THE NOVA SCOTIA MEDICAL BULLETIN

SYMPOSIUM ON ORTHOPAEDIC SURGERY AND TRAUMATOLOGY

Coordinating Editor: Dr. A. J. Buhr

Scientific Writing: "I Mean What I Say"

Since the first six years of school are spent in learning to read and write, it is strange that the majority of adults do both so badly. Remedial reading courses and those intended to improve reading speed and understanding are recent developments for the treatment of reading defects, but little attention has been paid in institutions of higher learning to improving the quality of writing. Consequently, many physicians with something to report in writing are unable to express their thoughts on paper, so that the thought dies stillborn, lost forever. Those with stronger motivation may succeed in putting something down on paper, but often this is only a muddled jumble of words that fails to convey the writer's true meaning, or — worse still — leads others to believe that he holds views different from or even opposite to those he has tried to express.

But the act of writing is a creative art, and men feel strongly protective toward anything that they have created: surprise, pain, shock, anger and hostility appear in turn when the product of their pen is mutilated and torn apart by the editor, and the conflict is often resolved by a firm determination never to produce another brain-child that can be so mistreated. Finally, the editor plays his ace: the article appears in print, and the writer immediately recognizes it as his own thoughts clearly expressed. He graciously accepts the congratulations of his colleagues, ascends to the status of a scientific writer, and modestly circulates reprints on every possible occasion. Now he has a reputation to uphold, and when next smitten by the urge to write, will do so more carefully, more clearly and with more pleasure in his achievement than before.

These first faltering steps in the art of clear lucid expression teach a valuable lesson: "The act of writing externalizes thought; captures what was previously elusive, pins surging thoughts on paper so they can be examined. In the cold light of subsequent reflection, what a man captures may turn out to be moonshine but at least, having written it down and reflected on it, he will know it for moonshine."[1] Critical examination of what one has written leads to the search for clearer expression, refining the ore until pure gold remains. Practised continually, it becomes a habit that reflects the quality of the thoughts, which emerge in a more concise and logical form.

Since scientific advance depends upon logical and painstaking examination of hypotheses, the university-trained graduate could be expected to excel in the art of writing — but, as Woodford and his colleagues of the Council of Biology Editors have noted: "Scientific writing is heavy, verbose, pretentious, and dull." The reasons why "these highly educated and intelligent men and women should express themselves so obscurely, so wordily, and therefore so ineffectually" were stated to be complex, although "one contributing factor seems clear: few universities provide formal training in scientific writing, and few even encourage their students to develop a rational technique for writing scientific papers. . ."[2]

One example of the effectiveness of a technique of encouragement is that set by the Department of Medical Publications of The Hospital for Sick Children in Toronto. In a recent issue of the *Canadian Medical Association Journal*, Dr. Reid reported the results of a workshop of two weekend sessions attended by 60 participants selected from the residents, fellows and staff members of that hospital.[3] Each participant had the material for a paper he wished to write, and at least one page of his first draft was critically examined and rewritten with the help of his fellow writers. The majority felt that they had been helped to a clearer understanding of the requirements for publishing a good paper.

In the Province of Nova Scotia, there is no lack of material to report but a great reluctance on the part of physicians to publish their observations. The *Nova Scotia Medical Bulletin* provides the doctors of this province with a medium through which they can communicate their observations to their colleagues both here and throughout the world. They should know that the Editorial Board of The Bulletin is always ready to offer advice and encouragement to the budding scientific writer, but this may not be enough. Like many other committees, the Editorial Board is always relearning its job, and needs professional assistance which, until recently, was lacking.

Now, however, Dalhousie University has established an Editorial Service, under the capable direction of Miss U. F. Matthews, whose experienced hand has recently guided the Proceedings of the Dalhousie Centennial Symposia, to publication.[4] Although the Editorial Service is aimed primarily at influencing the quality and quantity of writing within the Faculty of Medicine, the extension of the university into the area of continued medical education makes it logical to offer this service to all medical practitioners in the province. In addition to assisting in the review of all papers submitted for publication in The Bulletin, the Editorial Service will assist any practitioner with a case report, or observations he may wish to publish or present at a meeting, by pointing out areas where the draft manuscript is not clear or where additional information is required.

