinal movement (flexion, extension and rotation) and spinal curvature may be assessed. Patients with disc problems may have a reversal of the normal lumbar lordosis and may present with "sciatic" scoliosis.[11]

Table 3.
Questions helpful in distinguishing organic low back pain.*

A yes to any of the following questions indicates the possibility of an organic rather than mechanical or radicular cause of low back pain:

1. Is this new onset of low back pain in an elderly patient ?
2. Is the pain worse at rest ?
3. Is the pain associated with fever and/or weight loss? (particularly in drug addicts, immunosuppressed or diabetic patients or patients with recent spinal surgery)
4. Is this severe pain causing writhing or pain associated with urinary incontinence and caudal anesthesia ?

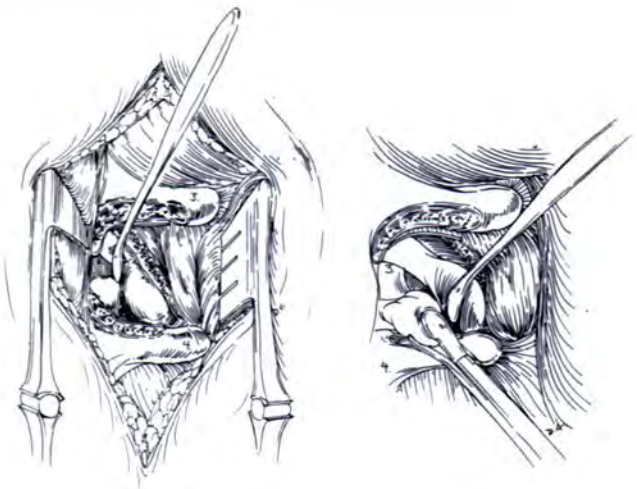
**from Margo in Vanderburgh, D.F. and Kelly, W.M. Radiographic assessment of discogenic disease of the spine. Neurosurgery Clinics of North America, 4:13-33 (1992).*

INVESTIGATIONS

The most revealing diagnostic tool is the MR scan. (Fig. 4) The clarity of soft tissue contrast it provides in addition to its ability to reveal spinal and associated abnormalities is remarkable.[12] In centres where MRI is not readily available, CT scans can be equally useful, although the clarity of the image is not as good. Some care must be taken when interpreting such scans. Recent studies have shown that 90-95% of patients aged younger than 40 with discogenic symptoms have evidence of disc bulging on CT; however, 20% of patients in the same age group who are asymptomatic also have this CT finding. These studies also have shown that the abnormality rate increases with increasing age, so much so that up to 50% of people aged 40 and older had evidence of disc bulging.[3,13] Similar studies using MRI have yet to be performed, but one would expect the same results. It is therefore worthwhile to review these scans only after the patient has been examined, and to treat the patient as opposed to the scans. Fager also points out that many radiologists who are not experienced in the area of disc prolapse and herniation are quick to label a simple degenerative process as disc herniation.[9] Despite this, the MR scan is an excellent way to confirm suspicions of disc herniation found after a proper history and physical examination.

Plain film x-rays are not very useful in diagnosing low back pain. They can only reveal narrowing of a disc space from which it is impossible to determine whether the narrowing has occurred due to simple degeneration or to disc prolapse. On the other hand, they may be useful in diagnosing other abnormalities of the spine such as spondylolisthesis or spondylosis and less commonly, inflammatory and neoplastic conditions. Plain film or CT myelography can be useful in the diagnosis as it allows one to see the outline of the spinal canal and also the exiting nerve roots. However, the benefit over a regular CT is arguable and it is certainly no better than MRI.

Additional procedures are less frequently used as they provide less information. However, if MR or CT are not available, other methods may be employed. Discography involves the injection of water soluble contrast dye into the nucleus pulposus in an attempt to reproduce the pateint's



a) b)

Figure 5. Microdiscectomy to remove a herniated lumbar disc. a) The nerve is identified and retracted. b) Disc material is removed.

habitual pain. It also allows for visualization of the disc space and the extent of the rupture. Opponents of discography point out that puncture of the disc may lead to further disc degeneration and/or premature rupture. Also, inflammation and sepsis can occur subsequent to the procedure, albeit rarely.

Another procedure which may provide further information is the use of nerve root blocks at the level of the suspected lesion. With the advent of MRI, however, such procedures are rarely used. Blood tests including ESR and CBC are of little use except in aiding in the diagnosis of some rheumatologic, neoplastic or inflammatory conditions. Bone scans may provide useful information in certain conditions.

NON-SURGICAL MANAGEMENT

As mentioned earlier, the majority of people with low back pain will have complete remission of symptoms without relapse within 4-6 weeks of the initial episode. This is less true of people with disc herniations. However, complete spontaneous recovery is still frequent so conservative management is in order. A review of the literature reveals much confusion with regard to treatment of disc herniations. Bed rest was once thought to be, and to some degree still is, the first line of treatment. It has been shown that bed rest decreases intradiscal pressure and hence may be beneficial for a limited period of time.[3] If the patient is not markedly better after two solid weeks of bed rest, one may opt for surgery provided that the problem is unequivocally discogenic.[14] Pharmaceutical management is widely debated in the literature, although no one study has shown that any one pharmacologic remedy is best. Non-steroidals, steroids and occasionally muscle relaxants may help to reduce pain for the short term. However, long term use in chronic sufferers has shown little efficacy.[1,14] Similarly, use of narcotics may be indicated for acute attacks, however long term use may lead to dependence and their use should last no longer than one week. One pharmaceutical treatment that does seem to have some beneficial effects is the use of antidepressant therapy (e.g. imipramine) for chronic pain sufferers.[3]

Physical therapy, chiropractic manipulation and exercise have been advocated by several sources as beneficial especially for the treatment of idiopathic low back pain.[15,16] In fact, the effects of these treatments may be largely due to psychological factors.[11,15] Studies have shown that there is short term benefit derived from such procedures; however, there is no conclusive evidence that these approaches affect the long term outcome of patients with a herniated disc.[1,3]

More invasive therapy such as the use of epidural steroids remains controversial. One study reported that 76% of patients who were shown to have a herniated disc showed partial or complete symptom resolution one year following epidural injection.[3] Other studies are not as positive, reporting much lower numbers and attributing much of the outcome to a placebo effect.[1]

SURGERY

The relapse rate, quoted as high as 15%, has given rise to considerable debate with regard to the efficacy of surgery for lumbar disc herniations.[3,17] Nevertheless, lumbar disc herniation is probably one of the few causes of low back pain where surgical intervention is indicated. Patients with a clear history of radiculopathy, corresponding physical findings and obvious evidence of disc herniation radiologically are excellent candidates for surgery. Referral to a surgeon should be made after a sufficient trial of bed rest with persisting symptomatology and physical signs (e.g. positive straight leg raise, muscle weakness, numbness etc.). Referral should be made even when certain aspects of the clinical or radiologic picture are missing, as long as there is enough evidence to suspect disc herniation. Urgent referral should be made in any case of suspected cauda equina compression.

