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The Medical Model

UNDER ATTACK

We live in interesting times! As physicians, we have come under close scrutiny, by society, government and even ourselves as everyone worries about whether we can afford our health care system. Until the last 4 to 5 years, this system has had smooth sailing but, in retrospect, it was a time of approaching storm.

The issues are many and most of us are aware of them including potential underfunding of health care, changing methods (and lesser amounts) of physician payments, restriction of bargaining rights for doctors, and possible rationing with decreasing levels of care. Even the medical model is being questioned as we examine other alternatives, often ill defined and unproven in effectiveness and costs. As physicians, we of course have representatives who attempt to carry our opinions forward in these issues and indeed, take stands. This is difficult without proper communications occurring both in and outside of the profession. Now, more than ever before, clearly stated policy and justification for the medical profession is becoming important.

Suggestions that funds be removed from our "sickness system" to fund preventive and wellness programs that are sometimes unproven, are something most doctors cannot understand. Most of us believe that physicians already do much prevention and wellness counselling; even more disturbing is to understand that funding for emergency medical services, mental health, medication for the poor and elderly, acute care beds, is inadequate. Dalhousie Medical School, where many of us learned the medical model, is in constant financial need and is having difficulty maintaining programs and research.

While remaining open to change, we must also ensure basic programs are maintained and funded. It is difficult not to sound obstructive, as the medical model is attacked. Now, more than ever, we are being called upon to justify the basic premises of our profession. The justification that is to us obvious, will have to be communicated in every way possible, using both the lay press and our own publications. *Informed* is, of course, one vehicle and this *Journal* is another. Once again an invitation is issued to physicians to use this *Journal*, especially, possible guest editorials as a means of stimulating discussion and explaining positions. Also, rational, logical well supported articles in the lay media would seem to be urgently needed if our treatment systems for the ill of this province are to be maintained. Dependence on our present information system will not be enough and doctors in all specialties in all areas of the Province will have to take an interest in justifying our existence. It is as basic as that. □

J.F. O'C.

Barbara Blauvelt

FORTY-FIVE YEARS OF DEDICATED SERVICE

Dorothy A. Grant*

"I'll never forget that day! It was January and bitterly cold. I had left the Y.W.C.A. in Halifax and walked up Morris Street looking for Dalhousie University's Medical School. I got lost and went to the wrong place. Fortunately, some kind soul pointed me in the right direction and I found the school which at that time was located in the Public Health Clinic."

Barbara Blauvelt creates a vivid picture of her first day at Dalhousie University. She smiles when she talks about this memory and admits that when she arrived in Halifax in 1948 she had only planned to stay two weeks. Little did she know that she was about to begin a career that would last almost forty-five years and would result in her making a significant contribution to the university's Medical School and its Alumni office. Now, as she contemplates retirement, she fondly recalls how it all came about.

Born near Yarmouth, Barbara grew up in Darling Lake which she describes as "a dear place." Proudly, she remembers walking two and a half miles to and from school each day. "In winter too," she emphasizes, "We didn't have school buses."

After graduating from high school, she worked for a short time at a department store in Yarmouth. Later, she accepted a secretarial position with four local physicians.

One day close to Christmas 1947, Barbara happened to meet a Halifax doctor. He was Dr. Harry Grant, the Dean of Medicine at Dalhousie University. At the time, he had a serious problem. His secretary was recuperating from a bad accident and she was finding it hard coping with two jobs: handling her Medical School responsibilities and her duties as the secretary to Dr. Grant who was the Executive Secretary of The Medical Society of Nova Scotia.

One of doctors Barbara worked for felt very sorry for Dr.Grant, who was a good friend of his. The Yarmouth physician suggested his very efficient secretary consider assisting the Dean of Medicine. She agreed saying she would be happy to help out - "for a few weeks."

In fact, the "temporary" arrangement worked out extremely well. Barbara soon realized she had no interest in returning to Yarmouth. Almost immediately, she also came to the realization that being Dr.Grant's secretary was an extremely demanding job. Her responsibilities included performing all the secretarial work for Medicine, Surgery, Paediatrics, and all specialities with the exception of Obstetrics and Psychiatry. She also prepared exams for all of the medical students. Looking back, she says she often working well into the evening frequently staying at her desk until eight or nine at night.

"Many a night the janitor would find me in my office at the Public Health Clinic and insist on escorting me to the place where I got the tram car home."

As retirement rapidly approaches, she is able to chronicle the many changes that have occurred during the years she has spent at Dalhousie University. As she points out, the five Deans of Medicine she has served had a major impact on the evolution of the Medical School.

"Dean Grant - I greatly respected and had the highest regard for him. Dean Chester Stewart - he was a great innovator and had tremendous ability. Dean Lloyd Macpherson - he and his wife were close friends of mine. He was also a conciliator who brought stability to the Medical School. He was also responsible for instituting representation of students on Committees. Dean Donald Hatcher - medical research was his forté. Dr T.J. (Jock) Murray - he excelled in the Humanities."

Barbara Blauvelt delights in talking about some of the famous people she met during her years at Dal's Med School. Meeting Dr. Wilder Penfield was a particularly memorable occasion. She glows when she recalls the day she was presented to the Queen Mother. Many of the physicians she met during her time at Dalhousie have also earned a special place in her recollections.

"Dr. Charles Gass, of Tatamagouche. He was very active in founding the College of Family Practice. And then there was Dr. Harold "Benge" Atlee. He was a colourful character. Dr. Bob Jones also stands out as does Dr. Harris Miller. Dr. Miller was a veteran of the Second

* Director of Communications and Public Affairs, The Medical Society of Nova Scotia.

World War when he entered Medical School. He is a marvelous man. Dr. Murray Fraser was a fine family doctor and Dr. John Wickwire, he is a jewel of a man."

But it is when Barbara speaks of the many medical students she has known that one notes her real admiration.

She recalls with sorrow a number of students who died tragically while in medical school. Others stand out because of their courage. "I'll never forget one young man who was having a very bad time. When we investigated, we learned he was living in a tent in Bedford. He did not have any money for clothes or food. Of course, we were very upset and with the help of Dr. Tabby Bethune at the Victoria General, we got him a room and made sure he had regular meals. He is now retired and lives in New York state."

In 1981, Barbara Blauvelt left the Dean of Medicine's office to turn her attention to Alumni office endeavours.

No one was surprised when she instituted changes that had a dramatic effect on the office's profile. Prior to her arrival, contributions were small. Under her guidance, yearly gifts rose substantially. In the process, she has managed to develop detective skills that even Sherlock Holmes would envy! She has been able to trace almost every alumnae member and has made a point of maintaining close contact with them.

During the years she has focused her attention on Alumni affairs, gifts from alumnus have made a significant contribution to the Kellogg Health Science Library and audio-visual departments and to many other areas of the Medical School including the creation of a number of entrance and other scholarships. Barbara fervently hopes that after she retires, this kind of outstanding philanthropy will continue to be nurtured.

Barbara Blauvelt admits that she has not been completely happy with all of the changes that have occurred at the Medical School during her tenure. She says she misses the days when classes were smaller and everyone knew each other. She also believes that the huge amount of knowledge contemporary medical students have to absorb places a tremendous strain on them.

She does have one regret, "I would like to have had a family and I am sorry that never happened. But I take pride in the knowledge that thousands of students have gone through medical school during my years at Dalhousie University. I believe they all knew my door was always open to them. I don't think there is one of them I couldn't put a face to. They were my life."

Barbara Blauvelt's life at Dalhousie University will soon wind down but she already has plans. She wants to begin writing a history of the Medical School. Hopefully, it will also chronicle her fourty-five years service as secretary to five Deans of Medicine as well as her dedicated work at the Alumni Office.

Without doubt, her book is bound to make for some very engrossing reading. □

Correspondence

To the Editor:

DISABILITY INSURANCE COVERAGE

I am writing this letter on behalf of the members of the Dartmouth Medical Society, a Branch of the N.S. Medical Society, to express some concerns that were raised at a recent meeting about the absence of comprehensive disability insurance for HIV/HBC/HCV infections. We are greatly disturbed that our current insurance does *not* cover us unless we are medically disabled by these diseases. Although ethically and morally obliged to report ourselves if infected, we receive no compensation for curtailment of or removal of our hospital privileges while we wait for our practices to dry-up as word inevitably gets around to our patients. All because these infections are not considered disabling, except in their final or acute stages. The cases of Dr. Yabsley and Eric Smith clearly demonstrate that society is not yet ready to allow infected professionals to continue to practise, even if it is safe to do so. These are the realities, despite position papers by the CMA and NSMS, who tell us that it is safe to practise and that we have the right to continue. Without patients or a hospital affiliation, this becomes a farce.

A consensus was reached on four major points at this meeting: 1) That physicians in Nova Scotia should be made aware of the fact that they are not insured for these infections and the potential ramifications. Therefore, they should think twice if they are being asked for routine or non-essential serology screens: 2) Our insurers, especially the OMA Group, should be made aware of our demands for appropriate coverage in this area and our willingness to pay extra and/or go elsewhere to get it; 3) A fund should be negotiated between the NSMS and the government to allow for retraining and/or relocation for infected physicians, such that they can continue as productive members of society and 4) This lack of protection should be made clear to the N.S. Government and the public at large as a point that is not being addressed at the current negotiations. The risks we run should be very much a part of these negotiations.

In conclusion, this is an emotive issue for those of us who staff the Emergencies and Operating Rooms of this province, placing our health and very lives at risk every day. We deserve protection and assistance if we suffer grievous harm as a direct result of our professional activities. The current state of affairs is dangerously inadequate and something must be done now!

Sincerely,

M.W. Ellis (Pres.)
Dartmouth Medical Society
176 Portland St.
Dartmouth, N.S. B2Y 1J3

□

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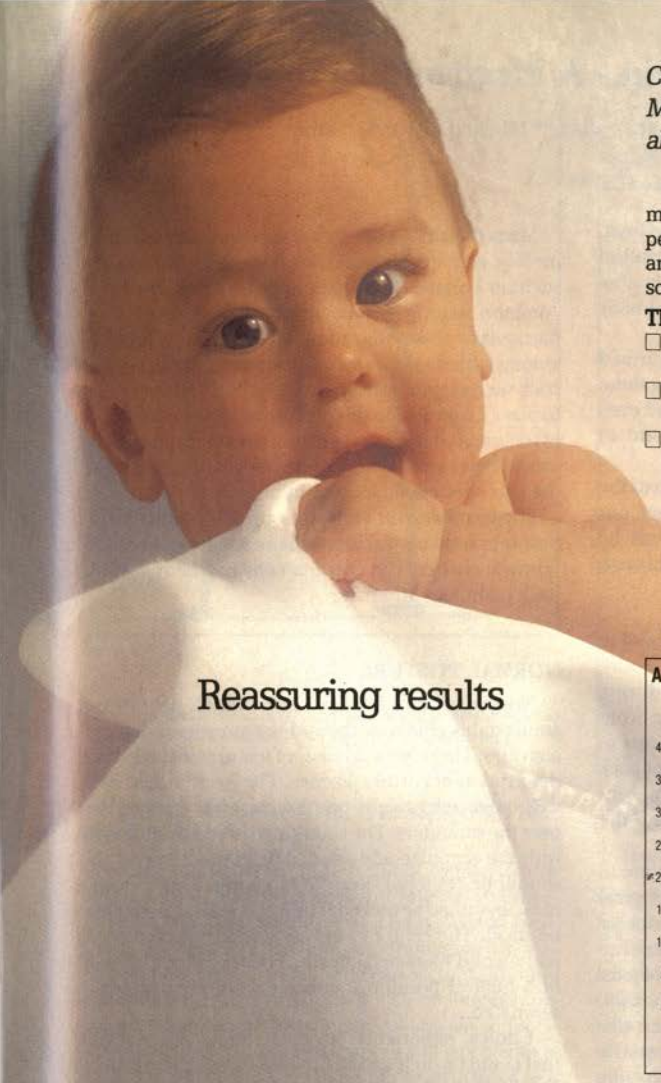


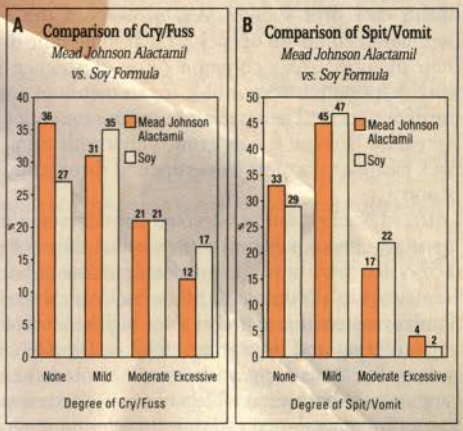
Reassuring results

Canadian clinical study confirms: Mead Johnson Alactamil is an acceptable alternative to soy-based formula.*[1]

Recently, 84 "fussy" children, aged one to six months, were recruited from five Canadian pediatric practices to compare their acceptance and tolerance of Mead Johnson Alactamil with soy-based formula.

The results:

- ☐ Degrees of Crying/Fussing were not clinically different. (Chart A)
- ☐ Degrees of Spitting/Vomiting were not clinically different. (Chart B)
- ☐ Stool consistency was predominantly Soft/Formed in both groups.
- ☐ Diarrhea and constipation were not prevalent on either formula.
- ☐ Acceptance and tolerance of Mead Johnson Alactamil is not different from acceptance and tolerance of soy formula.



[1]Data on file. October 1990 *T.M. Authorized User © 1992 Mead Johnson Canada

Conclusion: Mead Johnson Alactamil is an acceptable alternative to soy-based formula in the treatment of common feeding problems.

When counseling parents of infants with common feeding problems, Canadian pediatricians are now suggesting Mead Johnson Alactamil as an alternative to soy. It allows the baby to stay with milk protein while replacing lactose with glucose polymers as the carbohydrate source.

Lactose-free Mead Johnson Alactamil, the step before soy.

MeadJohnson Alactamil

Common Back Pain - A Mechanical Problem

Jan Prsala,* PhD, Robert E. Stalker,** MD, and Richard J. Hoyle†

Halifax, N.S.

Back pain is a difficult condition to diagnose and treat. In most cases it is caused by augmented mechanical stresses on the spinal tissues, particularly in the lumbo-sacral region (L5-S1), in persons with an incorrect body posture.

Analysis of mechanical stresses on a spine deformed in an antero-posterior plane strongly suggests that these stresses can lead over a period of time to micro and even to macroscopic changes in the spinal tissues and to development of pain.

It follows, then, that only an adequate reduction of the mechanical stresses on the involved spinal tissues can provide long term relief of back pain. This can be accomplished by improving the body posture through the use of specific corrective exercises.

Back pain is a very common presenting complaint to health care professionals. About 80 % of the entire population complain of back pain at some time during their lifetime.[1] As a common cause of absence from work, back pain is responsible for tremendous costs to the economy.[2,3,4] The assessment and treatment of back pain have become a new growth area with numerous back treatment centers being opened througthout the country.