Who knows whether he is a budding medical writer until he tries the art? For all you know, your pen may be mightier than the stethescope or scalpel especially when sharpened by the talents of those who can help you to say what you mean, when you know what you want to say. We need your contributions — won't you please try?

I.E.P. □

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Presidential Address

F. A. Dunsworth, M.D.
Halifax, N. S.

The annual address to the Medical Society of Nova Scotia permits the retiring president to draw on his experience in his office, reflect on the happenings of his year in office and with or without alarm view the future.

In the name of continuity the office of President of your Society contributes in an extensive way to major decisions for a three year period. In addition I was a member of the Physicians Services Insurance Committee for an additional two years before it was reorganized: hence my experience covers the past four years. Thus I offer the following brief personal comments that are meant to stimulate action, not to produce gloom.

Medical Economics

Basically we physicians are naive. Many years ago we went on record as favoring pre-paid medical insurance but we obviously did not reckon with the universal comprehensive criteria which were incorporated into the Federal Medicare legislation. We should have anticipated and prepared ourselves for what we have been experiencing in the past few years.

I suggest we should seriously consider the establishment of a group within the Society whose task would be to think in economic and especially political terms and thus be able to anticipate the potentional conflicts between medical and political aims. Only one of the many items would be the intensive study of the Hall Commission[1] reports as I'm sure we've not heard the last of its recommendations.

The economic aspects of Medicine are staggering when one realizes that between 5.3% and 5.7% in the early 1960's of the Gross National Product of Canada was spent in health care[2], a figure that continues to increase.

The costs of hospitalization are sky-rocketing, as are the costs of drugs and with a universal medicare scheme physicians' fees will be clearly shown as a drain on the public purse.

Time only permits brief discussion of the latter item but it must be realized that with increasing demands for medical services, physician's incomes, already the highest by the Department of National Revenue standards, will further increase. It will be relatively easier to limit physicians incomes, much easier than hospital costs and even drug costs.

Every resident of Canada realizes that increased costs can be met only by taxes. The money required for Health care will be competing with the needs for other public expenditures and thus the amounts expended for physicians services will be closely scrutinized, compared to other incomes not only by contrast with professional incomes but also in relation to regional disparities in income.

Not only will the question be raised, "can this province afford it?" but the next question will be "are the funds paid out for medical services worth it? What is the calibre of the medical services given to the public?"

We may permit our standards of medical care to deteriorate. This may appear in many guises, most of them synergistic. The physician, already over-working by average employment standards, may attempt to cope with increasing demands for medical services by working even longer hours. His scientific acumen will likely suffer but even worse, physically and mentally he will be limited in how long he can carry on a satisfactory level of medical practice. My fear is that such an existence will not be attractive in the recruitment of the best young people into medicine.

Another pattern may be for the physician to decrease the time available for each patient. A sort of "assembly-line" medicine will appear and there will not be the time available to establish the rapport so necessary to a good doctor-patient relationship. There will be little time to reflect on the medical problems with which we are faced and a mechanistic routine may appear. The Art of medicine and the Science of medicine will suffer, but especially the Person of the doctor, such an essential factor in the treatment and amelioration of the multiple psychological and psychosomatic afflictions of the public may be lost. Instead of the interested and obviously well-trained physician we may witness a greatly increased referral rate and the over-utilization of chemical, laboratory and investigative procedures. The dehumanizing of medicine, unfortunately already so prevalent, will tend to increase and the status of the family practitioner may be further diminished.

To return to my earlier comments, with the necessary demands for other public services, in conflict with medical services insurance costs plus lowered standards of care that we have permitted to appear, we can expect increased criticism of physicians' fees and thus the cycle worsens!

Changes in Patterns of Remuneration

The next cause for apprehension has to do with changes appearing in patterns of remuneration for medical services.

Medicine is probably splitting on what most of us think is the fundamental method of payment. On one side we see the pattern of fee–for–service which in the history of medicine has only emerged in the treatment of the developing middle class and the entrepreneurs since the Industrial Revolution.

On the other side is the salaried approach with its many modifications. Historically payment for medical services was not a fee–for–service, physician-patient contract but the physician, who often did not have a high socio-economic status, was retained to offer medical services to a given group. In 19th Century Europe and Britain a pattern developed to cover many industrial organizations by a system of a yearly salary or of "panels." More recently in this century medical schools have developed an increasing number of full time appointments and this pattern has spread to medical clinics and the active medical staffs of many hospitals.