The extent, position and nature of the disc herniation dictates the surgical procedure to be performed. The most common and most conservative method is termed a microdiscectomy, and can be employed for most posterolateral herniations. The patient lies prone, with the abdomen raised to avoid congestion of the extradural veins. Alternatively, the patient may be placed on the table with both knees and hips flexed to allow optimum opening of the interlaminar spaces. A posterior midline incision is made and carried down through the lumbar fascia. The paraspinal muscles are stripped from the spinous processes and laminae on the affected side are retracted. The ligamentum flavum is then cleaned of all surrounding tissue and removed. The laminotomy is then enlarged by removing the medial part of the facet joint and parts of the lateral laminae, exposing the root and disc.[18] The nerve root may then be gently retracted to fully visualize the disc (Fig 5).

If the annulus fibrosus is found intact it must be excised in conjunction with the posterior longitudinal ligament via a small incisive "window". In cases where the annulus is ruptured along with the posterior longitudinal ligament, a sequestrated fragment may be visible in the canal and should be removed. It is also necessary to keep in mind that fragments may migrate

some distance up or down the canal. Removal of all sequestrated fragments is essential. In most cases, evidence of sequestration is available pre-operatively via the MR scan. In either case, the disc space should be gently cleared as far as possible, although the amount of removal remains a controversial point.[1,7] Great care must be taken not to damage the anterior annulus during the evacuation for fear of damaging nearby structures and to prevent scarring.

More radical procedures, such as hemilaminectomy or complete laminectomy, were performed more often in past years, but are now reserved for certain situations. For example, large central protrusions often require laminectomy to relieve excess pressure on the cauda equina, but the above procedure is sufficient in most cases.[14] Fusion following discectomy is no longer indicated. Recent studies have shown that patients who do not undergo fusion recover faster and have a better prognosis than those who do.[6]

Postoperatively, patients generally require 4-5 days hospitalization. Movement may be resumed immediately post-op and the patient can often walk within 24 hours. Following this, flexion, extension and rotation should be kept to a minimum for at least the first 3 weeks. The patient should then be encouraged to perform simple flexion/extension exercises, while refraining from heavy lifting and excessive bending for 2 months. After this period, no restrictions should be placed on the patient.

Other procedures exist in order to remove herniated discs, including percutaneous discectomy or digestion of the disc material via the injection of chymopapain. These procedures have been much less successful in general and hence are not in common use.[1]

PROGNOSIS

The prognosis following surgery is good with 90-95% of patients reporting complete alleviation of leg pain. However only 60% report permanent relief of back pain.[7] The relapse rate for surgery ranges from 10-15% and may be the result of several factors. Disc prolapse at the same level recurs at a rate of approximately 5%. Recurrent symptoms may also be due to disc prolapse at another level, sequestrated fragments missed at the initial operation, or adhesions of the affected root to the disc. There is also a small proportion of patients who report to be no better as a result of the operation despite a well performed and uncomplicated discectomy.

In general, 90% of all people who experience low back pain undergo spontaneous recovery within 4-6 weeks regardless of treatment. However, the remaining 10% experience symptoms for at least 6 months, with 2% still complaining of symptoms 1 year later. Unfortunately, chronic low back pain of this type is often of unknown origin and no suitable treatment is available.[19] It is vital for the physician to maintain a positive attitude toward such patients and to use all resources available including referral to a chronic pain clinic.

ACKNOWLEDGEMENT

I would like to thank Dr. John Girvin for his valuable suggestions regarding the preparation of the manuscript.

REFERENCES

1. *Kahanovitz, N. Diagnosis and Treatment of Low Back Pain. Raven Press, New York, NY (1991).*
2. *Loesser, J.D., and Volinn, E. Epidemiology of low back pain. Neurosurgery Clinics of North America, 2:713-714 (1991)*
3. *Margo, K. Diagnosis, treatment and prognosis in patients with low back pain. American Family Physician, 49:171-179 (1994)*
4. *Kuslich, K.D., Ultrom, C.L. and Michael, C.J. The tissue origin of low back pain and sciatica : a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. Orthopedic Clinics of North America. 22:181-187 (1991).*
6. *Watts, C., and Smith, H. Disc disease. In: R.G. Grossman, (ed). Principles of Neurosurgery. Raven Press, New York, NY, pp 415-438 (1991).*
5. *Gerwin, R.D. Myofascial aspects of low back pain. Neurosurgery Clinics of North America, 2:761-779 (1991)*
7. *Jennett, B., and Lindsay K.W. An Introduction to Neurosurgery. Butterworth-Heinmann, Oxford, UK (1994).*
8. *Bogduk, N. The lumbar disc and low back pain. Neurosurgery Clinics of North America, 2:791-803 (1991).*
9. *Fager, C.A. Identification and management of radiculopathy. Neurosurgery Clinics of North America, 4:1-12 (1993).*
10. *Porter, R.W. Management of back pain (2nd Edition). Churchill-Livingston, London, UK (1993).*
11. *Fager, C.A. Lumbar discectomy. In: C.B. Wilson, (ed). Neurosurgical Procedures : personal approaches to classic operations. Williams & Wilkins, Baltimore, MD (1992).*
12. *Vanderburgh, D.F. and Kelly, W.M. Radiographic assessment of discogenic disease of the spine. Neurosurgery Clinics of North America, 4:13-33 (1992).*
13. *Nachemson, A.L. Newest knowledge of low back pain. Clinical Orthopedics and Related Research, 279:8-17 (1992).*
14. *Hardy, R.W. and Davis, C.H. Extradural spinal cord and nerve root compression from benign lesions of the lumbar area. In: J.R. Youman (ed), Youmans Neurological Surgery (3rd Edition), pp 2664-2693 (1990).*
15. *Rodriquez, A.A., Bilkey, W.J. and Agre, J.C. Therapeutic exercise in chronic neck and back pain. Archives of Physical Medical Rehabilitation, 73:870-875 (1992).*
16. *Shekelle, P.G. et al. Spinal manipulation for low back pain. Annals of Internal Medicine, 117:590-599 (1992).*
17. *Fisher, W.S. Selection of patients for surgery. Neurosurgery Clinics of North America, 4:35-43 (1992).*
18. *Hudgins, W.R. Micro-operative treatment for lumbar disc disease. In: J.R. Youman (ed), Youmans Neurological Surgery (3rd Edition), pp 2704-2714 (1990).*
19. *Gilchrist, I. Chronic low back pain. The Practitioner. 237:37-38 (1993).* Ω

STITCHES IN TIME - IV
GASEOUS GOOFINESS

W.D. Colby, MSC, MD, FRCP(C)

"I pass gas in the hospital." These were the first words that I heard an anaesthetist speak. After contemplating the double meaning for a moment, I thought that I had found the perfect avocation. Passing gas for a living! What a pleasant idea.