Dr. Al Nachemson has devoted considerable time to the study of back pain and contends that the etiological factors that cause most patients' back pain are unknown.[5] He states that "the treatment of low back pain is predominantly symptomatic, aimed at reducing the level of pain and suffering and improving mobility. Despite a wide variety of treatment approaches implemented, their efficacy seems to be questionable and controlled research is sparse. In general medication is no solution to chronic back pain, though it may be useful in acute pain problems".[6]

Typical treatment for back conditions in this country consists of analgesics, non-steroidal anti-inflammatories and physiotherapy. According to Fast there is no scientific study which shows that conventional physical modalities (e.g. ultrasound, short-wave diathermy, superficial moist heat, ice, massage and hydrotherapy) modify the natural history of low back disorders.[7] He also feels that there is no proof that manipulative therapy has a long term effect.

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Many patients wonder why, with the many advances in medicine over the last decades, back pain continues to be such an enigma. Back pain appears to be much more common among people involved in manual labour, particularly those who must stand for long hours and among athletes whose sports place excessive stress on the back such as gymnastics or hockey. Postural factors seem to play a major role in this problem and perhaps the focus of treatment should change from concentrating on symptomatic measures to addressing mechanical factors with the aim of effecting long term benefit.

Physicians and other health care professionals should become familiar with the mechanics of the spine and how postural changes can create an environment leading to back pathology and pain.

NORMAL POSTURE

Normal posture implies a spine that is pain free and comfortably able to withstand the stresses of day to day activities. There are a number of features included in the description of correct posture. The head should be in a well balanced straight position with the ears centered over the shoulders. The shoulders should be pulled back with the scapulae adducted. The gleno-humeral joints should be centered over the hip joints. The abdominal muscles should be tucked. The lumbo-sacral angle should be small. The center of gravity should fall over the first and second metatarsal heads. The feet should function in a neutral position, neither excessively pronated or supinated.

Clinical experience suggests several other features that could be included in this description. The ratio and length of the kyphotic and lordotic curves should be considered. Ideally the kyphotic curve should be long and mildly curved and the lordotic curve should be short and mildly curved (Fig. 1).

ABNORMAL POSTURE

It is felt that abnormal posture, by definitition, results in excessive stresses being placed on the spine, which when present over a period of time, lead to pathology of the associated soft and osseous tissues. Rash *et al* include the following features in their description of incorrect posture: head inclined forward, shoulders held forward (slouching, scapulae abducted), abdomen protruding, and a large lumbo-sacral angle.[8] Other features might also be included, namely a long accentuated lordotic curve and a short kyphotic curve which must become accentuated as well in order to maintain an upright posture (Fig. 1).

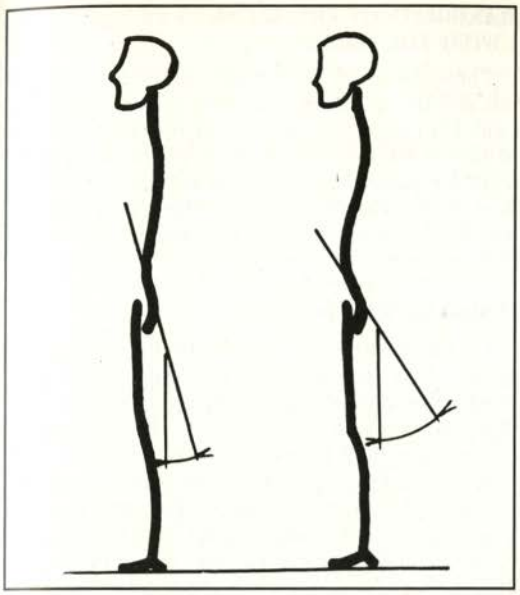

Figure 1

Change of the spinal curves and of the sacral angle as a result of a too powerful force couple created by the upperbody weight and ground reaction force.

ANALYSIS OF MECHANICAL STRESSES ON THE SPINE

Everyone is familiar with the result of sudden severe force on a joint, torn ligaments and internal derangement being possible consequences. The associated swelling and disability are well known. Damaging stresses need not always be so dramatic and can occur insidiously and silently until the involved tissue can no longer function normally. Many patients with mechanically induced problems do not recall a specific injury and it probably is appropriate to explain the failing of the structures as a result of accumulated stress.

The effect of gravity on a normally aligned spine can contribute to the degenerative changes associated with aging. If the spine is abnormally aligned degenerative changes can become obvious at a much younger age than anticipated. These changes frequently are seen at the lumbo-sacral junction and sacro-iliac joints. The weight of the torso when standing can have a significant gravitational effect on this area.

FORMATION OF THE SPINAL CURVES

The spine does not divide the body into two symmetrical halves. It supports the head behind the center of the skull and is located completely behind the rib cage and the abdominal cavity. The resultant forces on the spine result in the kyphotic curvature of the upper back. The remainder of the spine compensates by developing a lordotic neck and low back curvature, but this does not totally reduce the asymmetry. Balance of the body is maintained through the interplay of numerous forces. This inherent asymmetry creates mechanical stresses on the whole spine particularly at the L5-S1 level, witnessed by the moment arm labeled x1 in Fig. 2. The chest forms

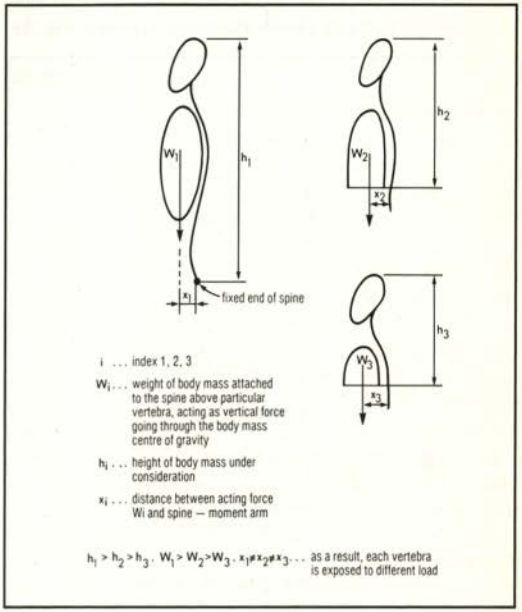

Figure 2

Loading of vertebrae with upperbody weight and its moment.

an anterior load upon which gravity is constantly exerting a rotatory force (torque).[9]

The spinal curves cannot be created by the rectangular vertebrae unless each vertebra assumes an angular relationship to its adjacent vertebrae. This angular alignment creates a situation where there is much more stress on the restraining structures as compared to vertebrae piled perfectly vertically on top of one another. The last lumbar disk, facet joints and associated soft tissues are under enormous stresses since they have to bear the weight of the whole trunk, including the arms and head.

EFFECT OF VERTICAL FORCES ON THE VERTEBRAE

The gravitational force of the weight of the trunk has a profound effect on the asymmetrically compressed disk and on the compressed facet joints. "The intervertebral disk, which has many functions is subjected to a considerable variety of forces and moments. Along with the facet joints, it is responsible for carrying all the compressive loading to which the trunk is subjected".[10] The lower lumbar disks have to support total loads of as much as 100 to 175 kilograms when the subject is seated. In the standing position total loads of between 90 and 120 kilograms have been calculated.[11]

The axial force would be equally distributed over the whole surface area of the disk and the facets if there was

a parallel relationship between the adjoining vertebrae. Because the vertebra above the disk is compressed asymmetrically (Fig. 3), the posterior aspects of the disk and facet joints are under much greater compression stress than the anterior portion. The vertebral unit is stressed by this axial force and also by a turning moment (Fig. 4).

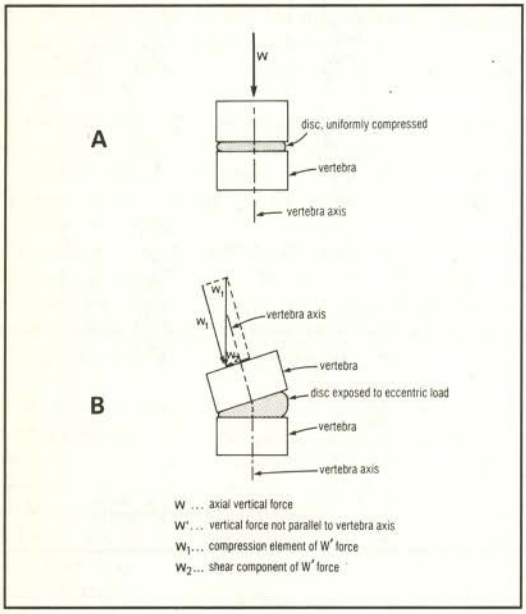

Figure 3

Symmetric and asymmetric compression of vertebra

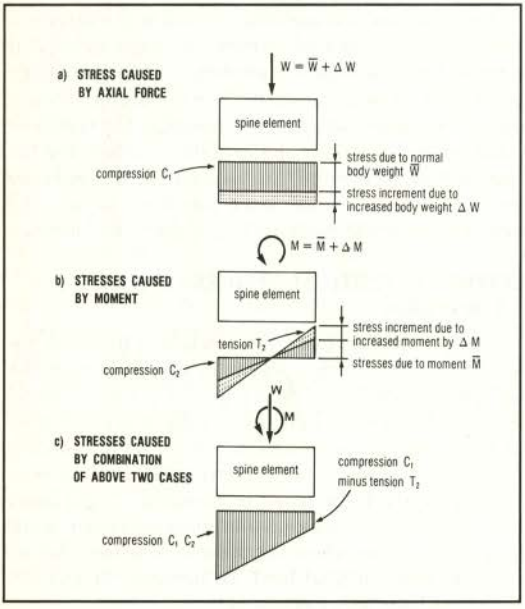

Figure 4

Spine element under different stresses (by axial force and by moment)

FLEXIBILITY OF THE TRUNK AS COMPARED WITH THE PELVIS

The spine is very flexible in both the antero-posterior and lateral planes. The pelvic girdle is relatively rigid. The transition between trunk and pelvis is an area of great stress and this is borne by the lower vertebral segments particularly the L5-S1 disk and associated facet joints. The frictional and shear forces at this junction are of sufficient intensity to cause damage to these structures.

LUMBO-SACRAL ANGLE

The lumbo-sacral angle is formed by the top proximal surface of the sacrum and the horizontal. This narrow forwardly inclined platform sets up a situation where the torso has a tendency to slide forward. This increases the frictional and shear forces on this area. The intensity of these forces is very dependent on the angle – the greater the angle, the greater the forces. Those people with a long lordotic curve, sharper spinal curves and a large lumbo-sacral angle are at a greater risk of developing problems as a result of chronic abnormal stress on the area as compared to people with normal lumbar and sacral posture (Fig. 5). Perhaps these abnormal shear and frictional forces lead to spondylolysis and spondylolisthesis typically seen in the lower lumbar area.

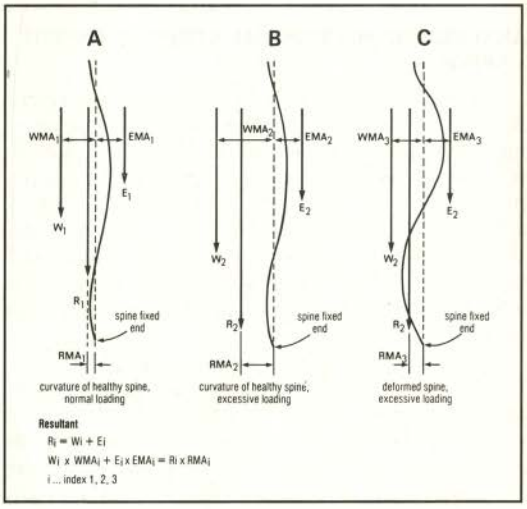

Figure 5

Kyphotic and lordotic changes of spine

LUMBAR FACET JOINTS

The function and spatial orientation of the last pair of lumbar facet joints can contribute to increased stress on these joints, particularly in people with poor posture. The facet joints of L1-L3 have an antero-posterior orientation, permitting relatively good ranges through flexion and extension because of their gliding action. The facet joints of the third, fourth and particularly the fifth lum-

bar vertebrae are situated at a gradually increasing angle from this plane. This limits their flexibility since the gliding action of the facets during flexion and extension of the trunk is technically impossible. This is replaced by a rocking action of the facets.

Bony interaction is created at both limits of their range of motion. It would appear that this bony interaction has a protective purpose. It limits any excessive and unhealthy flexibility through the last three facet joints. The transition from the flexible spine and inflexible pelvis occurs more gradually and prevents all the stress from being focused on the L5-S1 level. If there was excessive flexibility at this level, joint dislocations would be a likely consequence.

The more lateral orientation of the lower facet joints gives a protective function, although this may have negative consequences in a person with abnormal back posture. A person with a long lordotic curvature through the lower thoracic and lumbar area, tends to lean backward more than a person with a short lordotic curve. The lower facet joints are more extended exposing them to constant wear and tear due to bony interaction between the facets. These stresses are accentuated by excessive standing or sitting incorrectly. If the stresses on the facet joints are excessive the normal healing process is unable to cope with them.

DEVELOPMENT OF ABNORMAL POSTURE

The body is exposed to considerable stress on a daily basis with normal day-to-day activities. The spine is often placed in a very asymmetrical position with bent over posture at work, in athletics and during other leisure activities. Such inclined positions are very demanding on the spinal structures. The erector spinae muscles are responsible for maintaining these postures, and although they can become quite strong, they often become quite shortened in the process. Their degree of contraction is very high because they have to support the heavy, forwardly-inclined torso. The muscles acting upon the spinous processes and the ribs have a very poor mechanical advantage. The moment arm of the applied force is then much shorter than that of the resistance.

The erector spinae muscles shorten when the trunk is in a flexed position. A study by Hart *et al* showed that the muscular activity was inadequate while lifting in a bent over posture with the lumbar spine flexed, but was very high when the lumbar spine was held straight or arched[12]. Their research also showed that the resistance moment arm was longer when the lumbar spine was flexed than when it was straight or arched. Therefore only a straight or arched spine can sustain bent over postures requiring the lumbar portion of the erector spinae to be shortened. The initial phase of weight lifting is a good example of such asymmetric loading of the spine.

It is believed that the inevitable strengthening and shortening of the erector spinae can lead to further exaggeration of the lordotic curve. This curve is normally formed by the five lumbar vertebrae, but in abnormal situations can involve lower thoracic vertebrae as well in a manner like a bow bending when its string is shortened. Since the erector spinae originate in a common tendon on the sacrum, their shortening can also increase the angle formed by the sacrum and the vertical. This results in increased stress on the posterior structures of the vertebral unit and can ultimately lead to pain.