It is in these latter groups that the statement "the medical profession is putting itself on a salary basis" seems to be emerging. The future implications for the practice of medicine because of the change in methods of remuneration may be very serious indeed and will need careful evaluation.

Some Suggested Principles for Our Profession

1. The first principle is that we must maintain and improve our standards of medical care.

   This principle has broad implications. Starting from the recruitment of bright and well motivated young people, it spreads into the best of medical education and into the highest possible standards of medical practice and continuing medical education. It infers the profession as a group will constantly re-examine its own standards and the practices of fellow colleagues and be prepared to offer the leadership required.
2. Thus we reach the second principle, that a responsible profession must exercise self-discipline. This involves self–discipline in all respects, professional competence, the setting of ethical, legal and moral standards and the cooperative liaison with fiscal bodies in encouraging financial propriety of its members. I have elaborated on these in my recent addresses to the Branch Societies.
3. The third principle is that the profession will be the guiding agency for its members. At times it will instruct, at times it will protect but at all times will it exercise its responsible role for its professional members.

   At times the role of protection may seem to be in conflict with the role of self-discipline but I submit that our Society cannot be "responsible" unless it fulfills *many* roles.
4. The final principle is that the medical profession is entitled to set responsible fee schedules like any other responsible profession.

   It was this principle that was more fully elaborated at the Special Council and Special General Meeting in March 1969 just prior to the introduction of M.S.I. (Medical Services Insurance-Nova Scotia) when the contentious Presidential letter No. 9[3] was presented to the profession of this province and does not need repetition at this time.

   Like any definitive and abbreviated statement, sections or phrases could be taken out of context and produce adverse reactions. Only the future can judge if the assessments and prognostications were correct.

It is impossible for me to thank so many who have laboured for the good of the medical profession in this province during my term in office but I must mention some by name, with apologies for those I miss.

For Doug. Peacocke our Executive Secretary for service above and beyond the call of duty.

For my fellow members of the Medical Care Insurance Committee, George Saunders, Art. Sutherland and Lea Steeves for their active involvement in our many many deliberations and encounters on your behalf.

For the officers and executive of the Society for their support and encouragement.

For Brian O'Brien and Peter O'Brien and the Public Relations Committee for their counsel and clarification in the field of communications and last, but far from least, to my own public relations agent, my wife Frances, who constantly gave me such support in such trying times and kept my long-suffering, and neglected family together!

Finally, as your president and president-elect I've visited all but one of the Branches, and many I've visited four or five times. I've been greatly impressed by the calibre of our membership and I will always treasure the honour that you gave me by electing me your president. □

References

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Lea C. Steeves, M.D.

President
1969-1970

The Medical Society of Nova Scotia

Lea Chapman Steeves, B.A., M.D., C.M., officially took office in November as President of the Medical Society of Nova Scotia for 1969-1970.

Well known to doctors, medical students and educators throughout the province, Dr. Steeves has served for the past year as the Society's President Elect during a time when significant and far reaching changes in the application and administration of medicine were underway.

A New Westminster, B.C. native son, Dr. Steeves is a graduate of Mount Allison University, Sackville, N.B., and holds a diploma in Medicine from Montreal's McGill University.

During World War II Dr. Steeves served as Surgeon-Lieutenant, R.C.N., V.R., in Halifax, N. S., St. John's, Nfld., and St. Hyacinthe, Que. His current appointments include: Associate Dean, Faculty of Medicine, Dalhousie University; Director, Division of Continuing Medical Education, Dalhousie University; Associate Attending Physician, Victoria General Hospital, Halifax; Consultant to the Halifax Infirmary on Medical Education; Medical Specialist at Halifax's Camp Hill Hospital; Consultant to the Halifax Children's Hospital; President (1969-1970), the Medical Council of Canada, and the 1968-1969 Presidency of the Canadian Association for Continuing Medical Education.

Dr. Steeves is also a member of the Halifax Medical Society, the Nova Scotia Society of Internal Medicine, the Nova Scotia Cardiovascular Society, the Canadian Cardiovascular Society, a Fellow and Council Member of the Royal College of Physicians (Canada), a Fellow and Atlantic Provincial Governor of the American College of Physicians, and Vice President for Canada of the Phi Rho Sigma Medical Fraternity.