Anaesthesia had always been an interesting specialty. I particularly like the workmanlike approach of the anaesthetists—show up, pass the gas and leave; well-defined hours, little call, applied pharmacology and if patients get boring... ALAKAZAM! Off to "Never-Never Land". I was totally unprepared for my first day on the Anaesthesia rotation. I sat down amidst an incredible tangle of hoses, lines, electrical cords, inflating bags, accordion-like structures, and all kinds of intriguing controls, dials and buttons. The anaesthetist had already induced the patient before my arrival and the machine merrily breathed for the patient while the surgeons did their work. The anaesthetist very quickly pointed out the functions of all the pieces of equipment and also the ten syringes, all recapped, which lay in a neat row on the table.

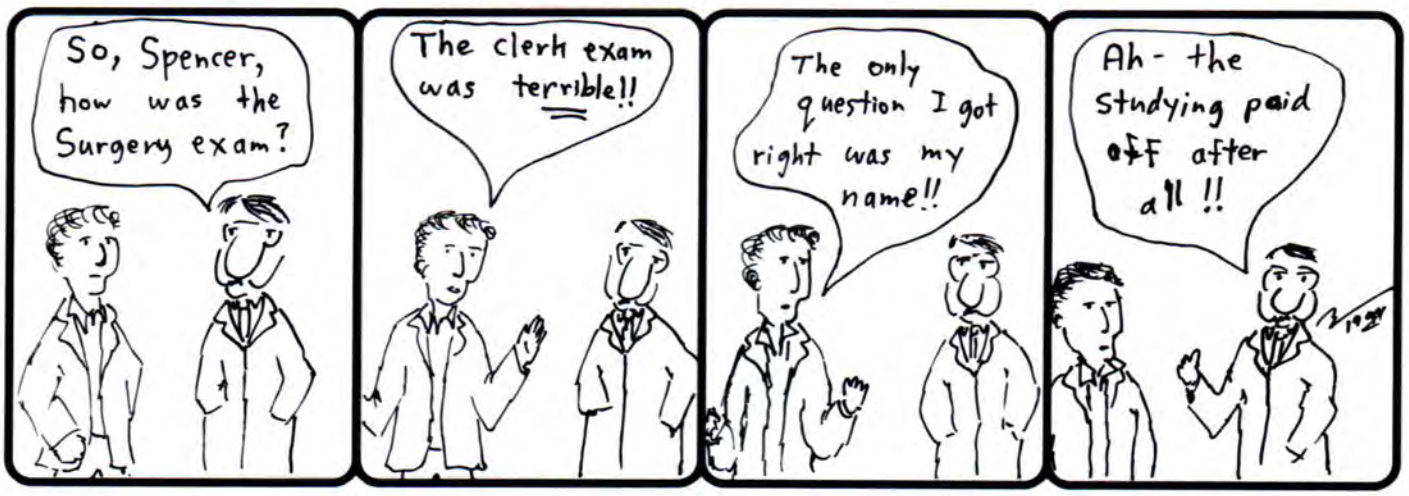
"Now then", said the anaesthetist, "If the patient gets a bit light, zap him with some more nitrous oxide or else increase the concentration of halothane or you could give him another blast of succinylcholine or even pentothal. Mind you, if the operation is over, fire the retrorockets with some atropine and Narcan. Don't forget to watch the oxygen saturation and blood pressure! Any questions?" I pointed to the fence, which is a towel partition that divides the surgeon's working area from the anaesthetist's working area. "Oh, that's what we call the blood-brain barrier", the anaesthetist said with a laugh. The surgeon scowled.

"Well, I'll be off then", said the anaesthetist, and I was left in charge of an unconscious patient. Alone with not even a sentinel canary! I was ticking along just fine, enjoying the sense of power of reading all these exotic instruments and adjusting them as required until I came to the point where the surgeon said, "He's a bit tight. Will you do something about it?" "Tight?" I thought. "Tight? I wouldn't mind being a bit tight right now, myself", I thought. So I let the patient have some more nitrous oxide and hoped this would do the job.

It didn't. "He's tight!" yelled the surgeon, scowling again. I gave the patient another blast of succinyl dicholine and that apparently did the trick. I didn't take this personally, mind you, because conflicts between surgeons and anaesthetists are legendary. I logged the patient's blood pressure and suddenly got curious as to what type of operation was going on. A quick scan of the patient's chart revealed that this was a male-to-female sex change operation, but the interesting comment in the history was that the patient had tried to do the job himself, once before. (If this had happened recently, I would have thought, "What Bobbittry is this?") Unable to contain my curiosity, I ventured outside the anaesthetist's fence and had a good look at what was going on in the surgical arena. Sure enough, a ring-shaped scar encircled the base of the man's penis from a previous self-directed attack. My cremaster muscles still contract at the very thought of it.

Eventually, the anaesthetist returned and I was able to relax. Anaesthesia is a very relaxing specialty, most of the time. It has been described as boredom punctuated by panic. Patients can go into cardiac arrest, vomit as they are being intubated and suffer many other sudden

Jason Bowels M.D. . Surgical Resident



By Dr. Michael Rieder

PrBIAXIN®

CLARITHROMYCIN

NAME OF DRUG: BIAXIN® (clarithromycin film-coated tablets) 250 mg

THERAPEUTIC CLASSIFICATION: Antibiotic

ACTION AND CLINICAL PHARMACOLOGY: BIAXIN exerts its antibacterial action by binding to the 50S ribosomal subunit of susceptible bacteria and suppressing protein synthesis. The absolute bioavailability of 250-mg clarithromycin tablets is approximately 50%. Food slightly delays the onset of clarithromycin absorption but does not affect the extent of bioavailability. Therefore, BIAXIN tablets may be given without regard to meals. In fasting healthy human subjects, peak serum concentrations are attained within 2 hours after oral dosing. Steady-state peak serum clarithromycin concentrations, which are attained within 2 to 3 days, are approximately 1 mg/L with a 250-mg dose twice daily and 2 to 3 mg/L with a 500-mg dose twice daily. The elimination half-life of clarithromycin is about 3 to 4 hours with 250 mg twice daily dosing, but increases to about 5 to 7 hours with 500 mg administered twice daily. The nonlinearity of clarithromycin pharmacokinetics is slight at the recommended doses of 250 mg and 500 mg administered twice daily. With 250 mg twice daily, the principal metabolite, 14-OH clarithromycin, attains a peak steady-state concentration of about 0.6 mg/L and has an elimination half-life of 5 to 6 hours. With a 500-mg twice daily dose, the peak steady state of 14-OH concentrations of clarithromycin are slightly higher (up to 1 mg/L) and its elimination half-life is about 7 hours. With either dose, the steady-state concentration of this metabolite is generally attained within 2 to 3 days.