REHABILITATION

The deformation of the spinal curves tends to worsen with time if the person does not do appropriate stretching and strengthening. The associated microscopic trauma escalates to the point of macroscopic damage and pain. The episodes of pain become more common to the point of being chronic. Unless the excessive stresses on the lumbar vertebrae units are reduced through rehabilitation in order to restore normal posture, the pain will remain an untreatable condition.

Excessive mechanical stresses on the lumbar spine are probably responsible for many of the situations when a person presents with back pain. If specific corrective measures are not taken to restore and maintain proper posture, then it is not surprising that many of the conventional treatments have no lasting benefit. The authors feel that a mechanically oriented solution should be considered and would likely succeed in providing long term relief of back pain. Such postural exercises could also serve as a feasible preventative measure in those people engaged in activities commonly asssociated with back pain, such as manual labourers and many athletes such as gymnasts and hockey players. □

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Old age is the most unexpected of all the things that happens to a man.

Leon Trotsky (1879-1940)

Near-Hangings

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Near-hangings refer to individuals who survive the initial trauma of an accidental or attempted suicidal hanging.

The clinical manifestations and degree of recovery from a near-hanging varies with the amount of time in which the brain is without oxygenated blood. Total recovery can be expected in some cases but as the length of time of arterial occlusion increases the degree of recovery decreases.

Sequelae of near-hangings include varying degrees of neurologic damage, adult respiratory distress syndrome, airway obstruction, superficial skin manifestations, and cardiovascular and neuropsychiatric problems.

In Canada, hanging is one of the most common forms of suicide, ranking third to firearms and poisoning. In 1986 alone there were 840 suicidal hanging deaths (707 male; 133 female). Of these, 44 occurred in the Atlantic Provinces *(Canada Yearbook 1988)*. Unlike most judicial hangings, not all attempted suicidal hangings result in death and these are referred to as near-hangings.[9] The reason for this difference between judicial and suicidal hangings lies in the methods used in the two types of hanging. The clinical presentations of near-hangings are also variable as are the degrees of recovery in these patients.

In a judicial hanging the condemned person stands on a trapdoor and, when the trap is released, he falls several feet until he is stopped by the rope.[7] In 1913, Frederic Wood-Jones described the "ideal lesion produced by judicial hanging" which resulted in "traumatic spondylolisthesis of the axis with bilateral fracture through the neural arch and dislocation of vertebra C2 on C3".[12] Suicidal hangings, on the other hand, are rarely associated with this degree of cervical injury since such extreme drops are not employed here.[9] In a suicidal hanging a drop of six feet six inches to seven feet six inches (depending on body weight) would be necessary for cervical fracture or dislocation.[9] When death does result from a suicidal hanging, it is usually the result of interruption of cerebral blood flow resulting in cerebral ischemia.[9]

Suicidal hangings have been described as *typical* or *atypical*, and as *complete* or *incomplete*.[11] In a typical hanging, the ligative mark is located in front of the neck between the hyoid bone and the thyroid cartilage and the groove will converge upwards towards a point in the midline at the back of the neck. In an atypical hanging, the groove is located at the side of the neck or in front. With a complete hanging the body is freely suspended, as opposed to an incomplete hanging where part of the body is supported by the ground.[11]

As mentioned, the clinical manifestations found in near-hanging patients are variable. One factor used to explain this is the length of time the person was suspended, since the act of hanging cuts off flow through the carotid arteries and therefore results in cerebral hypoxia/anoxia.[2,9] If flow is restored soon after the person loses consciousness, then full neurologic recovery should follow, but as the length of time of arterial occlusion increases the degree of recovery decreases.[9] Boyarsky *et al.* describe variable neurologic manifestations depending on the length of time of the hanging event and the degree of hypoxia/anoxia.[2] Mild hypoxia is associated with "early inattention and muscle inco-ordination with complete resolution". Anoxia of less than five minutes will result in coma but recovery is usually complete (though it takes longer for recovery than it does with mild hypoxia). If the hypoxia is greater than five minutes severe injury and encephalopathy will result.[2]

Irreversible cell injury from anoxia is, in general, most likely due to decreased ATP and cell membrane damage.[6] With decreased oxygen there is also decreased production of ATP and subsequent decreased function of the sodium/potassium pump. This leads to increased intracellular sodium and osmotic cellular swelling. The cellular swelling is also caused by an increased cellular osmotic load of lactic acid, inorganic phosphate, and purine nucleotides.[6] Cerebral lactic acidosis results not only in cerebral edema, but also vasomotor paralysis. These two factors add to postischemic compromise of the microvascular circulation.[8]

Cell membrane damage is due to progressive loss of phospholipids (due to activation of phospholipases by increased intracellular calcium) and by an increase in lipid breakdown products which accumulate in ischemic cells and have a detergent effect on the cell membrane.[6]

After flow is restored, additional cellular damage and edema can result from the production of oxygen radicals which are in part produced by PMN at the site of ischemia.[6,9]

It has also been suggested that the altered calcium metabolism which occurs with ischemia can cause vasoconstriction and therefore with reperfusion there is a perfusion/demand mismatch, with some areas of ischemia persisting, leading to further edema.[9]

Also associated with near-hangings is the possible development of Adult Respiratory Distress Syndrome

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(ARDS).[4] ARDS is a syndrome of severe pulmonary insufficiency and occurs as an aftermath of several conditions, one of which is neurologic injury.[3] The course of events seen with ARDS is: an initial insult; a period when lung function appears normal; followed by rapid onset of impaired pulmonary function.[3] The lung damage seen with ARDS, regardless of etiology, is mainly confined to the alveolar endothelium and epithelium. This damage leads to increased permeability to proteins, and interstitial and alveolar edema resulting in impaired gas transport.[7]

The physiologic manifestations of ARDS are a ventilation/perfusion mismatch, right-to-left intrapulmonary shunting, decreased functional residual capacity (FRC) and decreased total lung capacity (TLC).[7]

Several hypotheses have been proposed for the development of ARDS in near-hangings. McHugh described the development of ARDS as being secondary to the "centroneurogenic lesion".[9] Moser described a "centroneurogenic respiratory distress" involving alveolar-capillary leakage secondary to brain injury.[10] McHugh also discussed the possible role of altered catecholamines (due to cerebral hypoxia) in the constriction of pulmonary post-capillary sphincters.[9] Sternback *et al.* proposed that the development of ARDS due to a hypoxic cerebral injury may be the result of histamine release.[13]

The pathology associated with ARDS is pulmonary congestion, edema, hemorrhage, surfactant inactivation and atelectasis.[9] Sternbach described a near-hanging involving a 14 year old male who subsequently developed ARDS.[13] He presented with respiratory distress, pink frothy sputum in the airways which became increasingly difficult to suction, and on chest x-ray showed diffuse interstitial edema.

In the management of ARDS, intermittent positive pressure ventilation has shown some success in increasing alveolar oxygenation.[1] Positive end-expiratory pressure (PEEP) at pressures which exceed the critical closing pressure have been shown to minimize atelectasis, increase FRC, decrease intrapulmonary shunting, and allow alveoli to remain open through all phases of the respiratory cycle.[1] It should be noted that "low levels of PEEP (5 to 10 cm H_20) are usually well tolerated, but as PEEP is increased, there will be a significant reduction in cardiac output due to decreased venous return which is significant enough to be harmful to tissues".[13] The administration of oxygen is effective in increasing arterial oxygen content in the immediate care of ARDS but as intrapulmonary shunting occurs it is of little value. There is also the additional risk of oxygen toxicity which can cause lung microvascular and cellular injury due to the generation of superoxide radicals.[7] Even with treatment, ARDS is associated with a fifty percent mortality rate.[1]

Another clinical manifestation of near-hangings is airway obstruction due to edema and hemorrhage in the paratracheal and laryngeal areas. This obstruction can occur anywhere up to 24 hours after the initial insult and therefore the patient must be carefully monitored for this.[8]

Left ventricular failure has been associated with cerebral hypoxia.[8] It is hypothesized that cerebral hypoxia causes peripheral vasoconstriction which in turn leads to increased left ventricular afterload and ultimately to left ventricular failure and pulmonary edema.[8] Cardiac arrest has also been associated with near-hangings and can be due to altered parasympathetic or sympathetic tone.[9] Increased parasympathetic tone is due to pressure on the carotid bodies or vagal sheath by the ligature, and increased sympathetic tone is due to pressure on the pericarotid areas.[9]

Superficial manifestations of a near-hanging include a groove on the neck outlining the compression caused by the ligature. Petichial hemorrhages are seen on the subconjunctival areas and on the skin.[4]

Neuropsychiatric problems are often a complication of a near-hanging. These may be manifested initially as violent behaviour and restlessness, followed by the development of psychosis, amnesia, dementia and/or Korsakoff's Syndrome.[2,9] These patients require additional psychiatric help for both their psychiatric problems caused by the hanging and for the reasons leading up to the attempted suicide.[2]

Near-hanging victims represent a diverse group of patients presenting clinically with varying signs and symptoms which can lead to a complete recovery or to long term neurologic, respiratory, cardiovascular, and neuropsychiatric problems. □

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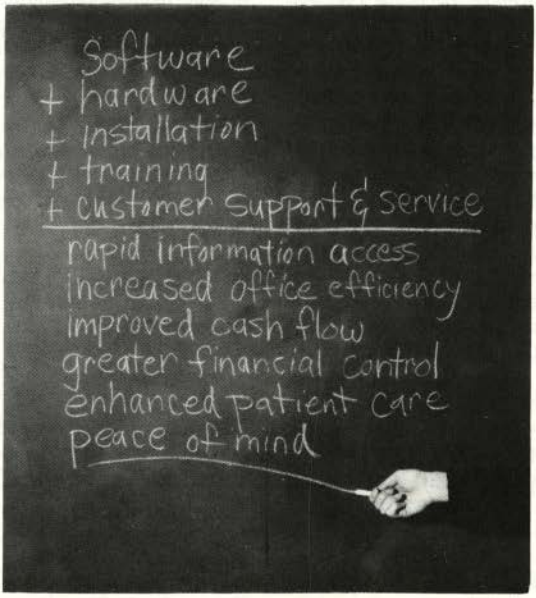
The whole is greater than the sum of its parts.



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Percutaneous Endoscopic Gastrostomy

ARE STERILE FIELD AND VISCERAL/PARIETAL PERITONEAL APPOSITION NECESSARY?

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Gastrostomy tube placement has traditionally envolved preparation of a sterile field on the abdomen and cutaneous fixation of the Gastrostomy tube (G-tube) to ensure maintenance of cather position and visceral/parietal peritoneal apposition. This paper challenges the necessity of sterile field preparation and visceral/parietal peritoneal apposition via G-tube traction.

27 Percutaneous Endoscopic Gastrostomies (PEG) were performed without sterile field preparation on the abdominal wall or G-tube traction to appose the visceral and parietal peritoneal surfaces. The average operative time was 14 minutes. Follow-up was terminated with G-tube removal or death. No peritoneal leaks, peristomal infections or inward or transmural migration of G-tubes were encountered. There was no morbidity associated with catheter placement. The use of cutaneous button fixation is not necessary and promotes gastric mucosal pressure induced ischemia and hemorrage, transmural migration of the catheter and peristomal skin excoriation from entrapped secretions. Sterile field preparation and visceral/parietal peritoneal apposition are not necessary and the latter may be contraindicated during this quick, safe and portable procedure.

Egeberg first attempted the construction of a gastrostomy in 1837 but it was not until 1876 that the first successful gastrostomy was performed by Verneuil. Gastrostomies are frequently performed today but require transport of the patient to the operating room with a general anesthetic and the performance of a minilaparotomy. In 1980, Gaundeier and Ponsly first introduced the method of establishing a gastrostomy percutaneously using an endoscopic technique.[1] Since then, the relative ease of Percutaneous Endoscopic Gastrostomy (PEG) and its lower morbidity has been well characterized in the literature.[2-4] This paper addresses the issue of the necessity of a formal sterile field and visceral/parietal peritoneal apposition via G-tube traction. The portability of the technique of PEG is also introduced.

MATERIALS AND METHODS

All patients referred to the Camp Hill Hospital Surgical Service for gastrostomy for the years 1987-1991 were considered for the procedure. Inclusion criteria of a patent esophagus, the absence of gastric outlet obstruction or a gastric motility disorder were met by all patients. After 4% xylocaine viscus gargle and I.V. Diazepam sedation, gastroscopy was performed. Using transillumination and palpation, a site just to the left of midline and at least 2 cm inferior to the costal margin was selected. The antrum and gastric pacemaker were avoided. 2% xylocaine was infiltrated into the skin and fascia over the selected point.

A 14 French gastrostomy tube was then placed, using the "push-through" or Sacks-Vine technique. Following the placement of the catheter, gastroscopy was then repeated to confirm that the tip of the catheter was in the stomach and against gastric mucosa. No sterile field was prepared on the abdomen, no prophylactic antibiotics were administered, and no traction was placed on the gastrostomy tube. A skin disc or "button" was not used and no suture material was used.

The time from start to finish of each procedure was accurately recorded. Tube feeding started on the following day. Patients were followed until the catheter was discontinued or to death. Follow-up of the patients still alive and still using their gastrostomy tubes was accomplished by telephone calls to the primary health care provider. The end of the follow-up period was December 1, 1990.

RESULTS

Twenty-seven gastrostomies were performed on twenty-two patients. The male to female ratio was 1.9:1. The average age was 71 years with a range of 53 to 83 years. The indications for the procedure were primarily neurological. Thirteen patients had cerebrovascular accident (CVA), three had amyotrophic lateral sclerosis, two were psychiatric patients and refused to eat, two were ICU patients with sepsis but functioning GI tracts and one had organic brain syndrome and would not eat. Follow-up ranged from 1 to 74 weeks with an average of 34 weeks.

The operative time ranged from 8 to 50 minutes with an average of 17 minutes. The 50 minute case involved retrieving a misplaced catheter. If this case is disregarded, the average operative time was 14 minutes.

There were no deaths related to placement or use of the tube. Two patients developed stomal related complications. One had some peristomal erythrema without drainage four weeks after G-tube insertion, which responded to antibiotics. One developed some hypertrophic

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granulation tissue two years after G-tube placement, which resolved with silver nitrate cauterization. There were no peristomal infections perioperatively, no difficulties with post-operative ileus and no cases of peritonitis. No problems were encountered with tube migration into the distal GI tract or externally through the gastric wall. Several tubes were pulled out by confused or agitated patients but no bleeding or infection resulted from this. There were no problems of pneumoperitoneum, stomal leaks, upper GI bleeding, gastroenteric fistula or injury to neighboring viscera.

There were no placement failures. Three patients had to have their tubes replaced, two were pulled out and one was accidently cut while a dressing was being changed. One patient had to have her tube replaced twice. Her tube was pulled out once. Then a foley catheter was placed through the tract and the inflated bulb eroded through the gastric wall and had to be removed with a new tube inserted at a different site.