He is an Elder of the Fort Massey United Church and a member of the Advisory Boards to both the Canadian and Nova Scotia Heart Foundations.

He is married to the former Katherine Grace Fraser — like him, a Mount Allison graduate — and has five sons: Donald Chapman Steeves, M.D.; Alexander Lea, Medicine, Dalhousie University, Class of '71; Gordon Fraser and John MacFarlane, Mount Allison University, Classes of '71 and '70 respectively, and James Ian, a student at Queen Elizabeth High School, Halifax.

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o our guests and exhibitors the Medical Society of Nova Scotia says "Thank you, for being with us."

SHOCK
Fundamental Aspects

A. S. MacDonald, M.D., F.R.C.S. (C.).*
Halifax, N. S.

Definition

As defined by Simeone[1] shock is a "clinical condition characterized by signs and symptoms which arise when the cardiac output is insufficient to fill the arterial tree with blood under sufficient pressure to provide organs and tissues with adequate blood flow."

A working classification of shock is presented in Table I as adapted from Shires and Carrico.[2]

TABLE I
CLASSIFICATION OF SHOCK

I. CARDIOGENIC
- 1. Myocardial infarction
- 2. Serious cardiac arrhythmias
- 3. Miscellaneous
  - a–tension pneumothorax
  - b–mediastinal shift
  - c–vena-caval obstruction
  - d–cardiac tamponade

II. HYPOVOLEMIC
- 1. Blood loss
  - a–external
  - b–internal
- 2. Plasma loss
  - a–external, as in burns
  - b–internal, as in pertonitis
  - c–crushing injury
- 3. Water loss
  - a–water deprivation
  - b–water loss, as in intestinal, biliary or pancreatic fistulae, vomiting and diarrhoea

III. CHANGES IN RESISTANCE VESSELS
- 1. Decrease in resistance
  - a–spinal paralysis (anaesthetics, trauma, etc.)
  - b–neurogenic reflexes as in acute gastric dilatation.
  - c–end stages of hypovolemic shock
- 2. Septic Shock
  - a–change in peripheral resistance
  - b–change in venous capacitance

Pathophysiology of Shock

The pathophysiology of haemorrhagic shock is illustrative of the complexities of the shock state in man. The majority of the investigations on shock have been made in animals, particularly the dog, supplemented by clinical observations in man and a few experiments involving bleeding to preshock levels in human volunteers. It has become increasingly obvious, though there are a few species with specific differences, that by and large the reactions of the dog to shock parallel the human situation.

In the steady state in man the cardiovascular system is controlled by the innate activity of the heart as pump and the autonomic activity of the vasomotor center which varies the resistance of the arterial vasculature, the heart rate, and the capacity of the venous system to maintain the blood supply of the brain within relatively narrow ranges of blood pressure and flow.

There are only two organs which cannot incur oxygen debts, the heart and the brain. It would appear that the sum total of the body's defense mechanisms are directed towards preservation of adequate perfusion of these two organs.

Loss of blood, as in haemorrhagic trauma, results in a drop in blood pressure which is immediately corrected by a number of reflexes.

The drop in pressure results in a diminution of the discharges from the baroreceptors in the right and left atria, aortic arch and carotid sinus. The baroreceptors send afferents to the vasomotor inhibitory center carrying impulses at a continuous slow rate inhibiting the vasomotor pressor center. With a fall in pressure these discharges from the baroreceptors diminish or cease, and the released vasomotor pressor center in the brain stem causes a widespread vasoconstriction involving all the arteries and veins of the body, except those of the brain, which do not change, and those of the heart which actually dilate.

Simultaneously, the vasomotor center sends impulses to the hypothalamus resulting in ACTH release, and impulses to the adrenal medulla with resultant outpouring of adrenaline and nor-adrenaline.

Vagus inhibition of the heart rate is suppressed and the sympathetics stimulate the heart directly resulting in tachycardia.

The venoconstriction results in decreased venous capacity (the venous system contains 70% of the blood volume) with increased venous return, thus shifting blood into the arterial side of the circulation.

The spleen contracts and a small amount of blood is discharged into the circulation in this fashion.

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The degree of vasoconstriction which occurs is considerable, particularly in the kidney. For example, in one experiment, decreasing the cardiac output by 70% decreased renal blood flow by 91%[3], and in the splanchnic area and skin, constriction is severe.