INDICATIONS AND CLINICAL USES: BIAXIN may be indicated in the treatment of mild to moderate infections caused by susceptible strains of the designated microorganisms in the diseases listed below:
Upper Respiratory Tract: Pharyngitis/tonsillitis caused by *Streptococcus pyogenes* (Group A beta-hemolytic streptococci). Acute maxillary sinusitis caused by *Streptococcus pneumoniae*. **Lower Respiratory Tract:** Acute bacterial exacerbation of chronic bronchitis caused by *Streptococcus pneumoniae*, *Haemophilus influenzae* (including beta-lactamase-producing strains), *Moraxella (Branhamella) catarrhalis* (including beta-lactamase-producing strains). Pneumonia caused by *Streptococcus pneumoniae* and *Mycoplasma pneumoniae*. **Uncomplicated Skin and Skin Structure Infections** caused by *Streptococcus pyogenes*, *Staphylococcus aureus*. Appropriate culture and susceptibility tests should be performed prior to initiating treatment in order to isolate and identify organisms causing the infection and to determine their susceptibilities to BIAXIN. Therapy with BIAXIN may be initiated before results of these tests are known. However, modification of this treatment may be required once results become available or if there is no clinical improvement.

CONTRAINDICATIONS: BIAXIN is contraindicated in patients with a known hypersensitivity to clarithromycin, erythromycin, or other macrolide antibacterial agents. BIAXIN is contraindicated as concurrent therapy with astemizole, or terfenadine (See PRECAUTIONS: Drug Interactions).

WARNINGS: BIAXIN should be administered with caution to any patient who has demonstrated some form of drug allergy, particularly to structurally related drugs. If an allergic reaction to clarithromycin occurs, administration of the drug should be discontinued. Serious hypersensitivity reactions may require epinephrine, antihistamines, or corticosteroids. **Pregnancy:** BIAXIN should not be used in pregnancy except where no alternative therapy is appropriate, particularly during the first 3 months of pregnancy. If pregnancy occurs while taking the drug, the patient should be apprised of the potential hazard to the fetus. Clarithromycin has demonstrated adverse effects on pregnancy outcome and/or embryo-fetal development in monkeys, mice, rats, and rabbits at doses that produced plasma levels 2 to 17 times the serum levels obtained in humans treated at the maximum recommended doses. Pseudomembranous colitis has been reported with nearly all antibacterial agents, including macrolides, and may range in severity from mild to life threatening. Therefore, it is important to consider this diagnosis in patients who present with diarrhea subsequent to the administration of antibacterial agents, including BIAXIN. Treatment with antibacterial agents alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is a primary cause of "antibiotic-associated colitis." After the diagnosis of pseudomembranous colitis has been established, therapeutic measures should be initiated. Mild cases of pseudomembranous colitis usually respond to discontinuation of the drug alone. In moderate to severe cases, consideration should be given to management with fluids and electrolytes, protein supplementation, and treatment with an antibacterial drug effective against *Clostridium difficile*.

PRECAUTIONS: Clarithromycin is principally excreted by the liver and kidney (See DOSAGE AND ADMINISTRATION). In patients with both hepatic and renal impairments or in the presence of severe renal impairment, decreased dosage of BIAXIN or prolonged dosing intervals might be appropriate.

Drug Interactions
Theophylline: BIAXIN use in patients who are receiving theophylline may be associated with an increase of serum theophylline concentrations. Monitoring of serum theophylline concentrations should be considered for patients receiving high doses of theophylline or with baseline concentrations in the upper therapeutic range. **Carbamazepine:** Clarithromycin administration in patients receiving carbamazepine has been reported to cause increased levels of carbamazepine. Blood level monitoring of carbamazepine may be considered. The following drug interactions have not been reported in clinical trials with clarithromycin; however, they have been observed with another macrolide, erythromycin: Concomitant administration of erythromycin and digoxin has been reported to result in elevated digoxin levels. There have been reports of increased anticoagulant effects when erythromycin and oral anticoagulants were used concomitantly. Concurrent use of erythromycin and ergotamine or dihydroergotamine has been associated in some patients with acute ergot toxicity characterized by ischemic reactions. Erythromycin has been reported to decrease the clearance of triazolam and thus may increase the pharmacologic effect of triazolam. The use of erythromycin in patients concurrently taking drugs metabolized by the cytochrome P450 system may be associated with elevations in serum levels of these other drugs. There have been reports of interactions of erythromycin with cyclosporine, hexobarbital, and phenytoin. Serum concentrations of drugs metabolized by the cytochrome P450 system should be monitored closely in patients concurrently receiving erythromycin. **Terfenadine:** Macrolides have been reported to alter the metabolism of terfenadine resulting in increased serum levels of terfenadine which have occasionally been associated with cardiac arrhythmias. In a study involving 14 healthy volunteers, the concomitant administration of BIAXIN and terfenadine resulted in a two to three-fold increase in the serum level of the acid metabolite of terfenadine, MDL16,455, and in prolongation of the QT interval which did not lead to any clinically detectable effect (See CONTRAINDICATIONS). **Pregnancy:** There are no adequate and well-controlled studies in pregnant women (See WARNINGS). Four teratogenicity studies in rats (three with oral doses and one with intravenous doses up to 160 mg/kg/day administered during the period of major organogenesis) and two in rabbits (at oral doses up to 125 mg/kg/day or intravenous doses of 30 mg/kg/day administered during gestation days 6 to 18) failed to demonstrate any teratogenicity from clarithromycin. Two additional oral studies in a different rat strain at similar doses and similar conditions demonstrated a low incidence of cardiovascular anomalies at doses of 150 mg/kg/day administered during gestation days 6 to 15. Plasma levels after 150 mg/kg/day were 2 times the human serum levels. Four studies in mice revealed a variable incidence of cleft palate following oral doses of 1000 mg/kg/day during gestation days 6 to 15. Cleft palate was also seen at 500 mg/kg/day. The 1000 mg/kg/day exposure resulted in plasma levels 17 times the human serum levels. In monkeys, an oral dose of 70 mg/kg/day produced fetal growth retardation at plasma levels that were 2 times the human serum levels. Embryonic loss have been seen in monkeys and rabbits. **Nursing Mothers:** Clarithromycin is excreted in human milk. **Pediatric Use:** Use of clarithromycin tablets in children under 12 years of age has not been studied. **Geriatric Use:** Dosage adjustment should be considered in elderly patients with severe renal impairment. In a steady-state study in which healthy elderly subjects (age 65 to 81 years old) were given 500 mg every 12 hours, the maximum concentrations of clarithromycin and 14-OH clarithromycin were increased. The AUC was also increased. These changes in pharmacokinetics parallel known age-related decreases in renal function. In clinical trials, elderly patients did not have an increased incidence of adverse events when compared to younger patients.