There were no episodes of obvious tube-feed regurgitation reported. Eleven patients had pre and post-procedure chest radiographs and only one developed pneumonia post-operatively that had not had pneumonia pre-operatively. This patient had a tube placed after having suffered a CVA with resultant dysphagia. Four months later he was recovering and ambulating but fell and broke his hip. He died a few weeks later of post-operative pneumonia after having his hip pinned.

Six patients had pre- and post-operative weights recorded. All gained weight after having a gastrostomy feeding tube placed. The average weight gain was 13 kg with a range of 4 to 37 kg. If we disregard the 37 kg weight gain, the average gain was 8.6 kg or 0.7 kg/wk of tube usage.

At the time of follow-up, seven patients were still alive and six still had functioning tubes. One tube is still in place after 3 1/2 years. One patient who suffered a CVA stopped using her tube after fourteen months as she had regained her ability to swallow.

DISCUSSION

With twenty years of experience of PEG, the technique is still gaining popularity and is being more widely used in the surgical community. Studies comparing surgical gastrostomies with both endoscopic and non-endoscopic percutaneous gastrostomies have been done.[5,6] PEG has been shown to have less procedure related morbidity then the standard surgical gastrostomy. PEG has also been shown to be more cost effective.

Our study confirms the very low morbidity and mortality associated with PEG. This is a relatively simple technique that is quick, with an average O.R. time of fourteen minutes for first-time gastrostomies. We performed two PEGs in the ICU, illustrating the mobility of the equipment (certainly easier to transport then ventilated ICU patients.)

There has been concern expressed in the past about the risk of regurgitation and aspiration in patients fed by gastrostomy tubes. However, the study by Johnson *et al* showed increased lower esophageal sphincter tome and lower reflux scores (as determined with 24 hour esophageal pH monitoring) in patients fed by gastrostomy compared to patients fed by jejunostomy tubes.[8] This suggests that using the stomach promotes improved gastroesophageal sphincter competence. Furthermore, Cogen, *et al* found no correlation between age, mental status or method of gastric tube feeding with the development of aspiration pneumonia. They did find a correlation with the presence of pneumonia prior to initiation of gastric feeds.[9] This suggests that this population of patients are chronic aspirators and that the association with the gastric feeding may have been coincidental rather than etiological. Our study supports these data in that no patient developed pneumonia after placement of a G-tube who did not have pneumonia prior to G-tube placement. One patient did develop pneumonia after having a hip pinned.

No attempt was made to create a sterile field at the percutaneous site and we encountered no difficulties with wound, stomal or peritoneal infections. Previous authors have advocated systemic antibiotics as well as antimicrobial gargle prior to the procedure. It appears that there is no morbidity associated with passing a catheter through the oral pharynx, esophagus, stomach and abdominal wall without the use of antimicrobial materials.

Historically, the concern of intraperitoneal leakage of gastric contents around the G-tube encouraged surgeons to ensure that the gastric and abdominal walls were approximated. This is accomplished with interrupted sutures in a surgical gastrostomy and by G-tube traction and the placement of a cutaneous button inpercutaneous gastrostomies.

Several aspects of this should be considered. With a surgical gastrostomy, the gastrostomy is initially larger than the G-tube, and a pursestring is used to try and form a watertight seal. This is not a problem with the percutaneous placement of a G-tube since the gastrostomy is dilated up only to the size of the G-tube and no larger. This made-to-fit gastrostomy is not associated with leakage. Internal migration of the catheter is not a problem with the T-tip design of the newer G-tubes as was previously described with bulb-tipped catheters. Therefore, anchoring the tube externally is not necessary. The use of cutaneous buttons to both anchor the catheter and apply traction to ensure visceral/parietal peritoneal apposition probably caused more morbidity than they prevented. Traction on the tube promotes pressure ischemia on the gastric mucosa with bleeding and transmural migration of the catheter tip. We encountered one such problem in one patient who had a balloon tipped foley catheter in place.

Cutaneous buttons prevent good skin care around the gastrostomy site, thereby allowing secretion accumulation with the associated skin excoriation. We have demonstrated that gastric leak induced peritonitis and peristomal complications as well as gastric bleeding and

transmural migration of the catheter tip can be prevented by the omission of the G-tube traction and the cutaneous button.

CONCLUSION

PEG is a quick, safe, mobile procedure for the establishment of a feeding gastrostomy. Visceral/parietal peritoneal apposition is not necessary with the percutaneous technique and in fact is probably contraindicated. Creation of a sterile field on the abdomen is not necessary for this procedure. □

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Preoperative Risk Assessment in Patients Undergoing Urological or Orthopaedic Surgery

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Objective: **To assess the applicability of the Goldman Cardiac Risk Index for patients undergoing urologic and orthopaedic surgical procedures and to determine if other factors influence perioperative morbidity.**

Design: **Prospective study of all patients undergoing anesthesia using a set protocol.**

Setting: **Teaching hospital.**

Patients: **All patients over the age of 40 undergoing general or spinal anesthesia on Urology from September 27 to November 18, 1986 and Orthopaedics from April 17 to July 10, 1988.**

Intervention: **General or spinal anesthesia plus appropriate surgical procedure.**

Measurement and main results: **Of 386 patients who underwent 443 procedures, only 8 serious postoperative complications were noted. Post-operative silent ischemia was detected in 5.5% of patients who had a post-operative ECG. Symptomatic ischemia occurred in 5 patients and one of these patients had elevation of cardiac enzymes compatible with a myocardial infarction. Diastolic blood pressure > 90 mm Hg emerged as a risk factor in disagreement with Goldman's results.**

Conclusions: **The Goldman Cardiac Risk Index correctly predicted the operative risk in Orthopaedic patients, but underestimated the risk in supposedly low-risk Urology patients. Routine postoperative ECGs appear to be indicated in patients with one or more risk factors for ischemic cardiac disease.**

Internists are frequently asked to ascertain a patient's risk for developing postoperative cardiac complications. In general, they are able to comment only on whether or not patients would fall into a high or low risk category. Patients who are in a high risk category are those who have known ischemic heart disease, a history of poor lung function or other major illness. Disconcerting to both patient and physician alike, however, is the individual who is not perceived to be at high risk preoperatively but who develops cardiac complications postoperatively.

Various classification schemes have been adopted by different physician groups, in an attempt to assign risk factors to a given patient. For example, anesthesiologists frequently use the American Society of Anesthesiologists Physical Status Measure (ASA) (Table I).[1] A risk index proposed by Goldman[2] (Table IIa, IIb) has been studied prospectively by other investigators.[3-7] In general, clinicians have found the Goldman Index to be sensitive to high risk patients,[3,4] but Jeffrey *et al* questioned the possible underestimation of serious cardiovascular complications in Goldman's low risk groups.[5]

All the above studies have contributed to our understanding of perioperative risk. However, each was carried out in a somewhat different patient population, and had different study criteria. For example, Goldman's original study evaluated all patients over the age of 40 going to surgery on the General Surgery, Orthopaedic Surgery or Urological service, excluding patients undergoing transurethral resection of the prostate.[2] Detsky *et al* performed preoperative consultations on patients undergoing noncardiac surgery.[3] This population was more selected as the surgeon had already identified the

TABLE I[1]

AMERICAN SOCIETY OF ANESTHESIOLOGISTS PHYSICAL STATUS MEASURE (ASA)

Class 1 There is no physiologic, biochemical, or psychiatric disturbance. The pathological process for which the operation is to be performed is localized and not conducive to systemic disturbance. Examples: a fit patient with inguinal hernia; fibroid uterus in an otherwise healthy woman.

Class 2 Mild to moderate systemic disturbance caused either by the condition to be treated surgically or by other pathophysiological processes. Examples: presence of mild diabetes; essential hypertension; anemia.

Class 3 Rather severe systemic disturbance or pathology from whatever cause, even though it may not be possible to define the degree of disability with finality. Examples: severe diabetes with vascular complications; moderate to severe degree of pulmonary insufficiency; angina pectoris or healed myocardial infarction.

Class 4 Indicative of the patient with a severe systemic disorder already life-threatening and not always correctable by the operative procedure. Examples: advanced degrees of cardiac, pulmonary, hepatic, renal, or endocrine insufficiency.

Class 5 This category embraces the moribund patient who has little chance of survival but is submitted to operation in desperation. Examples: the burst aneurysm with the patient in profound shock; major cerebral trauma with rapidly increasing intracranial pressure; massive pulmonary embolus.

Emergency Operation (E) Any patient in one of the classes listed above who is operated upon as an emergency is considered to be in somewhat poorer physical condition. The letter E is placed beside the numerical classification.

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TABLE IIA

GOLDMAN CARDIAC INDEX[2]

Criteria	Points
History	
Age > 70 yr	5
MI in previous 6 mo	10
Physical examination	
S3 gallop or JVD	11
Important VAS	3
Electrocardiogram	
Rhythm other than sinus or PACs on last preoperative ECG	7
5 PVC's/min documentation at any time before operation	7
General Status	
PO2 < 60 or PCO2 > 50 mm Hg K < 3.0 or HCO3 < 20 meq/litre BUN > 50 or Cr > 3.0 mg/dl Abnormal SGOT, signs of chronic liver disease, or patient bed-ridden from noncardiac causes	3
Operation	
Intraperitoneal, intrathoracic or aortic operation	3
Emergency operation	4
Total possible	**53 points**

*MI denotes myocardial infarction, JVD - jugular venous distension, VAS - valvular aortic stenosis, PAC's - premature atrial contractions, ECG - electrocardiogram, PVC's - premature ventricular contractions, PO2 - partial pressure of oxygen, PCO2 - partial pressure of carbon dioxide, K - potassium, HCO3 - bicarbonate, BUN - blood urea nitrogen, Cr - creatinine and SGOT - serum glutamic oxalacetic transaminase.

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TABLE IIB

GOLDMAN'S CARDIAC RISK SCALE[2]

Class	Point total	None or only minor complications	Life-threatening complications	Cardiac death
		(%)	(%)	(%)
I	0 - 5	99	0.7	0.2
II	6 - 12	93	5	2
III	13 - 25	86	11	2
IV	> 26	22	22	56

need for a preoperative consultation.[3] As a surgery resident, Zeldin was able to study all patients under his care during his four year residency. Hence, his population consisted of General Surgery, Vascular and non-cardiac Thoracic Surgery patients who had not been preselected.[4] Jeffrey, on the other hand, evaluated only patients going for abdominal aneurysm repair.[5] The geriatric patient undergoing non-cardiac surgery was studied by Gerson *et al*, who confirmed the earlier study of Goldman, but also suggested that the inability to exercise was an important predictor of cardiac outcome in this age group.[6] Charlson *et al* have clearly outlined the varied usefulness of the several different indices when each index is applied to heterogenous populations.[7]

To determine the validity of the Goldman Index in a group of patients who were not well evaluated in the above investigations, a study of cardiac risk in unselected patients on the Urology and Orthopaedic Surgery services was performed at the Victoria General Hospital, an 800 bed, adult teaching hospital, in Halifax, Nova Scotia, Canada.

METHODS

The study was divided into two phases. From September 27 to November 18, 1986, all patients over the age of 40 years, admitted to the Urology Service for elective or emergency surgery, under general or spinal anesthesia, were admitted to the study. Urology patients were selected for pragmatic reasons. The Urology Department at the Victoria General Hospital consists of 55 beds and cooperation with the 5 attending surgeons was excellent. Although Goldman had included Urology patients in his original study group of 1001 patients, very few Urology patients were actually studied, as the majority of Urology procedures were considered safe.[2] Any complications that did arise in Urology patients, therefore, should be predicted on the basis of the Goldman Index.

At the end of the first phase of the study, it was decided to look at a different patient population pool. As the Urology group was heavily male dominated, we sought a surgical population in which the sexes would be better represented. Orthopaedic Surgery with 66 beds was selected, again because of the cooperation from the surgeons. In contrast with Urology, this subset of patients had been more fully represented in Goldman's original study. From April 17 to July 10, 1988, all Orthopaedic patients over the age of 40 years admitted for elective or emergency surgery, under general or spinal anesthesia, were entered into this study.

All the Urology patients were seen preoperatively by ES or LH who were in the third year of Internal Medicine residency training. In the second phase of the study, a physician assistant was hired. Any findings felt to be abnormal preoperatively were checked by one of the two resident co-authors.

All patients on these two services were approached preoperatively and informed about the nature of the study. All agreed to participate and signed an informed consent sheet, approved by the Research Ethics Committee at the Victoria General Hospital. Relevant history was recorded and physical examination pertaining to the cardiovascular and respiratory systems was performed. Laboratory data included a complete blood count, serum electrolytes, blood sugar, blood urea nitrogen, serum creatinine, electrocardiogram and blood gases. The patients were then assigned to one of the Goldman's four cardiac classes.[2] Postoperatively, all patients' charts were

reviewed daily until discharge. If the patient had no symptoms, an electrocardiogram was obtained on the day of discharge or on postoperative day five, whichever was sooner. If the patient had symptoms, appropriate investigations were initiated.

The data were analyzed using an exact goodness-of-fit test to determine the applicability of the Goldman Index to Urology and Orthopaedic populations, individually and combined. Then all variables including type of anesthetic were subjected to multivariate discriminant analysis, based on the step-wise addition of the variable that contributed the largest increase in the Rao V value as computed by the standard program of the Statistical Package for the Social Sciences. ASA classification (Table I) could not be included in the analysis as it was not recorded by the anesthetists for all patients.

RESULTS

A total of 386 patients were admitted to the study, including 202 Orthopaedic patients and 184 Urology patients (Table III). Operative procedures performed included cytoscopy/biopsy, transurethral and open prostatectomy, nephrectomy, orchiectomy, stone extractions, joint replacements, fracture repair, spinal fusions and decompressions. Forty-one Urology patients (34M, 7F) did not have a postoperative EKG obtained. All Orthopaedic patients had a postoperative EKG.

Postoperative complications recorded were those defined by Goldman.[2] These included life-threatening cardiac complications, including pulmonary edema, myocardial infarction and/or witnessed ventricular tachycardia. There were no cardiac deaths in the study group.