Sympathetic discharge from the vasomotor center results in constriction in muscle vessels as well, though this effect is counteracted to some extent by circulating epinephrine, which though in general a vasoconstrictor causes vasodilatation in muscle.

The adrenal medullary hormones have chronotropic and inotropic effects on the heart, increasing its stroke volume and rate.

The ACTH outpouring results in adrenal-cortical release of cortisol which also has chronotropic cardiac effects.

The renal vasoconstriction causes release of renin from the juxtaglomerular apparatus with production of angiotension, a potent vasoconstrictor and cardiac stimulant.

Angiotensin II also stimulates adrenal cortical release of aldosterone.

The vasomotor center apparently is responsible for the release of ADH.

All these above mechanisms initially maintain a normal blood pressure and perfusion pressure in the heart and the brain, though sacrificing perfusion in the rest of the body.

Neuronal Effects of Shock

The importance of this maintenance of cerebral perfusion is emphasized by the extreme vulnerability of neuronal tissue to anoxia and to departures from an optimal chemical environment. A continuous and adequate renewal of glucose and oxygen is provided under steady state conditions by a relatively enormous blood flow, approximately 55 ml/100 gm/ minute. An interruption of these nutrient supplies rapidly leads to functional impairment and irreversible change. Thus, a complete cessation of cerebral circulation results in loss of consciousness within ten seconds, the approximate time interval required to exhaust the estimated stores of oxygen within the brain. Syncope occurs when cerebral blood flow falls to approximately 30 ml/100 gm/ minute, regardless of the mean arterial pressure.[4] As haemorrhage becomes more severe the cerebral circulation receives a greater and greater percentage of the cardiac output.

The neuronal responses show progressive deterioration and disappear at arterial pressures of 30 mm.Hg., a hydrostatic pressure that approximates to the critical opening and closing pressure for small blood vessels. At this point sudden cardiovascular collapse or ventricular fibrillation often occurs.

The cardioinhibitory portion of the vasomotor center is more resistant to shock than the vasopressor center whose function deteriorates early. It is the vagus stimulation resulting from the preterminal excitation of the inhibitory centre which results in cardiac arrest. This occurs even if the heart is separately perfused in an extracorporeal circulation by normally oxygenated blood[5].

Cardiac Effects of Shock

There is good experimental and clinical evidence to explain the apparent resistance of the heart to anoxia. In the irreversible hemorrhagic shock model in the dog there is no evidence of a significant shift to anaerobiosis, despite a marked fall in oxygen content and saturation.[6] The decreased coronary vascular resistance during shock fails to compensate the lowering of the perfusion pressure completely. In this sense, then, the oygen supply of the heart during shock is relatively deficient. If the shock state is associated with a considerable decline in arterial pressure or oxygen saturation or both, the coronary venous oxygen tension may approach the critical pO_2 level of 10 mm Hg.

In addition, experiments have suggested that some unknown substance arising in the peripheral circulation during shock may directly damage the myocardium. For example, if the brain and heart are protected by extra corporeal circulation while the rest of the animal is first shocked, then retransfused, then the cardiovascular integrity restored, the heart cannot handle massive loading infusions that it normally can. The demonstration that digitalization *prolongs* the survival of dogs subjected to *protracted* haemorrhagic shock, and that such therapy also improves the function of the non-failing heart, provides a sound reason for administering rapidly acting cardiac glycosides to the patient in shock.

The Micro Circulation in Shock

Since the brain is protected, at least initially, and the heart is relatively resistant to the effects of shock, then the profound disturbances of the syndrome of shock, particularly as measured by changes in the blood chemistry, must occur in the tissues of the remainder of the body. If an area of the mesentery is studied under the microscope in a haemorrhaging dog, arteriolar constriction followed by constriction of sphincters, first in precapillary sphincters, then in metarteriolar and arteriolar sphincters is seen as blood volume loss progresses.[7]

At 30% volume loss capillary flow decreases due to progressive closure of sphincters. When sphincter constriction is complete flow in capillaries ceases.