ADVERSE REACTIONS: The majority of side effects observed in clinical trials involving 3563 patients treated with BIAXIN were of a mild and transient nature. Fewer than 3% of patients discontinued therapy because of drug-related side effects. During these clinical studies the following adverse reactions were reported: **BODY AS A WHOLE** - headache (2%), asthenia, infection, back pain, pain, and chest pain. **DIGESTIVE SYSTEM** - nausea (4%), diarrhea (3%), abdominal pain (2%), dyspepsia (2%), vomiting (1%), constipation, flatulence, dry mouth, stomatitis, gastrointestinal disorder, anorexia, oral moniliasis, and hepatomegaly. **NERVOUS SYSTEM** - dizziness, vertigo, nervousness, anxiety, insomnia, nightmares, somnolence, depression, confusion, and hallucinations. **RESPIRATORY SYSTEM** - rhinitis, cough increased, dyspnea, pharyngitis, and asthma. **SKIN AND APPENDAGES** - pruritus, rash, sweating; allergic reactions including urticaria, mild skin eruptions, and rare cases of anaphylaxis. **SPECIAL SENSES** - taste perversion (2%), ear disorder, abnormal vision, and conjunctivitis. **UROGENITAL SYSTEM** - hematuria, vaginal moniliasis, vaginitis, and dysmenorrhea. **HEMIC AND LYMPHATIC SYSTEM** - eosinophilia, anemia, leukopenia, and thrombocythemia.
Changes in Laboratory Values: Changes in laboratory values with possible clinical significance were as follows: **Hepatic** - elevated SGPT < 1%, SGOT < 1%, GGT < 1%, alkaline phosphatase < 1%, LDH < 1% and total bilirubin < 1%. **Hematologic** - decreased WBC < 1% and elevated prothrombin time (1%). **Renal** - elevated BUN (4%) and elevated serum creatinine < 1%.
Others: The following adverse reactions have not been observed in clinical trials with BIAXIN but they have been occasionally reported with erythromycin, another macrolide: Pseudomembranous colitis, hepatic dysfunction, cardiac arrhythmias such as ventricular tachycardia and torsades de pointes in individuals with prolonged QT intervals, central nervous system side effects (including seizures, hallucinations, confusion, and vertigo), anaphylaxis, and reversible hearing loss occurring chiefly in patients with renal insufficiency and in patients receiving high doses of erythromycin. In studies of pneumonia comparing clarithromycin to erythromycin base or erythromycin stearate, there were significantly fewer adverse events involving the digestive system in patients treated with clarithromycin.

SYMPTOMS AND TREATMENT OF OVERDOSAGE: Reports indicate that the ingestion of large amounts of clarithromycin can be expected to produce gastrointestinal symptoms. Adverse reactions accompanying overdosage should be treated by the prompt elimination of unabsorbed drug and supportive measures. Clarithromycin is protein bound (70%). No data are available on the elimination of clarithromycin by hemodialysis or peritoneal dialysis.

DOSAGE AND ADMINISTRATION: BIAXIN may be given with or without meals. **Adults:** The usual adult dosage is 250 mg to 500 mg every 12 hours for 7 to 14 days.

	Dosage Guidelines
Infection	**Dosage (BID)**
Upper Respiratory Tract	250-500 mg
Pharyngitis/Tonsillitis	250 mg
Acute maxillary sinusitis	500 mg
Lower Respiratory Tract	250-500 mg
Acute exacerbation of chronic bronchitis and pneumonia	
Uncomplicated Skin and Skin Structure Infections	250 mg

For more severe infections or those caused by less susceptible organisms, the upper dosage should be used. In the treatment of Group A streptococcus infections, therapy should be continued for 10 days. In patients with renal impairment and a creatinine clearance less than 30 mL/min., the dosage of BIAXIN should be reduced by one-half, i.e., 250 mg once daily, or 250 mg twice daily in more severe infections. Dosage should not be continued beyond 14 days in these patients. In patients with both hepatic and renal impairments or in the presence of severe renal impairment, decreased dosage of BIAXIN or prolonged dosing intervals may be appropriate.

Composition: BIAXIN contains 250 mg clarithromycin per tablet. Non-medicinal ingredients: cellulosic polymers, crosscarmellose sodium, D&C Yellow No. 10, magnesium stearate, povidone, propylene glycol, silicon dioxide, sorbic acid, sorbitan monooleate, pregelatinized starch, stearic acid, talc, titanium dioxide and vanilin. BIAXIN does not contain tartrazine.

Storage Recommendations: Store tablets at controlled room temperature (15° to 30°C) in a well-closed container. Protect from light.

AVAILABILITY OF DOSAGE FORMS: BIAXIN is supplied in HDPE bottles of 100 and 250 tablets as yellow, oval, film-coated tablets containing 250 mg of clarithromycin.

MICROBIOLOGY: Clarithromycin is active *in vitro* against various aerobic and anaerobic gram-positive and gram-negative organisms, as follows: **Gram-positive Aerobes:** *Streptococcus pyogenes*, *Streptococcus pneumoniae*, *Streptococcus agalactiae*, *Listeria monocytogenes*. **Gram-negative Aerobes:** *Moraxella catarrhalis*, *Neisseria gonorrhoeae*, *Neisseria meningititis*, *Campylobacter jejuni*, *Legionella pneumophila*, *Haemophilus influenzae*, *Bordetella pertussis*. **Other Aerobes:** *Mycoplasma pneumoniae*, *Chlamydia trachomatis*, *Mycobacterium avium intracellulare*. **Gram-positive Anaerobes:** *Clostridium perfringens*, *Propionibacterium acnes*. **Gram-negative Anaerobes:** *Bacteroides fragilis*. Additionally, the 14-OH clarithromycin metabolite also has significant antimicrobial activity which may be additive to the activity of the parent compound. Against *Haemophilus influenzae*, 14-OH clarithromycin is twice as active as the parent compound *in vitro*. Beta-lactamase production should not have any effect on clarithromycin activity.
Susceptibility Testing: The standard single disc susceptibility test (using the 15-µg clarithromycin disc) and the dilution susceptibility test should be interpreted according to the criteria in the following table.

	Zone Diameter (mm)	Appropriate MIC Correlate (mg/L)
Susceptible	≥ 18	≤ 2
Intermediate[1]	14 - 17	4
Resistant	≤ 13	≥ 8

[1] Indicates that the test results are equivocal; therefore, dilution tests may be indicated.
N.B. These criteria and the definition are in agreement with NCCLS. Order Code M2A4.[2]
Control limits for monitoring clarithromycin susceptibility tests are given in the following table.

		MIC (mg/L)
S. aureus	ATCC 29213	0.06 - 0.25
E. faecalis	ATCC 29212	0.25 - 1.0

A report of "Susceptible" indicates that the pathogen is likely to respond to monotherapy with clarithromycin.
[2] National Committee for Clinical Laboratory Standards, Performance Standards for Antimicrobial Disc Susceptibility Tests - Fourth Edition. Approved Standards NCCCLS Document M2A4, Volume 10, No. 7 NCCCLS, Villanova, PA 1990.
A report of "Intermediate" indicates that the result be considered equivocal and, if the organism is not fully susceptible to alternative clinically feasible drugs, the test should be repeated. This category provides a buffer zone which prevents small uncontrolled technical factors from causing major discrepancies in interpretations. However, standardized diffusion methods for routine *in vitro* susceptibility testing, using the 15-µg clarithromycin disc, do not measure the antimicrobial activity of the 14-OH metabolite and thus may not be predictive of the drug's potential activity against *Haemophilus influenzae*. *Haemophilus influenzae* isolates falling into the "intermediate" category may respond to treatment. A report of "Resistant" indicates that achievable drug concentrations are unlikely to be inhibitory, and other therapy should be selected.