TABLE III

DEMOGRAPHICS OF PATIENTS STUDIED

	Urology	Orthopedics
	(n = 184)	(n = 202)
M:F ratio	144:40	75:127
Age (yrs) + S.D.	68.5 +/- 9.6	66.3 +/- 2.9
range	44 - 93	40 - 91
Type of anesthesia (a)		
general	159	157
spinal	25	45
Procedures performed (b)		
Cystoscopy	102	
Stone extraction	52	
Prostatectomy	41	
Gold seeds	10	
Nephrectomy	8	
Cystectomy	7	
Misc. urologic	19	
Joint replacement		121
Spinal effusion		13
HTO		8
Excision of tumor		6
Hoffman/Keller		5
Laminectomy		5
Misc. orthopedic		46

(a) Patients requiring epidural or regional anesthesia were excluded
(b) 443 procedures on 386 patients

TABLE IV

PREDICTIVE VALUE OF GOLDMAN INDEX BY SURGICAL GROUP

Goldman Class	No. patients	Post-op complications (%)	% predicted by Goldman Index	Goodness of fit
ORTHOPEDICS				
I	162	2 (1.23)	0.7	
II	23	0 (0)	5	p = 0.489
III	17	1 (5.88)	11	
UROLOGY				
I	162	4 (2.47)	0.7	
II	17	1 (5.88)	5	p = 0.0559
III	4	0 (0)	11	

The two patient populations differed by age and type of anesthetic used (Table III) and did not have the same outcome characteristics. Table IV illustrates that complications were correctly predicted in the Orthopaedics population, but not in the Urology group. The original Urology population studied by Goldman did not include those patients undergoing so-called "low risk" procedures, such as cystoscopy and transurethral prostatic resection, but these patients were included in our study.

Table V illustrates the number of patients who fitted into each class of the Goldman Index. Urology and Orthopaedic patients were combined in this table as they were in Goldman's original study. Not surprisingly, the majority of patients (94.3%) were low risk (Class I or II), and the incidence of postoperative cardiac complications in the total population was low (4.3%), as predicted by the Goldman Index (5.7%). The number of patients who fulfilled the criteria for Class III and IV was small (5.7%). However, low risk patients (Class I as predicted by the Goldman Index) represented the majority of complications which occurred. When the two surgical patient populations are combined, Goldman's Index was not validated. An exact goodness-of-fit test indicated that the patients utilized in this study had a significantly different outcome than the patients utilized to derive the original Goldman Index (p=0.029). Class IV was excluded from the analysis as there was only one patient in this group.

EKG evidence of ischemia was noted in several asymptomatic patients (Table VI). Silent ischemia was defined as EKG evidence of ischemia or infarction in the absence of chest pain or anginal equivalence, and required one millimetre flat ST segment depression or one millimetre T wave inversion in two continuous leads. Using these criteria, 5.5% of all patients with normal preoperative EKGs who were included in the study were found to have silent ischemia. One patient had a silent myocardial infarction. The incidence may have been higher if all patients had had a postoperative EKG. Of the 5.5% of patients who exhibited silent ischemia, 10.5% had a prior history of angina. The remainder had no previous symp-

toms. Of those patients who experienced symptomatic ischemia postoperatively, 20% had a past history of angina.

TABLE V

PREDICTIVE VALUE OF GOLDMAN INDEX IN ALL PATIENTS STUDIED

Goldman Class	No. patients (%)	Post-op complications (%)	% predicted by Goldman Index	Goodness of fit*
I	324 (83.9)	6 (1.85)	0.7	
II	40 (10.4)	1 (2.50)	5	p = 0.0290
III	21 (5.4)	1 (4.76)	11	
IV	1 (0.3)	0 (0)	22	

* excludes Class IV

TABLE VI

FREQUENCY OF POST-OPERATIVE SILENT ISCHEMIA IN ALL PATIENTS WITH PRE- AND POST- OP ECGs *

	Frequency	Percent	Average age (yrs)
Yes	19	5.5	70.52
No	326	94.5	67.54

symptomatic ischemia n = 5

* excludes 41 Urology patients who did not have post-operative ECG

Multivariate analysis utilizing the methods outlined by Goldman were applied to all variables measured preoperatively.[2] Table VII outlines the preoperative factors related to postoperative myocardial ischemia, including both symptomatic and silent ischemia. Hypertension would appear to be the most important individual variable, as three out of the seven discriminant factors are associated with hypertension (diastolic blood pressure: > 90 mm Hg, systolic blood pressure > 140 mm Hg, EKG evidence of left ventricular hypertrophy).

TABLE VII

PRE-OPERATIVE FACTORS RELATING TO POST-OPERATIVE ISCHEMIA

(Multivariate analysis)

Factors in order of decreasing significance	Stepwise significance when added to previous factors in column
DBP > 90 mm Hg	p = 0.021
SGOT >2 x N	p = 0.018
Palpitations	p = 0.038
SBP > 140 mm Hg	p = 0.036
ECG : PVC	p = 0.042
ECG : LVH	p = 0.002
Peripheral edema	p = 0.020

DISCUSSION

Goldman's Index appears valid in the Orthopaedics population studied but underestimated the incidence of postoperative cardiac complications in the Urology patients. The number of complications in Class I patients was more than twice the percentage predicted by the Goldman Index. Applications of the Detsky Index did not assist in predicting those patients on the Urology service who experienced postoperative cardiac complications.[3] These data suggest that Urology patients, including those undergoing so-called "minor" procedures require a careful medical perioperative evaluation.

We regard this study as a pilot study, and hope that a larger and more extensive study of the preoperative Urology patient population will be pursued. Factors such as hypertension, diabetes mellitus, smoking history and age should be re-evaluated as risk factors for so-called "low risk" procedures such as those commonly performed on the Urology service.

The literature documents the relationship between known ischemic heart disease and silent ischemia.[8] Only a minority of those patients with silent ischemia in this study had a preceding history of either symptomatic ischemia or documented ischemic heart disease. Changes in T wave morphology following anesthesia and surgery are common recovery room phenomena but our patients had postoperative cardiograms 2 - 5 days after surgery and were still noted to have evidence of silent ischemia.[9] Five additional patients had symptomatic ischemia. Only 1 patient in this study had persistent ECG changes and elevation of cardiac enzymes diagnostic of a myocardial infarction. This supports Breslow's study which suggested that postoperative ECG changes involving T waves alone are relatively common and non-specific.[9] However, it would appear prudent to request a postoperative ECG on those patients over the age of 60 who have a history of smoking, hypertension, or diabetes mellitus. These individuals are clearly at risk of manifesting ischemic changes when placed under the stress of anesthesia. Any changes in the ECG suggestive of ischemia should be followed up by evaluation of creatine phosphokinase (CPK) MB isoenzyme levels.

Although this study confirms that the Goldman Index correctly predicts the cardiac risk associated with Orthopaedic procedures, our study suggest that these indices may underestimate the potential cardiac risk of "low risk" Urology procedures. Because the number of patients in this study experiencing serious postoperative complications was small, this conclusion requires confirmation in a much larger population of Urology patients. □

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Risk Factors for Abdominal Aortic Aneurysms

A PRIMARY CARE PHYSICIAN'S GUIDE TO OFFICE BASED SCREENING

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The incidence of abdominal aortic aneurysms has doubled over the past twenty years.[9,10,31] Part of this increase is due to the increasing age of the population.[37] The prevalence of abdominal aortic aneurysm in autopsy studies of men over the age of 65 years may reach as high as 11%.[3] Although improved diagnostic capabilities with ultrasound and CT has aided the incidental detection of small asymptomatic aneurysms, a two fold increase in symptomatic abdominal aortic aneurysm has also been noted.[31] The mortality of abdominal aortic aneurysm was rising by 4% per year in certain centres[13] despite a decrease in the the operative mortality for elective repair from 13% in 1955 to less than 5% in recent series.[34] This low mortality of elective surgery for abdominal aortic aneurysm has similarly been found with patients greater than seventy-five[47] or even eighty years old.[25]. Reasons for the decreased mortality include improvements in surgical and anaesthesia techniques as well as perioperative care.[18]

Definitions for abdominal aortic aneurysm vary between different texts. Collins has suggested that an abdominal aortic aneurysm is present when the maximal external diameter of the infrarenal abdominal aorta is either greater than 4.0 cm or exceeds the diameter of the aorta between the superior mesenteric artery and the left renal artery by 0.5 cm.[17] The natural history of aneurysms is to enlarge[12] at a median rate of 0.3 cm per year.[24] Surgeons will repair aneurysms when risk of rupture is greater than risk of surgery.[9] Treatment of abdominal aortic aneurysms less than 5 cm is controversial, as the risk of rupture varies from 0-8% over a 5 year period.[19,32] Better data is required to determine those that require surgery.[23] With the lower operative mortality, asymptomatic aneurysms 5 cm or greater in diameter should be repaired because the 5 year cumulative rupture rate is 20-25% in recent series.[24,32] Large aneurysms (greater than 6 cm) should be surgically treated since risk of rupture is 50% within one year.[47] All symptomatic aneurysms should be repaired urgently since 33% rupture within one month.[51]

Mortality of ruptured abdominal aortic aneurysm is unacceptable – 100% in those patients who do not reach hospital. Patients entering hospital with ruptured abdominal aortic aneurysm have an operative mortality of 23-59%[4,18,45] for an overall mortality of 75-95%[7] which has not improved significantly over the last twenty years.[33] The post operative cost of ruptured abdominal aortic aneurysm is higher because of greater morbidity resulting in prolonged intensive care unit and hospital stay.[34] Elective repair of abdominal aortic aneurysm is more cost efficient and it returns patients to a near normal five year survival of 65% (expected 75%).[20,34,36]Therefore the best approach is to detect an abdominal aortic aneurysm when it is small and asymptomatic.

Screening programs for abdominal aortic aneurysm in high risk populations have been evaluated hypothetically with projected costs and in small trials with favourable results.[7,8,21,22,33,39,43] Abdominal palpation is a poor diagnostic tool for abdominal aortic aneurysms unless the patient is thin.[5,6,11,36] CT scan is a reliable but expensive screening tool.[36] Abdominal ultrasound can identify the abdominal aorta in 92% of patients screened[42] and reliably predict size.[26] Furthermore, ultrasound is the most cost effective means of detection and surveillance of abdominal aortic aneurysms.[36] In these times of economic restraints it is unlikely that government would fund a generalized screening program for abdominal aortic aneurysm. Patients with increased risk of abdominal aortic aneurysm however could be screened by their primary care physicians and if necessary be referred to a vascular surgeon.

The purpose of this paper is to review the risk factors for abdominal aortic aneurysm so that primary care physicians can identify patients who should undergo abdominal ultrasound. The data of a retrospective study of a recent series of 67 patients who underwent repair of abdominal aortic aneurysm is also discussed. Controversy still exists as to the screening and treatment of abdominal aortic aneurysms, the personal views of the authors are presented.

RISK FACTORS

Age and sex

Abdominal aortic aneurysms generally occur in patients 55 years and older, rarely in men less than 50 years old and is extremely rare in women less than 55 years .[7] The incidence in men rises sharply over the age of 57 years and declines after 85 years while the incidence for women increases more slowly.[7] Death from ruptured abdominal aortic aneurysm is equal in males and females by age 90 years.[7] Screening for abdominal aortic aneurysm is likely to be more effective in males over 50 years and females over 55 years. A male preponderance for abdominal aortic aneurysm has been noted ranging from 8:1 to 15:1.[51]

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Family history

Some authors have suggested an X-linked pattern based on reported families, brothers (over the age of 50 years) of patients with abdominal aortic aneurysm had an incidence of aneurysm of 29% in one such study.[15] Contributing support for this theory is a well recognized mouse model of abdominal aortic aneurysm with X-linked inheritance and Ohno's law of conservation of X chromosome.[50]Males appear to be at greater risk and sons of mothers with abdominal aortic aneurysm especially should be screened.

First degree relatives of patients with abdominal aortic aneurysm have a higher incidence of abdominal aortic aneurysm. Johansen indicated an 11.6 fold increased risk of abdominal aortic aneurysm among first degree relatives of patients with abdominal aortic aneurysm and found that approximately 19% of patients with aneurysm had a previously affected relative which has been confirmed by other authors.[28] Approximately 20% of siblings of affected patients already had a known aneurysm.[11]. Together with the X-linked inheritance pattern at least two modes of autosomal dominant patterns of inheritance have been described.[49,50] Children of women with abdominal aortic aneurysm are especially at increased risk. Cole showed an incidence of 69% of abdominal aortic aneurysm in families where the mother was affected.[11] Similarly others have calculated a genetic component of aneurysmal disease of 70% further emphasising the importance of a positive family history in some[35] if not all.[37]

Race

Autopsy study of males has shown the incidence of abdominal aortic aneurysm to be twice as high in whites as blacks.[29] White females, black females and black males have approximately the same incidence of abdominal aortic aneurysm.[3,29]

Hypertension

Hypertension has been related to increased risk of abdominal aortic aneurysm since the 1950s. Fifty to sixty percent of patients with abdominal aortic aneurysm have systemic hypertension.[19,38] The incidence of hypertension in men with abdominal aortic aneurysm is 2.5 times greater than women with abdominal aortic aneurysm.[37] Rupture of abdominal aortic aneurysm is more common in patients with diastolic hypertension.[19] Some authors have suggested that hypertension acts only as a promoter of abdominal aortic aneurysm formation in affected individuals since many patients with abdominal aortic aneurysm are not hypertensive.[37,46]

Smoking

Smoking, especially heavy smoking (1-2 packages/day) of cigarettes increases the risk of abdominal aortic aneurysm by 8 times compared to nonsmoking individuals of the same age.[3] In some reported series up to 92% of patients were smokers.[30] Male smokers older than 65 years have a 16% incidence of abdominal aortic aneurysm in some series compared to 11% in all men.[3] Like hypertension, smoking appears to be statistically related to abdominal aortic aneurysm formation, however not all patients with abdominal aortic aneurysm smoke.[37] Smoking contributes in some way to the pathogenesis of abdominal aortic aneurysm through the formation of atherosclerotic lesions in susceptible individuals.[3,37]

COPD

Chronic obstructive lung disease, irrespective of tobacco abuse, is the single best predictor of rupture of abdominal aortic aneurysm when comparing chronic obstructive lung disease, smoking and diastolic hypertension.[19] Increased proteolytic enzyme activity affecting both the media of the abdominal aorta and the lung parenchyma has been implicated.[6,27]

Peripheral vascular disease

Patients with atherosclerotic peripheral vascular disease, including lower limb occlusive disease or atherosclerotic lesions of the extracranial carotid vessels, have at least twice the incidence of abdominal aortic aneurysm compared to the general population.[43] The literature shows a 57-65% incidence of coronary artery disease in patients with aneurysms, higher than the general population.[18,41]Atherosclerosis is known to affect up to 84% of patients with abdominal aortic aneurysm.[18]

Presence of other aneurysms

A very good predictor of abdominal aortic aneurysm is a previous popliteal aneurysm as 40% of these patients will have an abdominal aortic aneurysm.[2] Aortoiliac aneurysmal disease is present in 85% of patients with femoral and popliteal artery aneurysms.[21]

TABLE I

SUMMARY OF RISK FACTORS FOR ABDOMINAL AORTIC ANEURYSM

1. Age, >50 for males, >55 for females.
2. Males of Caucasian race
3. Affected first degree relative especially a mother.
4. Diastolic hypertension
5. Smoking
6. Chronic obstructive lung disease
7. Peripheral vascular disease
8. Presence of other aneurysms

This list of risk factors is not exaustive but covers the major factors which are easily identified.