At 50 - 90% volume loss, arterioles begin to return to original calibre, venules increase in size, and arteriolar and venular flow becomes non-existant, with marked sludging. Capillary calibre remains static, and pooling and trapping are extreme, leading to clotting. (This occurs at 40% volume loss if vasoconstrictors, e.g., nor-epiniphrine, are

given.) Re-transfusion at this stage aggravates the trapping and pooling.[8]

Metabolic Effects of Shock

The effects of lack of sustained flow at the capillary level are profound. The hypoxic state shifts metabolism to the anaerobic phase, utilizing the Embden-Myerhoff pathway for glycolysis instead of the normally functioning Krebs cycle. Although pyruvate is formed, in the absence of oxygen, oxidative decarboxylation to acetic acid (Acetyl CO-) cannot take place, and rather than enter the citric acid cycle, pyruvate is reduced to lactate by lactate dehydrogenase, an enzyme present in almost all cells. The Embden-Myerhoff reactions occur in the cytoplasm whereas the citric acid cycle occurs in the mitochondria. This anaerobic glycolysis results in only four high energy bonds (30,400 calories), whereas, with oxygen present and the Krebs cycle working, 38 high energy bonds are formed, (288,800 calories from the potential of 686,000 cal/mole glucose).

Pyruvic and lactic acids rapidly accumulate in the blood causing an acidosis. The liver can metabolize lactic acid in the normal state via the lactate-propanediol pathway, but its capacity is limited and is also energy dependent – and energy supplies are at a premium in the shocked state.

During this phase marked swelling of the mitochondria and interstitial oedema occur. These severe changes appear to be reversible if not allowed to continue until mitochondrial disruption occurs.

Self-potentiation is a distinguishing feature of hypoxic circulatory states. In clinical or experimental shock this presumably contributes to the established tendency towards irreversibility of shock regardless of cause. Self-aggravating mechanisms at the cellular or tissue level may play a sustaining role in the events of hypoxia due to circulatory disorders. There is inherent in many normal physiological mechanisms a basic instability that may, given the proper set of circumstances, provide a cycle of self-aggravaging events to the total detriment or even destruction of the organism. Homeostatic physiologic mechanisms may be regarded as negative feed-back systems, while virtually all pathological process are positive feed-back systems. Illustrating this is Table II. A clinical example of cellular swelling giving increased capillary resistance, decreased flow and hypoxia would be the anterior tibial compartment syndrome.

TABLE II

ANAEROBIC METAB ➤ ACIDOSIS
↑ ↓
HYPOXIA DECREASED O_2 CONSUMPT.
↑ CELLULAR SWELLING ↓
DECREASED CAP. FLOW ◄ INCREASED CAPILLARY RESIST.

Fluid Shifts in Shock

It has recently become apparent that calculation of interstitial fluid volumes following replacement of total blood and plasma loss, plus urinary excretion, and the amounts calculated for sweat and exhalation, still leave a significant interstitial fluid deficit amounting to as much as 25% of the total extra cellular fluid. It is now postulated that this fluid goes into the cells as a result of hypoxia.[9] In a haemorrhaging man, as blood is lost and the hydrostatic pressure falls, there is a decrease in the forces tending to push fluid out at the arterial end of the capillary, but no corresponding decrease in the forces (tissue pressure, osmotic pressure of the intracapillary fluid) drawing water into the capillary, thus the overall flow is from interstitial fluid to vascular compartment. It should be pointed out here that estimates of fluid loss are usually inaccurately low. For example, during laparotomy the insensible loss due to evaporation alone amounts to 150 to 200 mls./hr.[10]

At any rate, in haemorrhagic shock, one must estimate that replacement requirements will be the amount of blood lost, plus insensible losses, plus that amount which has shifted into the cells during the hypoxic period - i.e., for every 500 cc. blood lost approximately 500 cc of a balanced salt solution, such as Ringer's lactate, in addition to 500 cc. of blood must be given.

The Intestine in Shock

The effects of hypoxia in the dog in severe hemorrhagic shock, produce extensive haemorrhagic necrosis of the small bowel. It was postulated that this ischemic necrosis resulted in loss of the integrity of the bowel mucosa, with resultant absorption of toxic products from the bowel. However, in animals raised in a germ-free environment small bowel necrosis still occurred. Further, it was found that one could protect the mucosa if the pancreatic duct was tied off, or if the bowel was thoroughly irrigated with saline prior to shock, or if a trypsin inhibiting agent such as Trasylol(R) was instilled into the bowel. It is now obvious that the necrosis of the mucosa is due to the loss of the individual cell's defensive abilities during hypoxia, and its inability to prevent trypsin digestion of the cell walls. It has been demonstrated that intestinal flora bestows a protective effect against haemorrhagic necrosis occurring in the colon, and the lesions are thus more severe in germ-free animals.