References:

1. BIAXIN Product Monograph.

2. Dabernat H, Delmas C, Seguy M et al. The activity of clarithromycin and its 14-hydroxy metabolite against *Haemophilus influenzae*, determined by *in vitro* and serum bactericidal tests. *J Antimicrob Chemother* 1991;27(suppl A):19-30.

3. Hardy DJ, Swanson RN, Rode RA et al. Enhancement of the *in vitro* and *in vivo* activities of clarithromycin against *Haemophilus influenzae* by 14-hydroxy-clarithromycin, its major metabolite in humans. *Antimicrob Agents Chemother* 1990;34(7):1407-13.

4. Karma P, Pukander J, Penttilä M et al. The comparative efficacy and safety of clarithromycin and amoxicillin in the treatment of outpatients with acute maxillary sinusitis. *J Antimicrob Chemother* 1991;27(suppl A):83-90.

5. Neu H, Craft JC. Clarithromycin vs. cephalosporin therapy for the treatment of *H. influenzae* bronchitis. Abstract. Presented at the 1st International Conference on the Macrolides, Azalides and Streptogramins. Santa Fe, NM, Jan. 1992.

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PAAB

mishaps. This may be why there has been such a high incidence of substance abuse disorders among anaesthesiologists in the United States. Nevertheless, Anaesthesia was always popular as final destination among the crowd who specialty shopped (in the old days, one could qualify for several specialties just by continuing to enroll in different residency programs), sort of like channel surfing.

The big problem with Anaesthesia is that you have to get up so early in the morning because that's when the surgeons are up to do their operating, yet still manage to intubate while both you and the patient are sleeping. You do get to witness some incredible performances as an upside, if you enjoy that sort of thing (see Surgical Silliness, last issue). For instance, there was the time a surgical resident got 80 mg of Lasix along with his morning coffee. About ten minutes into the first procedure, his forehead broke out in sweat beads, and shortly he began shifting his weight from foot to foot in rhythmic fashion. After another few minutes, he asked the surgeon to be excused and he scrubbed out. Shortly, he returned, freshly scrubbed and regloved. Ten minutes later, he had more sweat beads and he began his little dance again. Again, he was excused, scrubbed out and was back after the pause that refreshes. After ten minutes, he was again in worse shape than "Swing Kids". He cried out "Oh Dr. _____! I don't know what's wrong, but I've got to go to the washroom again!" The surgeon scowled again and said, "Somebody Lasixed your coffee, you idiot! Now piss off and don't come back!" The resident was only too happy to oblige. The next morning, he drank three litres of orange juice in one continuous draught.

Another morning, a surgical team was operating on an elderly lady while I looked after the anaesthetic. One of the surgical residents, right in mid-sentence, sneezed twice quickly, said the word "and", and then sneezed again. To this, I replied, "Gesundheit, Gesundheit and Gesundheit." The resident then said, "Thank you, thank you and thank you", with just a touch of sarcasm in his voice. I rejoined, "You're welcome, you're welcome, and you're welcome." This was too much for the staff surgeon who said, "F@#$$ off, f@#$ off and f@#$ off!" The patient then piped up, "Doctor! That's no way to speak to that poor gentleman." The surgeon had forgotten that his patient was under local anaesthetic.

It was about at this point that I decided not to be a professional gas-passer as my life's work. Besides, I can pass gas recreationally any time I like! Ω

THINKING ON YOUR FEET

"Thinking On Your Feet" is an exercise designed to test your clinical decision-making skills based on a case presentation. Below is an exciting patient history with physical examination and laboratory results. Answers may be found on page 108 or within the case itself. Although this section is called "Thinking On Your Feet," thinking is acceptable in any position. Answer the questions in sequence. Please do not peek at the answers prematurely—this would not be ethical. Compare your responses with those of the case presenter. Award yourself one point for each match. The author of this issue's case study is Dr. Javed Sidiqi, a senior resident in neurosurgery at the University of Western Ontario. Dr. Sidiqi is a Rhodes Scholar who completed a degree in Political Science while in England, he travels regularly and has recently published a book on the World Health Organization.

Scoring:

85 - 100% — Excellent. Patient is recovering nicely;
72 - 84% — Strong Work. Patient is recovering—minor complications;
59 - 71% — Good Eye. Patient likely to survive—some disfigurement;
40 - 58% — Fair. Patient requires critical care & close monitoring;
0 - 39% — Uh-Oh. Red alert—emergency crisis—check A-B-C's.

A CASE OF UNILATERAL HEARING LOSS

A previously healthy 50 year old right-handed female business executive finds increasing difficulty hearing on the telephone for the past two years. She has had a ringing in her right ear for as long as she can recall. Other complaints include right cheek numbness for six months and imbalance for several weeks. Past medical history includes left eye blindness from birth.

PLEASE STOP AND ANSWER THESE QUESTIONS:

1. How do you classify hearing loss? How do you distinguish between these types clinically?

2. How likely is this hearing loss due to normal ageing?

3. Where do you localize the lesion?

4. What is the gold standard in investigations for this area?

PLEASE CONTINUE...

Sensorineural hearing loss is detected on the right side on formal testing. MRI of the head (with and without contrast) shows a 3 cm wide extra-axial space occupying lesion (SOL) in the cerebellopontine angle with invasion into the internal auditory canal. The radiologist reports "consistent with acoustic neuroma."

PLEASE STOP AND ANSWER THESE QUESTIONS:

5. Is the history consistent with the radiologic diagnosis?

6. What is the differential diagnosis for a SOL in the region specified in the MRI scan?

7. If the patient were 20 years old, with all else the same, how would your clinical judgment be influenced?

PLEASE CONTINUE...

Assuming the radiologic diagnosis is indeed correct.

PLEASE STOP AND ANSWER THESE QUESTIONS:

8. What is the therapy of choice in this patient?

9. What is the mechanism of the hearing loss and imbalance?

10. Why is the name given to this lesion a misnomer?

PLEASE CONTINUE...

The tumour is resected completely by a neurosurgeon, though not without some difficulty. Post-operatively, the patient is awake and alert but has some difficulty completely closing the right eyelid.

PLEASE STOP AND ANSWER THESE QUESTIONS:

11. How do you explain the eyelid problem?

12. Presuming slow recovery to normal, which specific treatment is critical for management of the eyelid problem?

MEDICAL VOCABULARY

This section is designed to test and expand your knowledge of medical terminology. How many items can you correctly define?

Scoring: [13-15]=Superior knowledge, [10-12]=Above average, [8-9]=Adequate, [5-7]=Fair, [1-4]=Sub-par.