RECENT EXPERIENCE WITH ABDOMINAL AORTIC ANEURYSM

Sixty-seven patients underwent repair of abdominal aortic aneurysm by a single surgeon at Camphill Hospital in Halifax, Nova Scotia from June 1, 1988 until May 31,1990. There were 62 males with an average age of 68.8

years (range 53-89 years) and 5 females with an average age of 72.6 years (range 56-88 years). Fifty-five of these patients underwent elective repair and 12 had emergency surgery. Six of the emergency surgeries were for ruptured abdominal aortic aneurysm and 6 were for symptomatic but not ruptured abdominal aortic aneurysm. The majority of patients in this series were diagnosed by physical examination and direct palpation of a pulsatile mass, or by radiological investigation for some other reason.

Preoperative risk factors included hypertension and ischemic heart disease in 31% of patients. Chronic obstructive lung disease was present in 34% of the patients. Transient ischemic attacks had occured in 10% of patients.

RESULTS

The average size of of the aneurysms was 6 cm – 12 of the aneurysms were less than 5 cm and were resected in conjunction with other aorta surgery. The overall in hospital mortality was 3.6% for elective repair and 25% for emergency surgery. Of the emergency patients, 1 of 6 with ruptured aneurysm died for a 17% mortality and 2 of 6 patients with a symptomatic aneurysm died for a 33% mortality in these subgroups.

Morbidity overall was 45%, with a 42% morbidity for elective repair and a 55% morbidity for emergency surgery.

DISCUSSION

Abdominal aortic aneurysm is the thirteenth leading cause of death in the North American population.[44] Screening for abdominal aortic aneurysm on a mass scale could be an enormous and expensive undertaking.[36]. By selecting those patients for screening who are at an increased risk for abdominal aortic aneurysm the number of positive tests will increase while the total number requiring an ultrasound will be less. Decisions about who to screen can be determined by referring to Table I.

Screening men of 50 years and women of 55 years increases the likelihood of finding an abdominal aortic aneurysm to greater than 0.5% based on age alone. By age 75 years the incidence is 5% for men and 1% for women.[7] Some have suggested that screening is justified in all patients over 50 years.[40] Other authors advocate screening women as well even though the cost of screening is higher since the cost is offset by a longer life expectancy.[39] There is a suggestion that the incidence of abdominal aortic aneurysm will rise in women as more of these females who come of age will be smokers.[13,39]

Screening patients with other risk factors will increase the yield of positive tests. Men greater than 74 years with peripheral vascular disease will have a 14% incidence, those with hypertension a 12% incidence, and those with carotid artery surgery a 12% incidence of abdominal aortic aneurysm.[7] Collin has suggested that all elderly hypertensive males be screened as part of their routine physical examination.[16]

Those patients with an affected first degree relative, especially a mother or brother and are over 50 years of age have absolute indications for screening.[11]The high incidence of abdominal aortic aneurysm associated with femoral and popliteal aneurysms makes this an absolute indication for abdominal ultrasound.[2,21]Femoral and popliteal aneurysms can be recognized or suspected on physical examination and will require sonography for investigation.

White males over the age of fifty years with any combination of chronic obstructive lung disease, atherosclerosis or heavy smoking are at risk of abdominal aortic aneurysm. Those patients with abdominal aortic aneurysm who also have chronic obstructive lung disease and diastolic hypertension are especially at risk of rupture.[19]

The data presented from this small series support the concepts of risk factors associated with abdominal aortic aneurysm. Men outnumbered women by a ratio of 12:1. Males were on average younger than females. Many of the patients had pre-existing chronic obstructive lung disease, atherosclerosis and diastolic hypertension. Although not stated previously, many of the patients were present or past heavy smokers. The operative mortality for elective surgery of 3.6% for this series has fallen from 5.9% presented by the same surgeon in 1983[51] and is within the standards of other published series.[1,10]

In Nova Scotia, the majority of abdominal aortic aneurysms are diagnosed by physical examination and direct palpation of a pulsatile mass or as an incidental finding of radiological investigation for some other reason. Since 9% of the patients in this series presented with symptomatic disease and 9% presented with rupture, the health care system is not effectively identifying patients at risk who require surgery similar to other world centres.[9] This problem is also seen at other centres in Canada such as the Toronto General where 24% of patients present with ruptured abdominal aortic aneurysm.[4]

The efficacy of repairing abdominal aortic aneurysm has been established, surgery extends survival.[13,34,41] The risk of surgery for asymptomatic aneurysms is less than 5% while risk of rupture for aneurysms 5 cm or larger is certainly equal to or greater and will increase as size of the aneurysm increases.[12] To give patients the best chance for survival, it is important to identify the disease before it is symptomatic. Mortality for emergency surgery has not improved over the years while mortality of elective repair has markedly improved and is now quite acceptable.

Abdominal aortic aneurysms do not tend to occur in otherwise healthy people.[51] On average each patient will have 2 other chronic diseases including Ischemic heart disease (65%), hypertension (37-60%), peripheral vascular disease (33%), pulmonary disease (27%).[19,38,41] These patients tend to come under the scrutiny of their primary care physician on a regular basis already.[22,23] Age alone is not a contraindication to elective surgery.[24,47] Elderly patients, often more fragile, are unlikely to do as well with emergency repair for abdominal aortic aneu-

rysm, therefore once a diagnosis of aneurysm is made refer early.

Screening with ultrasound should be every 5 years in high risk patients with otherwise normal aortas as it is unlikely that a normal aorta will go on to rupture over this time period.[14] Patients with a known abdominal aortic aneurysm are followed more closely by their surgeons.

The risk of abdominal aortic aneurysm is multifactorial, however identifiable. Ultrasonic screening is cheap, accurate, safe and highly acceptable to subjects.[39] The cost of abdominal ultrasound of the aorta in Nova Scotia is approximately thirty dollars, the technician's salary and equipment costs are not included because these are fixed. The cost compared with other screening programs for diseases such as breast cancer, is low and once the disease is corrected by surgery the patient returns to an almost normal life expectancy unlike patients with carcinoma.[42] Compared with screening programs in other countries such as the United States, screening for abdominal aortic aneurysm in Canada would be cheaper compared with hypothetical screening programs in American centres because of the difference in renumeration.[36]

Numerous reports have justified screening for both selected and unselected populations. Now is the time to institute at least selected screening in our province. The success of primary care physician office based screening will be measured in the number of asymptomatic patients with abdominal aortic aneurysm referred to vascular surgeons over the upcoming years and hopefully a declining mortality from abdominal aortic aneurysm. □

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PREOPERATIVE RISK ASSESSMENT IN PATIENTS UNDERGOING UROLOGICAL OR ORTHOPAEDIC SURGERY

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When the facts which come under observation are opposed to accepted theory, abandon the theory, however distinguished its supporters, and accept the facts.

Claude Bernard (1813-1878)

Medical Empowerment

THE MAKING OF A MICMAC SHAMAN

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The pathway to becoming a doctor in Nova Scotia is well marked. Public school, university studies, the writing of the MCAT, applications, interviews, medical school, and internship are the common experiences of almost every doctor in this province. It is an empowering pathway, intently supported by strong institutions. It allows individuals to have profound effects on people's lives and the lives of communities. Long before Dalhousie Medical School, there was another healing tradition in Nova Scotia – the shamans, or puoinaq, of the Micmac people. They rose to a similar prominence in their communities through a self-styled empowerment. Their path of empowerment, and the world in which it happened, is what this paper will explore.

A WORD ABOUT SOURCES

The Micmac are a group of Algonkian speaking people who once broadly inhabited what is now Atlantic Canada and parts of Maine. Today, the inhabit mostly government reservations on these lands. For more than four centuries, the Micmac culture has been absorbed or subjugated by European expansion. The People have survived but most of their "pre-contact" ways of thinking have been lost, as there was no original written language. Still, fragments of these thought patterns have survived through oral tradition (legends) and the Micmac dialect.

The purest source for examining shamanism is the legends and the language of the People themselves. There is a temptation to rely solely on the tidy historical accounts left by French explorers when researching this area. These accounts are useful, but deal mostly with appearances. Micmac shamanism should be interpreted in the light of Micmac legends rather than the light of European history.

THE IMPORTANCE OF PERSONAL POWER IN HUNTER-GATHERER SOCIETIES

The Micmac at the time of European contact were a hunter-gatherer society. Hunter-gatherer societies tend to develop systems based on the acquisition of personal power and individualistic orientation, unlike agricultural societies which tend to develop value systems based on balance and harmony.[2] This idea of personal power must be examined in order to understand the culture of the Micmac people and the formation of the role of the shaman.[3]

TYPES OF POWER IN LEGEND

The legends of the Micmac gives clues to the shades of power that existed in their world. Kinap, Mn'tu and Puoin are all Micmac words for power.[4]

Kinap is physical strength. A Person called kinapaq can run faster than the wind, tear trees in half and pound great holes in the ground as he dances.

Mn'tu is spirit-power (the more familiar form of this word is "manitou"). The legend of Nuji-Kesi-Kno'tasit tells of a youngest son who is given a gift of a small box with a mn'tu inside.[5] This mn'tu is in the form of a small man who is a spirit-helper of great power. He levels mountains and destroys an entire village for the youngest son.

Puoin is the third Micmac word for power and is best understood by regarding the people called puoinaq. Puoinaq are those who have power to transcend the boundaries of time and form. These are the shamans, or the healer/destroyers. Like the old woman in the legend Lamkisn, they can see "ten days into tomorrow".[6] Most notably the puoinaq are shape changers of great ability.

MANIFESTATIONS OF POWER IN LANGUAGE

With the addition of animate word endings in the Micmac language, the forms of power mentioned above can manifest themselves in a variety of combinations within the legends.[7] These forms include humans, animals, trees and some, but not all, plants. They also include stars, thunder, winds, mountains, lakes, icebergs and other objects which are usually not considered animate in European eyes except in the poetic imagination. The Micmac also give their words for seasons and directions the animate endings.

Recombinations of the Micmac language show how this array of animated beings also possesses the ability to change shape. In this world the boundaries between "human" and "not human" become blurred and faded. There are tree-persons, there is the bear-woman, the mouse-person, the star-person-hunters, and thunder-persons who are also giant birds.

EMPOWERMENT OF SHAMANS IN MICMAC SOCIETY

Between legend and language it is possible to understand where power manifests itself. The acquisition of a certain type of power (called puoin) is what creates a

* First Year Medical Student, Dalhousie Medical School, Halifax, N.S. This paper is the result of an elective under the preceptorship of Dr. Ian Cameron, Director of Residency Training, Dalhousie Medical School.

shaman. There are no set paths to this end as this is a society that emphasizes individuality.

The legend of Skun tells of an impoverished and isolated man who is at the deathbed of a puoinaq he had befriended in life.[8] He receives the clothes, and therefore the power, of this puoinaq. In another legend, Mi'Kmwesu is visiting Kluskap and soils himself in the night. Out of this misfortune he gets a bath, a new set of clothes, and a hair-string of power from Kluskap.[9] In another legend, Sakklo'pik is described as an ugly, lazy boy but through his alliance with a chief's son he gains power enough to call Putup, the whale-person. Under Putup's tongue there is a gift of powerful medicine.[10]

Power is never shapeless, so its acquisition always involves interaction with a person and usually involves forming some sort of alliance. Often the person of power makes a gift to the central character of the story.

When considering the candidates for shamanism, it is interesting to note that in the legends it is often outsiders or partial outcasts who are graced. Whether their misfortune is poverty, incontinence, or ugliness, it is often the disadvantaged that end up with puoin. Mythologist Joseph Campbell once commented, "The Shaman is the person, male or female, who in his late childhood or early youth has an overwhelming psychological experience that turns him totally inward . . . The whole consciousness opens up and the Shaman falls into it".[11] This opening of consciousness and psychological turning inward is an experience that could lead to ostracism or institutionalization in some cultures. Yet some individuals with great powers of expression have become artistic visionaries after such an experience. Perhaps it was these types, in Micmac society, who became the puoinaq.

THE PRACTICES OF THE PUOINAQ

The legends most often describe puoinaq as shape-changers in competition with one another. Sometimes these are friendly competitions, other times, to the death. Often the puoinaq use their power to protect themselves or their families. Their power resides not only in the ability to transcend time and form, but in knowledge and cunning.

In the legend Mimkitawo'qu'sk, two puoinaq have a growing contest.[12] One chooses to change shape into an elm tree, and his rival choose to be a pine. Both grow tall, but when a large wind blows the pine tree alone is knocked over. Only one shape-changer knew that pine trees have shallow roots and a type of branch that tends to catch the wind. In a similar legend a puoinaq named Mi'kmwesu wins a contest by choosing to become chain lightning to race against northern lights. When daylight comes, chain lightning is still able to run around the sky, but northern lights has to wait for darkness to come again to finish the race.[13] Power is not perceived to lie in physical strength alone, but also in foresight and in knowledge of the natural world.

THE MAN WHO MARRIED JIPIJKA'MI'SKM – A HEALING

The puoinaq are less often portrayed as healers attending to the sick. The Legend of The Man Who Married Jipijka'Mi'Skw is one story that portrays a healing in some detail and gives a rare look into the nature of the process.[14] This legend tells of a man who becomes fascinated by a furrow in the ground and falls under the spell of a Jipijka'm (these are horned serpent-people that live under the earth and under the water. They are not friends of the Micmac). He is changed into a Jipijka'm and follows the smell of a horned serpent woman to an underwater wigwam where he is married to the woman. The man's brother sees this tragedy unfold and in great alarm, solicits the help of a puoinaq.

The central action of the shaman is to climb a tree and cut off the branches below him as he climbs. Then, on this tree, he sits and waits. After a time, two serpent-persons come from beneath the earth. One coils around the tree. The puoinaq is able to see that this serpent-person is really the lost brother. The shaman pulls out his knife and cuts off the head of the serpent-person, and pulls the lost brother out of the carcass.

The central symbol of the story is the tree with no branches. The tree provides an elevated vantage point, and may represent a place the shaman goes in his consciousness to do battle with the malevolent forces that have captured the man. This place has a different, perhaps more complete, perspective. It is also a place he goes alone, made certain by his cutting of the lower branches of the tree (the individuality of the shamanic experience is here emphasized).

The healing is also a destroying. There is decisive violence in the shaman's actions and a symbolic death and bloody rebirth of the man is part of the healing. To the puoinaq, healing and destroying are the same thing. "This is not a reversal of role; it is simply another way of using power."[15]

The early French explorers' written accounts mention Micmac shamans working up into a state of "sweat and lather"[16], speaking to the mn'tu, and dancing with such "fury that they emitted foam as big as fists on both sides of the mouth".[17] Interpreted in the light of Micmac legends these actions were probably used to induce a trance state (a tree with no branches) in which the puoinaq could operate. Through this trance state he could transcend time and form. This trancendence was also valued for weather forecasts, predicting the outcome of battles, and for identifying good hunting ground.[18]

THE PLACE OF THE PUOINAQ IN THE MICMAC HEALTH TRADITION

The puoinaq constitute only a part of the health traditions of the Micmac. In 1675, Father LeClerq of New France was to write that the Micmac, "are all by nature physicians, apothecaries, and doctors, by virtue of the knowledge and experience they have of certain herbs, which they use successfully to cure ills that to us seem

incurable".[19] In the light of Micmac mythology, one could interpret this general knowledge of medicinal plants as a widespread alliance with animate plant-spirits, thereby being privy to their power. Indeed the legends bear this out with stories of "Plant-Persons, who visit as humans, to instruct the People in the use of their plant forms and properties."[20] This power is the same in principle as the puoinaq or kinapaq acquire. In practice, it was a widespread power, and therefore probably less revered.

In addition to being accomplished herbalists, the Micmac "excelled even European surgeons at healing wounds, setting fractures, and replacing dislocations" (all necessary skills amongst hunters and warriors).[21] Their sweat lodge was a "general remedy . . . which they did every month and even oftener" and was important for the rheumatism which their lifestyle could precipitate.[22] Dancing was considered "profitable for the preservation of their health."[23] Blood-letting, to arrest infection, was common to the point that LeClerq described them as "great lovers" of the procedure.[24]

Every health procedure that the Micmac used can be viewed as an alliance with the power of the spirit-person: The power of the plant-person provides the medicine; the power of the tree-person provides the splint for the broken arm; the power of the fire heats the rock that warms the sweathouse; and the power of the flint is needed for bloodletting. This was the Micmac way of moving through the world, making alliances of power. When an illness was beyond the more common of the power-alliances (with the plants, or the bloodletting flint), then the People would seek alliance with the puoinaq, whose network of alliances transcended those of most of the People.

THE DECLINE OF THE PUOINAQ

There are but a few Micmac shamans practising today. As early as the 1630s there are stories of Jesuit priests replacing Micmac shamans as spiritual advisors and healers.[25] This is a generalized pattern in transitions from hunter-gatherer societies to agricultural societies. Priests are empowered by social ordination rather than psychological experience and suit a more settled, organized mode of existence. This transition is documented in the Navaho and Apache cultures as they migrated and settled into agricultural life in the Southwestern United States.[26] There are typical stories of shamans being upstaged and ridiculed and priests taking over.[27]

Another factor in the power struggle between Jesuits and shamans was the introduction of European diseases to which the Micmac had no immunity, and no experience.[28] In the face of these new scourges (smallpox, typhoid, measles . . .) the Jesuits appeared to hold more power. It was natural then for the Micmac to seek alliance with this power through baptism and conversion to Catholicism. As early as 1638, the Jesuit LeJeune could write:

"The sorcerers and jugglers have lost so much of their credit that they no longer blow upon any sick person, nor beat their drums, except perhaps at night, or in isolated places, – but no longer in our presence . . . The other superstitions will be suppressed little by little. When any one of them does practise these, he does all he can to prevent our being informed of it, for fear of being reported".[29]

CONCLUSION

The making of a shaman is a process that may never be fully understood given the loss of the Micmac's original view of the world. From the legends and the language of the Micmac, one can begin to see that their's was a world populated by animate forms of power. To make one's way in this world required alliances with these powers and acquisition of personal power. The shamans were often outsiders who were given a gift of puoin, the power to transcend time and space. They may have used trance states to perform healings and make predictions. The puoinaq engaged in a self-styled career. There were no timetables for shamanism, and no guarantees of continued acceptance in their field except for the strength of their personal power and the strength of their alliances. □

ACKNOWLEDGEMENTS

I am grateful to Ruth Holmes Whitehead, and Sarah and Kevin Michael-Sack for time and conversation.

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An Appreciation

DR. C.R. BENSON AULD, FRCSC

Charles R. Benson Auld, M.D. died Feb. 2, 1992 after a short illness at age 68.

He was born in Charlottetown, P.E.I. where he received his early education, graduating from Prince of Wales College prior to entering Acadia University and pre-medical studies. He graduated from McGill University with his M.D. in 1949 and spent the next four years in Halifax, where he obtained his surgery specialty in 1953 at Dalhousie University.

He held active staff appointments at Camp Hill, Halifax Infirmary and the Children's hospitals and, most recently the Dartmouth General Hospital after his retirement from the University Surgery Department in 1990. He officially retired from practice on June 30, 1991. He taught both undergraduate and post graduate students and he will be remembered by most of them for his easy and relaxed approach in the classroom and the operating room. Bense always found time, even on busy days, to share a light hearted moment with other staff employees who returned to their duties with a smile on their faces. As a good friend and journalist wrote, "He touched the lives of many people in a very special way."

Community affairs benefited from his interest and participation which he shared liberally with many well known organizations – The VON, N.S. Lung Association, his favorite political affiliation and the First Baptist Church. He also found time to serve on the Criminal Injuries Compensation Board, which served those in the province whose lives were affected by a variety of unlawful criminal acts. His days were long and full and very often, time with his family, short, because of his inability to say no.

His wit and sense of humor were well known, and great numbers of people were entertained by his stories because he was an excellent story teller and his repertoire was usually of high quality. Smiles will cross the faces of friends and associates for a long time as they remember one of his many classic tales.

My fondest memories center around his competitive spirit and his exceptional desire to win. Golf, curling, bridge and fishing were his favorites and his thoughts were never about losing. A short story will serve to prove this point. He was entered in one of his favorite golf tournaments to be played on Saturday, but much to his other team members sorrow, he sustained an unfortunate accident the day prior, by amputating his ring finger while mowing his lawn. He spent the greater part of the evening in surgery and the recovery room and, upon awakening, he informed his team captain not to seek a replacement as he planned if permitted to participate himself. With permission from his Doctor and tournament director, he played in considerable pain and yes, won the event. Just a few years ago, he was honored by winning "Player of the Year Award" at the Halifax Curling Club where he was an active member for many years. He was also a member of Ashburn Golf Club for practically the same length of time he was in medical practice. Regardless of the event but especially in golf, a small wager always sweetened the competition and he made sure this was duly settled before the match began.

He will best be remembered, I believe for his love and devotion to his family. For those who were close to him and knew him personally, they will approve of this observation without reservation. Family matters came first and even his beloved sports were relegated to second place. He loved his family and it was very apparent during his illness, they accorded him their deepest affection and admiration. Being a strong family unit, sustained them through his illness and at the time of his death, I believe there was joy in sorrow. He was truly a caring family man and my family having known the Aulds somewhat intermittently for over thirty years have been enriched by this association.

As my two daughters might have said when they were quite young "the angels have a *new best friend* in their midst."

Deepest sympathies are extended his wife, Alice, daughters Sherrie, Marsha and Pamela, son Gregory and two sweet grand children, Anna and Nicholas.

R.L. Langdon, M.D.
Halifax, N.S. □

A good problem is one whose solution, rather than merely tidying up a dead end, opens up entirely new vistas. Most good problems are hard: in mathematics, as in all walks of life, one seldom gets something for nothing. But not all hard problems are good: intellectual weight-lifting may build mental muscles, but who wants a muscle-bound brain?

Ian Stewart, *The Problems of Mathematics*

Current Topics in Community Health

Selected by: Dr. David R. MacLean
Department of Community Health & Epidemiology
Dalhousie University, Halifax, N.S.

CHLAMYDIA INFECTION IN NOVA SCOTIA

Sexually transmitted diseases continue to be a significant clinical and public health problem in Nova Scotia, and are largely diseases of young people. This fact is a reflection in large measure of the significant degree of sexual activity among teenagers and youth and the failure of these individuals to consistently take protective measures which would significantly reduce their chances of acquiring these diseases. In Nova Scotia in 1991, approximately 25% of all sexually transmitted diseases occurred in individuals under the age of 19, and approximately 75% occurred in individuals under the age of 24.

Sexually transmitted chlamydia infections caused by *Chlamydia trichomoniasis* are one of the most common and most important of all sexually transmitted diseases. This infection is highly prevalent in the Nova Scotian population and has a number of significant long-term consequences such as pelvic inflammatory disease, etopic pregnancy and infertility in women. It is also very common worldwide, with over 300 million cases reported on an annual basis.

Clinical manifestations of the disease in men include, urethritis, epididymitis, prostatitis, proctitis and Reiter's syndrome. In women, the organism causes mucopurulent cervicitis, the urethral syndrome, endometritis, salpingolithiasis and perihepatitis. In neonates, chlamydial infection often is manifested by conjunctivitis and pneumonia as a result of maternal infection. As most clinicans recognize, chlamydia can at times be difficult to diagnose, due to asymptomatic infection or the presence of non-specific signs and symptoms.

Chlamydia infection is of significant public health importance in Nova Scotia. It appears to be increasing in incidence despite the fact that it is both preventable and treatable. The laboratory diagnosis has improved significantly over the last number of years and appropriate testing is now available to all physicians in the province. If we are to control this disease and ultimately reduce its incidence, greater efforts will have to be made to promote the testing of high-risk groups, more aggressive contact tracing, reporting and appropriate treatment. As chlamydia is now nationally notifiable in Canada, better analysis of trends should be available in the future with hopefully a decline observed as has occurred with gonorrhoea.

INTERIM GUIDELINES FOR THE TREATMENT OF UNCOMPLICATED GONOCOCCAL INFECTION

The Department of Health and Welfare are currently revising their guidelines for the diagnosis, management and treatment of sexually transmitted diseases in children, adolescents and adults. It is anticipated that these revised guidelines will be available in 1992. The Laboratory Centre for Disease Control in Ottawa has released interim guidelines for the treatment of uncomplicated gonorrhoea, primarily because of the increasing numbers of gonococcal organisms which are becoming resistant to penicillin and in some cases, tetracycline.

Below are the interim guidelines which have recently been released from Health and Welfare Canada for the treatment of adolescents and adults. These guidelines do not apply to pregnant women, nursing mothers and those under the age of 16 years. It is anticipated that treatment guidelines for complicated gonococcal infections and disease in children will be forthcoming later this year.

Urethral, Endocervical and Rectal Infections

Preferred:

• ceftriaxone 250 mg IM(c) in a single dose **PLUS** tetracycline/doxycycline(d)

Alternative:

• spectinomycin 2 g IM in a single dose **PLUS** tetracycline/doxycycline(d)

The following alternative oral regimens may be effective against penicillin and tetracycline-resistant organisms and should only be used if laboratory monitoring of susceptibility is being carried out:

•ciprofloxacin 500 mg in a single dose OR ofloxacin 400 mg orally in a single dose **PLUS** tetracycline/doxycycline(d). **OR** cefixime 800 mg orally in a single dose **PLUS** tetracycline/doxycycline(d) **OR** cefuroxime axetil 1 g orally in a single dose **PLUS** probenecid 1 g orally in a single dose **PLUS** tetracycline/doxycycline(d)

The following regimen should only be used in areas with active monitoring for resistance to penicillin ***AND*** *if the percentage of penicillin-resistant isolates is <3.0%(e)* ***AND*** *if the infection was acquired in the same geographic area:*

• amoxicillin 3 g orally **OR** ampicillin 3.5 g orally in a single dose **PLUS** probenecid 1 g orally in a single dose PLUS tetracycline/doxycycline(d)

a) for pregnant women and nursing mothers the treatment regimens for adults and adolescents should be followed except that quinolones (ciprofloxacin and ofloxacin) are contraindicated and tetracycline/doxycycline should be replaced by erythromycin 500 mg orally x 4/day for at least 7 days **OR** if not tolerated erythromycin 250 mg x 4/day for 14 days may be substituted. Erythromycin dosages refer to the use of erythromycin base. Equivalent dosages of erythromycin stearate or erythromycin ethylsuccinate may be substituted (erythromycin estolate is contraindicated in pregnancy). For those <16 yrs of age the treatment regimens for adults and adolescents should be followed except that quinolones (ciprofloxacin and ofloxacin) are contraindicated.

b) ceftriaxone, cefixime, cefuroxine, amoxicillin and ampicillin should not be given to persons with cephalosporin allergy or a history of immediate and/or anaphylactic reactions to penicillins.

c) ceftriaxone is the preferred treatment in cases where there are no antimicrobial susceptibility data to rule out resistant isolates. The preferred diluent for ceftriaxone is 1% lidocaine without epinephrine in order to reduce discomfort.

d) tetracycline 500 mg orally x 4/day for 7 days **OR** doxycycline 100 mg orally x 2/day for 7 days is required for treatment of presumptive or proven infection with *Chlamydia trachomatis.*

e) contact your local public health authority if you are unsure as to the situation in your area; if in doubt use *Preferred* regimen.

The above material was contained in *Canada Diseases Weekly Report*, 25 December 1991. □

OBITUARIES

The Medical Society is making a contribution to the Memorial Fund for Nova Scotia Physicians on behalf of **Dr. Charles M. McBride** of Houston, Texas who died on January 17, 1992 at the age of 66. Dr. McBride was a member of The Canadian Medical Association and has been a member of The Medical Society of Nova Scotia since 1957. The *Journal* expresses sincere sympathy.

Dr. Lloyd A. MacLeod, (74) of Truro, Nova Scotia died on March 25, 1992. Born in Inverness he received his medical degree from Dalhousie Medical School in 1942. He finished his post graduate training in general surgery in 1966 and from 1946 until his retirement in 1986, he practised as physician and surgeon in Liverpool. Following retirement, Dr. MacLeod and his wife moved to Truro. We offer sincere sympathy to his wife and family.

Dr. Geoffrey J. Whiston, (68) of Halifax, Nova Scotia died on April 19, 1992. Born in Edinburgh, Scotland, he was a pilot in the RAF after which he received his medical degree in Edinburgh in 1953, and he joined the Royal Canadian Air Force as a medical officer specializing in ophthalmology. He is survived by his wife to whom the *Journal* extends sincere sympathy.