1. **Athetoid movements**
a) slow, sinuous, aimless movements especially in the distal musculature of limbs
b) brisk, jerky and purposeless movements, resembling fragments of voluntary movements
c) rapidly alternating movements which are performed in a clumsy manner
d) intermittent or jerky movements

2. **Hypsarrythmia**
a) most common interictal EEG pattern associated with infantile spasm
b) commissure of the superior colliculi
c) saccadic eye movements
d) inability to read more than a few sentences with understanding

3. **Prosopagnosia**
a) defect in understanding the significance of sensory information
b) imparied recognition of previously known and familiar faces
c) poor verbal repitition skills with good comprehension and spontaneous speech
d) inability to perform useful motor tasks previously mastered in the absence of overt defiency

4. **Coma depasse**
a) fluent but circumlocutory speech caused by word finding difficulties
b) state beyond coma or brain death
c) central tegmental tract
d) encephalopathy involving the cerebellum

5. **Tic douloureux**
a) paroxysms of pain in the area of distribution of one of the divisions of the trigeminal nerve with periods of remission and exacerbation
b) interruption of the motor fibres of the trigeminal nerve leading to paralysis and eventual atrophy of the muscles of mastication
c) mask-like facies seen typically in Parkinson's disease
d) muscles innervated by the trigeminal nerve

6. **Gemmule**
a) giant pyramidal cell
b) dendritic spine
c) an interneuron
d) secondary sensory ending

7. **Torticollis**
a) lack of reaction of pupils to light
b) grimacing, protrusion and writhing of tongue associated with difficulty in speaking and swallowing
c) tilt and rotation of head to one side and restricted rotation to the other side
d) uncontrollable laughing or crying without cause

8. **Opsoclonus**
a) several groups of neurons situated between the anterior end of the temporal horn of the lateral ventricle and the ventral surface of the lentiform nucleus
b) neuronal degeneration in the corpus striatum
c) chaotic conjugate jerking of eyes
d) trigeminal neuralgia

9. **Amyoplasia**
a) congenital absence of an individual muscle
b) hepatolenticular degeneration
c) puppet-like movements due to the separation of voluntary movements that normally flow smoothly in a sequence
d) extensive folding of the the cerebellar surface

10. **Nemaline rods**
a) funnel shaped clefts in myelin sheaths
b) abnormal rod-shaped inclusion like structures within muscle fibres
c) slender bundles of fibres originating from the amygdaloid body
d) atypical astrocytes

11. **Ramsay Hunt syndrome**
a) tumor of the cerebellopontine angle
b) geniculate herpes
c) idiopathic unilateral facial paralysis of sudden onset
d) leukemic invasion of the seventh cranial nerve

12. **Arthrogryposis multiplex congenita**
a) generalized muscular hypertrophy with weakness
b) lesion of thalamus leading to abnormal sensation to touch, pain and temperature
c) cerebellar dysfunction, characterized by unsteady wide based gait
d) multiple congenital contractures

13. **Internuclear ophthalmoplegia**
a) centrifugal fibres in the optic nerve
b) inability of ipsilateral eye to adduct when the contralateral eye is abducted due to a small lesion in the medial longitudinal fasiculus
c) lesion of the fibres from the superior olivary nucleus which permit the reflex contraction of the stapedius muscle
d) paralysis of the stapedius muscle leading to hyperacusis

14. **Neurobiostaxis**
a) stimulation of cells in the superior salivatory nucleus
b) respiratory function of aquatic gill arches
c) tendency for neurons to migrate toward major sources of stimuli
d) contractile cells associated with capillary blood vessels in the central nervous system

15. **Medial medullary syndrome**
a) occlusion of medullary branch of vertebral artery
b) disorder characterized by skeletal muscle wasting due to progressive degeneration of cells in the anterior horn cells and medulla
c) motor neuron disease characterized by progressive degeneration of corticospinal tracts and anterior horn cells or bulbar efferent neurons
d) destruction of the nucleus ambiguous

ANSWERS TO CASE STUDY

1. Hearing loss can be classified as conductive or sensorineural. Patients with conductive hearing loss (CHL) usually have normal speech, compared to the loud voice of those with sensorineural nearing loss (SHL). Rinne and Weber tests should be done on all patients with hearing loss. In CHL, the Rinne is abnormal, showing air conduction hearing to be decreased with respect to bone conduction, while the Weber test lateralizes to the side of the hearing loss. In the SHL group, the Rinne is normal and the Weber lateralizes to the side with better hearing.

2. Unilaterality and associated findings such as tinnitus make the possibility of age-related hearing loss very unlikely.

3. Cerebellopontine angle.

4. MRI head with and without contrast is the gold standard, though a CT head is sometimes all that is available.

5. Yes. Unilateral SHL followed by Vth nerve involvement and vestibular symptoms suggests acoustic neuroma until proven otherwise.

6. 80% acoustic neuromas, 10% meningiomas, 5% epidermoids, 5% other (eg. arachnoid cyst, metastases, etc.).

7. Always consider neurofibromatosis (NF) in young patients with unilateral acoustic neuromas. A thorough family history and complete physical exam in search of hints of NF is essential.

8. Surgical resection.

9. Hearing loss is thought to be due to mass effect of the tumour on the auditory nerve, and/or ischemia of the nerve. Imbalance is from similar involvement of the vestibular nerve.

10. The tumour arises from the nerve sheath of the superior vestibular nerve, and should properly be referred to as a vestibular schwannoma.

11. The facial nerve has been injured intraoperatively.

12. Regular use of artificial tears prevents drying of the cornea and subsequent infections. A protective plastic eye "bubble" or tarsorraphy may be indicated where recovery is delayed or unlikely. Visual loss in the right eye would be particularly devastating in this patient as she is already blind in the other eye.

ANSWERS TO MEDICAL VOCABULARY

1. **Athetoid movements**
a) slow, sinuous, aimless movements especially in the distal musculature of limbs

2. **Hypsarrythmia**
a) most common interictal EEG pattern associated with infantile spasm

3. **Prosopagnosia**
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b) state beyond coma or brain death

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b) dendritic spine

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c) tilt and rotation of head to one side and restricted rotation to the other side

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14. **Neurobiostaxis**
c) tendency for neurons to migrate toward major sources of stimuli

15. **Medial medullary syndrome**
a) occlusion of medullary branch of vertebral artery

NEWS ABOUT TOWN

STUDENT SYMPOSIUM IN MEDICAL RESEARCH

The final symposium of the Summer Research Training Program (SRTP) was held on September 10, 1994 at the Medical Sciences Building. The event was attended by approximately 24 first and second year medical students and their summer research supervisors. The students and faculty members (although a few unexpected spectators were also present for the free juice and cookies) heard numerous interesting presentations from second-year students. Each student's presentation, which was based on his/her research over the last two summers, was evaluated by participating faculty members and students for selecting the SRTP awards. The top prize includes $1500 to assist the recipient in presenting his/her research at the National Student Research Forum in Galveston, Texas. This year's winner was Stephanie Bennett of Meds '96. The SRTP is a program for first and second year medical students in the Faculty of Medicine at Western who would like to work under the supervision of a faculty member on a research project for two summers.