Dr. F. Murray Fraser, (86) of Halifax, Nova Scotia died on May 4, 1992. Born in Halifax he received his medical degree from Dalhousie Medical School in 1932. He practised family medicine in Halifax for forty years. In 1954, he became the first president of Nova Scotia chapter of College of Family Physicians, in 1960 he was elected national president and in 1973 he received the Family Physician of the Year Award. He was president of Maritime Medical Care Inc. and the Dalhousie Medical Alumni Association and chairman of Medical Services Insurance. He is survived by his son, and three grandsons. The *Journal* extends sincere sympathy to his family. □

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Anti-inflammatory analgesic agent

ACTION and CLINICAL PHARMACOLOGY: Animal pharmacological studies have shown that ketoprofen possesses anti-inflammatory, analgesic and antipyretic properties. The anti-inflammatory action is not mediated through the pituitary adrenal axis. Its therapeutic effectiveness has been demonstrated by a reduction in joint swelling, pain and duration of morning stiffness, and by increased grip strength and an improvement in functional capacity. Clinical trials in patients with rheumatoid arthritis and osteoarthritis have shown that when given in a dose of 200 mg once daily, the anti-arthritic activity of Oruvail is comparable to that of a twice daily administration of ketoprofen (100 mg ketoprofen b.i.d.). Ketoprofen 200 mg daily induced less gastrointestinal bleeding than acetylsalicylic acid 4 g/day. **Pharmacokinetics properties:** Ketoprofen from Oruvail is slowly but almost completely absorbed from the gastrointestinal tract. Mean peak plasma levels of 2.2 and 4.2 mg/L are achieved about 5 hours following single oral doses of Oruvail 100 and 200 mg, respectively. Pharmacokinetics are linear over a dosage range of 100 to 200 mg. The systemic availability of Oruvail is 95% of that of conventional capsules. In a food-effect study, meal composition did not affect the extent of absorption of ketoprofen from Oruvail, although a heavy meal slightly but significantly delayed the absorption of the drug by about 2 hours by comparison to a light meal; in this study, there was no comparison with the fasted state nor with a conventional ketoprofen formulation. Steady-state plasma ketoprofen concentrations are achieved within 4 days with mean peak and trough levels of 4.3 and 0.91 mg/L respectively, after repeated doses of 200 mg once daily. There is some evidence that C_{max} and bioavailability are increased in the elderly as the result of an age-related reduction in volume of distribution since the apparent elimination half-life of about 8 hours is similar in both young and elderly patients. No or negligible accumulation of ketoprofen was found following repeated once daily dosing of Oruvail 200 mg capsules in either young or aged subjects. In arthritic patients treated with Oruvail 200 mg once daily for up to 3 months, the steady-state disposition of ketoprofen remains unaltered during chronic administration. When comparing to a group of healthy subjects, no differences with respect to AUC, C_{max}, and elimination half-life were found, indicating that inflammatory joint disease has no influence on the kinetics of Oruvail capsules.

INDICATIONS and CLINICAL USES: Oruvail (ketoprofen) is indicated in the treatment of rheumatoid arthritis, ankylosing spondylitis and osteoarthritis.

CONTRAINDICATIONS: Oruvail (ketoprofen) is contraindicated in patients with active peptic ulcers or active inflammatory diseases of the gastrointestinal tract. Oruvail is also contraindicated in patients who have demonstrated hypersensitivity to the drug. Because of cross-sensitivity, ketoprofen should not be given to patients in whom acetylsalicylic acid (ASA) and other nonsteroidal anti-inflammatory drugs induce symptoms of asthma, rhinitis or urticaria. Fatal anaphylactoid reactions have occurred in such individuals.

WARNINGS: Peptic ulceration, perforation and gastrointestinal bleeding, sometimes severe and occasionally fatal have been reported during therapy with nonsteroidal anti-inflammatory drugs (NSAID's), including Oruvail (ketoprofen). Unlike most adverse reactions, which usually manifest themselves in the first month if they are going to occur in an individual, new peptic ulcers keep appearing in patients under treatment with ketoprofen at a rate of greater than 1% per year. Oruvail should be given under close medical supervision to patients prone to gastrointestinal tract irritation particularly those with a history of peptic ulcer, diverticulosis or other inflammatory disease of the gastrointestinal tract. In these cases the physician must weigh the benefits of treatment against the possible hazards. Patients taking any NSAID including this drug should be instructed to contact a physician immediately if they experience symptoms or signs suggestive of peptic ulceration or gastrointestinal bleeding. These reactions can occur without warning symptoms or signs and at any time during the treatment. Elderly, frail and debilitated patients appear to be at higher risk from a variety of adverse reactions from nonsteroidal anti-inflammatory drugs (NSAID's). For such patients, consideration should be given to a starting dose lower than usual, with individual adjustment when necessary and under close supervision. See "Precautions" for further advice. **Use in Pregnancy:** The safety of Oruvail when administered to pregnant or nursing women has not been determined and therefore such use is not recommended. Pregnant rats who received ketoprofen 6 and 9 mg/kg/day p.o. from day 15 of gestation, showed dystocia and increased pup mortality. **Nursing mothers:** In rats, ketoprofen at doses of 9 mg/kg (approximately 1.5 times the maximum human therapeutic dose) did not affect perinatal development. Upon administration to lactating dogs, the milk concentration of ketoprofen was found to be 4 to 5% of the plasma drug level. Data on secretion in human milk after ingestion of ketoprofen do not exist. As with other drugs that are excreted in milk, Oruvail is not recommended for use in nursing mothers. **Use in Children:** The conditions for safe and effective use of Oruvail in children under 12 years of age have not been established and the drug is therefore not recommended in this age group.

PRECAUTIONS: Gastrointestinal system: If peptic ulceration is suspected or confirmed, or if gastrointestinal bleeding or perforation occurs Oruvail (ketoprofen) should be discontinued, an appropriate treatment instituted and patient closely monitored. There is no definitive evidence that the concomitant administration of histamine H_2-receptor antagonists and/or antacids will either prevent the occurrence of gastrointestinal side effects or allow continuation of Oruvail therapy when and if these adverse reactions appear. **Renal function:** As with other nonsteroidal anti-inflammatory drugs, long-term administration of ketoprofen to animals has resulted in renal papillary necrosis and other abnormal renal pathology. In humans, there have been reports of acute interstitial nephritis with hematuria, proteinuria, and occasionally nephrotic syndrome. A second form of renal toxicity has been seen in patients with prerenal conditions leading to the reduction in renal blood flow or blood volume, where the renal prostaglandins have a supportive role in the maintenance of renal perfusion. In these patients, administration of a nonsteroidal anti-inflammatory drug may cause a dose-dependent reduction in prostaglandin formation and may precipitate overt renal decompensation. Patients at greatest risk of this reaction are those with impaired renal function, heart failure, liver dysfunction, those taking diuretics, and the elderly. Discontinuation of nonsteroidal anti-inflammatory therapy is usually followed by recovery to the pre-treatment state. Ketoprofen and its metabolites are eliminated primarily by the kidneys, therefore the drug should be used with great caution in patients with impaired renal function. In these cases lower doses of Oruvail should be anticipated and patients carefully monitored. During long-term therapy kidney function should be monitored periodically. **Hepatic function:** As with other nonsteroidal anti-inflammatory drugs, borderline elevations of one or more liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful (3 times the upper limit of normal) elevations of ALT or AST occurred in controlled clinical trials in less than 1% of patients. A patient with symptoms and/or signs suggesting liver dysfunction, or in whom an abnormal liver test has occurred, should be evaluated for evidence of the development of more severe hepatic reaction while on therapy with this drug. Severe hepatic reactions including jaundice and cases of fatal hepatitis have been reported with this drug as with other nonsteroidal anti-inflammatory drugs. Although such reactions are rare, if abnormal liver tests persist or worsen, if clinical signs and symptoms consistent with liver disease develop, or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), this drug should be discontinued. During long-term therapy, liver function tests should be monitored periodically. If this drug is to be used in the presence of impaired liver function, it must be done under strict observation. **Fluid and Electrolyte Balance:** Fluid retention and edema have been observed in approximately 2% of patients treated with ketoprofen. Therefore, as with many other nonsteroidal anti-inflammatory drugs, the possibility of precipitating congestive heart failure in elderly patients or those with compromised cardiac function should be born in mind. Oruvail should be used with caution in patients with heart failure, hypertension or other conditions predisposing to fluid retention. Serum electrolytes should be monitored periodically during long-term therapy, especially in those patients at risk. **Hematology:** Drugs inhibiting prostaglandin biosynthesis do interfere with platelet function to some degree; therefore, patients who may be adversely affected by such an action should be carefully observed when Oruvail is administered. Blood dyscrasias associated with the use of nonsteroidal anti-inflammatory drugs are rare, but could be with severe consequences. Anemia is commonly observed in rheumatoid arthritis and is sometimes aggravated by nonsteroidal anti-inflammatory drugs, which may produce fluid retention or minor gastrointestinal blood loss in some patients. Therefore, patients with initial hemoglobin values of 10 g/dL or less who are to receive long-term therapy should have hemoglobin values determined frequently. **Infection:** In common with other anti-inflammatory drugs, Oruvail may mask the usual signs of infection. **Ophthalmology:** Blurred and/or diminished vision has been reported with the use of ketoprofen and other nonsteroidal anti-inflammatory drugs. If such symptoms develop this drug should be discontinued and an ophthalmologic examination performed; ophthalmic examination should be carried out at periodic intervals in any patient receiving this drug for an extended period of time. **Drug Interactions: Methotrexate:** The concomitant administration of ketoprofen and high-dose methotrexate has been associated with prolonged and marked enhancement of serum methotrexate levels resulting in severe methotrexate toxicity. This may also apply to some other nonsteroidal anti-inflammatory drugs. There were no abnormalities in methotrexate kinetics or evidence of toxicity when ketoprofen was given at least 12 hours after completion of high-dose methotrexate infusion. ORUVAIL should not be used in patients receiving high dose methotrexate. The potential for severe toxicity should be kept in mind when prescribing ketoprofen and low-dose methotrexate concurrently. ORUVAIL should not be administered within 12 hours of methotrexate infusion. **Acetylsalicylic acid (ASA):** concurrent administration of ASA decreased ketoprofen protein binding and increased its plasma clearance. The overall result was a 40% reduction in the AUC of ketoprofen. **Oral anticoagulants:** Ketoprofen has been shown to depress platelet aggregation and it can prolong bleeding time by approximately 3 to 4 minutes from baseline values. However, a study conducted in twenty patients undergoing therapy with coumarin and simultaneously receiving ketoprofen, failed to demonstrate potentiation of anticoagulant effect. Nevertheless, close monitoring of patients is recommended when Oruvail is given concomitantly with anticoagulants. **Diuretics:** hydrochlorothiazide, given concomitantly with ketoprofen, produces a reduction in urinary potassium and chloride excretion compared to hydrochlorothiazide alone. Patients taking diuretics are at greater risk of developing renal failure secondary to a decrease in renal blood flow caused by prostaglandin inhibition. **Antacids:** concomitant administration of magnesium hydroxide and aluminum hydroxide does not interfere with the rate or extent of the absorption of ketoprofen. **Lithium:** nonsteroidal anti-inflammatory agents have been reported to increase steady-state plasma lithium levels. It is recommended that plasma lithium levels be monitored when Oruvail is coadministered with lithium. **Probenecid:** concurrent administration of probenecid increases both free and bound ketoprofen through reducing the plasma clearance of ketoprofen to about one-third as well as decreasing its protein binding. ORUVAIL is not recommended in association with probenecid. Ketoprofen is extensively (99%) protein bound to human serum albumin and may compete for binding sites with drugs such as sulfonamides, oral hypoglycemic agents, phenytoin or lithium. Although no significant interaction has been documented, patients with such combination therapy should be monitored. **Clinical Laboratory Test:** The presence of ketoprofen and its metabolites in urine has been shown to interfere with certain tests which are used to detect albumin, bile salts, 17-ketosteroids or 17-hydroxycorticosteroids in urine and which rely upon acid precipitation as an end point or upon colour reactions for carbonyl groups. No interference was seen in the tests for proteinuria using Albustix, Hema-Combistix or Labstix Reagent Strips. Ketoprofen decreases platelet adhesion and aggregation. Therefore, it can prolong bleeding time by approximately 3 to 4 minutes from baseline values. There is no significant change in platelet count, prothrombin time, partial thromboplastin time, or thrombin time.

ADVERSE REACTIONS: Gastrointestinal: Gastrointestinal effects were the most frequently observed adverse reactions and were seen in approximately 13% of patients receiving Oruvail (ketoprofen). Ulceration and gastrointestinal bleeding have been observed in a few patients receiving Oruvail therapy (approximately 0.3%). Other adverse reactions in order of decreasing frequency were: gastrointestinal pain, dyspepsia, constipation, nausea and/or vomiting, diarrhea and flatulence. Such symptoms led to the discontinuation of treatment in 6.8% of patients. **Central Nervous System:** Central nervous system adverse reactions were next in frequency and included headache, fatigue, drowsiness, dizziness, depression, restlessness and nightmares. **Skin:** rash, eczema, flushing, pruritus, sweating and loss of hair were occasionally observed. **Allergic:** These were seen infrequently and included urticaria, angioedema and asthma. **Cardiovascular:** Mild peripheral edema, palpitation, bruising, arrhythmia, chest pain and exacerbation of circulatory disturbances were reported. **Auditory:** Tinnitus and deafness were reported on rare occasions. **Mouth:** The following symptoms were reported: dry mouth, mouth ulcers, sore tongue and inflammation of the mouth and gums. **Laboratory tests:** Abnormal alkaline phosphatase lactic dehydrogenase, glutamic oxaloacetic transaminase and blood urea nitrogen values were found in some patients receiving ketoprofen therapy. The abnormalities did not lead to discontinuation of treatment and, in some cases, returned to normal despite continuation of the drug. There have been sporadic reports of decreased hematocrit and hemoglobin values without progressive deterioration on prolonged administration of the drug.

SYMPTOMS AND TREATMENT OF OVERDOSE: Symptoms: Of 20 cases of overdosage (up to 5,000 mg) reported in Great Britain (5 children, 14 adolescents or young adults, and 1 elderly), only 4 had mild symptoms (vomiting in 3, drowsiness in 1 child). **Treatment:** Administer gastric lavage or an emetic and treat symptomatically: compensate for dehydration, monitor urinary excretion and correct acidosis if present. The drug is dialyzable; therefore, hemodialysis may be useful to remove circulating drug and to assist in case of renal failure.

DOSAGE AND ADMINISTRATION: Adults: The usual dosage is 150 to 200 mg once daily. The capsules should be taken with food and can be administered in the morning or evening. **Elderly and debilitated patients:** The dosage should be reduced in patients with impaired renal function and the elderly. The lower strength should be used in those cases. **Children:** Oruvail is not indicated in children under 12 years of age because clinical experience in this age group is insufficient.

Composition: Non medicinal ingredients: colloidal silicone dioxide, ethyl cellulose, gelatin, maize starch, shellac, sucrose, talc. Colouring agents: ORUVAIL 150 mg: erythrosine, titanium dioxide. ORUVAIL 200 mg: brilliant blue, erythrosine, titanium dioxide.

AVAILABILITY: Oruvail 150 capsules: each transparent pink capsule with opaque white cap (each half printed "Oruvail 150" in black) contains ketoprofen 150 mg as white pellets. Available in bottles of 100 and 250. **Oruvail 200 capsules:** each transparent pink capsule with opaque blue cap (each half printed "Oruvail 200" in yellow) contains ketoprofen 200 mg as white pellets. Available in bottles of 100 and 250.

REFERENCES:

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P One a day Oruvail® 150

Sustained-release ketoprofen capsules 150 mg

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