OSLER SOCIETY REVIVED

After a 17-year hiatus, the U.W.O. Faculty of Medicine once again has a student medical history society. The Osler Society-a revival of the original name-has been resurrected thanks to the efforts of a small group of students early in the academic year. The society was formed originally in 1927 as an honour society by Dr. J.W. Crane, one of Western's first full-time professors of medicine.

From the beginning, the society met to discuss matters of medical history. Each year an emminent medical historian gave a presentation relating some important contributions made by Sir William Osler. Osler was a Canadian-born pioneer in the development of medical education. Over the years, unfortunately, the membership gradually declined, and in 1977 the Osler Society was formally dissolved.

This year, a small group of medical students, with guidance of Dr. Paul Potter from the Department of History of Medicine, have banded together to explore medicine's colourful history through such activities as films, book discussions, field trips, and guest speakers. Inquiries can be forwarded to this year's president, David Martell (Meds'97).

ABOUT THE AUTHOR:

Bao Tang is a second year medical student at the University of Western Ontario.

ARTHRITIS SOCIETY CAMPAIGN

On Thursday October 13, 1994 more than 20 medical students from Meds '96 and '97 hit the streets in an effort to raise money for the London Arthritis Society. For the second year in a row, organizer Ron Curtis (Meds'96) was very happy with the interest and commitment of all involved. This year we raised over $880, surpassing last year's total of $803, for the Castlegrove area of London (total raised for all London in 1993 was $77,000!). Leanna from Meds '97 had the highlight of the night by catching a glimpse of some fine male anatomy at one of the houses she visited! After our door-to-door canvassing, most of us returned to Kelsey's for a few brewskies and some wicked Nachos. All in all, it was a fun and profitable night. Thanks to all those involved and we look forward to your continued support next year. For those interested, the Arthritis Society is located on Florence Street in London, and the regional director, Ms. Bonnie Spencer-Beer would be happy to talk with you. Look them up!!

UNDERGRADUATE MEDICAL CURRICULUM CHANGE?

The Steering Committee on Curriculum Revision was recently established by the Faculty of Medicine at Western. The committee, chaired by Dr. Michael Clarke (Dept. of Microbiology and Immunology), consists of 5 faculty members and 3 students. Its primary purpose is to gather data on the current undergraduate medical curriculum, and to form a plan for possible future changes.

The current curriculum, which was introduced in September 1990, is a mixture of some conventional lecture-based learning and some problem-based learning (PBL). Although these significant changes have brought an overall positive effect on the students' education, there are still a number of problems that exist, or have developed as the result of changes in licensure requirements: 1) the current PBL curriculum has reduced the length of the fourth year, and delayed the start of clerkship; this situation has negatively impacted students during the application process for residency positions; 2) the PBL has evolved into a "self-standing course" rather than an intended method of learning; also, there appears to be little or no integration of PBL into the rest of the curriculum; 3) there are deficiencies in teaching in certain relevant fields of medicine such as geriatrics, oncology, palliative care, rehabilitation, medical ethics, and primary/ambulatory care; and 4) information overload is still a recognized problem in the current curriculum.

The Committee is currently looking closely at these and other issues, and it welcomes input from faculty members and students. By early 1995, the Committee hopes to have some recommendations on curriculum improvement for the Undergraduate Medical Education Council.

MEDICAL FAX

DAMAGING THE BRAIN TO PROMOTE HEALING

Fish, unlike humans, have the ability to repair damage to the central nervous system (CNS). Using a compound taken from fish, researchers have managed to stimulate growth and synaptic formation between the cut ends of optic nerves in mice. This compound consists of interleukin-2 (an immune signal produced at sites of inflammation) and an enzyme called nerve-derived transglutaminase. Researchers hypothesize that CNS repair is inhibited by oligodendrocytes which produce a factor that suppresses axon elongation. The compound exerts its regenerative effects by selectively killing oligodendrocytes. Tests have indicated that these new axons are funtional and able transmit signals. However, it is not known whether these signals are perceived as visual information.
Scientific American. 271(4): 31-2 (1994).

PROTEIN AIDS NERVE REGENERATION

Studies contrasting the regenerative capabilities of the peripheral and central nervous system have lead to an interesting finding. A protein called myelin-associated glycoprotein (MAG) inhibits the growth of neurites in cell culture. This protein is present in the myelin of both central and peripheral nerves. MAG levels in the PNS , however, are one tenth of that in the CNS and it has been suggested that these reduced levels may allow for increased regeneration in the periphery. Furthermore, peripheral nerves clear away surrounding compounds more quickly following injury than do central nerves. Following this rationale, researchers applied anti-MAG antibodies to cultured central neurons and found that neurite growth was largely restored.
Science. 265(23): 1800-1 (1994).

DYSLEXIA GENE FOUND?

The combined efforts of four research centers may lead to the discovery of the gene responsible for dyslexia, a reading disorder that could affect up to 20% of children. The location has been narrowed to a region on chromosome six. Such a finding may lead to screening tests for children and therefore allow earlier intervention. Currently, those affected are not discovered until the third grade. At this stage, successful intervention is only witnessed in about 20% of the cases.

ABOUT THE AUTHOR:

Yacov Rabi is a second year medical student at the University of Western Ontario..

BORON THERAPY ON THE HORIZON

Boron therapy was a proposed treatment for cancer until it was discontinued over 30 years ago. The treatment was believed to be ineffective and even lethal to some patients. Recently a woman suffering from incurable brain cancer was the first to receive boron treatment in the U.S. in 33 years. The procedure has improved significantly over the past decades. The theory behind the treatment is that boron will selectively concentrate at the tumor site where the boron nuclei capture neutrons. As a result, the boron nuclei split, thereby releasing energy which kills the cancer cells. Preliminary animal studies have shown that boron treatment is able to cure rats that have had tumors artificially implanted. The patient appeared to be in good condition as of the publication date of this paper, however, her doctor warns that complications might still arise.

STROKE-DAMAGE REDUCED BY IMMUNOPHILINS

FK506 is a potent immunosuppressive drug that acts by binding with intracellular proteins known an immunophilins. This results in an inhibition of intracellular calcium binding which is necessary for generating the signals that stimulate T cells. It is believed that this inhibition of calcium is a crucial step since calcium may have an important role in neurodegeneration. Researchers have found that FK506 given at doses used for immunosuppression is able to reduce the damage resulting from focal cerebral ischemia by 63% when given within one hour of onset.
Nature. 371(22): 336-9 (1994).

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1. Cardizem® Product Monograph. 2. Ayd F: Once-a-day neuroleptic and tricyclic antidepressant therapy. Int Drug Ther Newsletter 1972;VII(9,10):33-40. 3. IMS Canada: Compuscript and Canadian Disease and Therapeutic Index: MAT June 1994.