It has been demonstrated that the products released by damaged bowel, such as histamine, serotonin and lysozymal enzymes (acid phosphatases, B-glucuronidases, cathepsins) are detoxified to some extent in the liver.

The Pancreas

Trypsin release in other clinical conditions has other important features as regards shock. For example, in the shock that accompanies pancreatitis, the loss of blood through haemorrhage and the ex-

tensive ascites that occurs furthering fluid losses, is complicated by the presence of circulating vasodilator substances, i.e., the kinins. As indicated in Table III these are vasoactive peptides and their liberation can produce a profound shock.

In a normal patient the kallidogen portion of α_4 globulins measures 2 - 7 Frei units. In pancreatitis it may be absent or markedly reduced indicating its breakdown to kallikrein.

Proteolytic enzymes have other major effects particularly on blood coagulation in that the plasminogen-plasmin system is activated resulting in excess fibrinolysis.

TABLE III

KALLIKREINOGEN (inactive plasma form)

|

proteolytic enzymes
lowered pH

KALLIKREIN (an enzyme)

KALLIDINOGEN (α_2 globulin) → KALLIDIN 10 (a vasoactive peptide)

|

aminopeptidase

KALLIDIN 9 (active)

carboxypeptidase

Inactive octapeptide + arginine

Blood Changes

The situation in endotoxic shock resembles that of pancreatitis changes to some extent. Following endotoxin injection there is initially a marked fall in platelets which virtually disappear from the circulation, an occurrence reminiscent of the Schwartzman reaction. These reappear a short time later and it has been demonstrated that they accumulate at sites of apparent damage along the capillary endothelium.

A slight prolongation of the thrombin time follows, suggestive of release of heparin or a heparinoid material. This lasts about 15 min. This is followed for the next 40 minutes by a hypercoagulable state with greatly shortened thrombin generation time indicating consumption of the clotting factors, which do indeed diminish. Factors II, V, VII, VIII, IX, XI, XII all decrease. At the same time there is marked plasminogen activator activity which apparently prevents thrombi formation. After two hours the blood is hypocoagulable.

The blood becomes hypercoagulable also during haemorrhagic shock. There is increased platelet adhesiveness, probably due to release of 5-hydroxytryptamine and adenosinediphosphate during severe hypotension, because it can be largely prevented with 1-methyl-D-lysergic acid (M) - butanolamide bimaleate, a 5-hydroxytryptamine antagonist. There is a measurable shift of lipid to the red cells. The non-esterified fatty acid concentration in the plasma is greatly increased due to the high levels of circulating epinephrine which mobilize this material. It is probable that NEFA is the major energy source for cardiac muscle during this period. The NEFA probably contributes to the change in surface charge on the red blood cells as measured by electrophoretic mobility.

Effects of Shock on the Lungs

The platelet and red cell aggregates are filtered in the lungs. It has been suggested that this may be responsible for the marked alterations in function, and the pathological changes that occur in the lung during haemorrhagic shock.

There is an increase in physiological dead space, an increase in the arterial-alveolar CO_2 gradient, and an increase in resistance to inflation.

Such alterations may only reflect a diminished pulmonary blood flow and vasoconstriction. Use of a respirator does not seem to prevent these changes although the pH and pCO_2 of arterial blood may be favourably influenced.

Pathological examination reveals oedema, extravasation of blood, and in the more advanced cases, liver-like areas of haemorrhage and atelectases in the lung. Leucocyte infiltration in and around blood vessels is notable.

Despite the extensive damage and the measurable deterioration in function, lung function is probably adequate until the final stages of shock, although the severe respiratory effort required of the extrinsic respiratory muscles and diaphragm, under hypoxic conditions may so aggravate existing acidosis and oxygen debt, that respiratory failure in this sense may intervene.

Effects of Acidosis

The increase in serum lactic acid is the major cause of increased H- ion concentration in shock, and in fact, this has some prognostic value. Huckabee has found that lactate excess above a certain level is associated with 100% mortality.

The Kidney in Shock

The renal blood flow falls from approximately 1200 cc/min or about 25% of the cardiac output to 50% of normal within minutes of a major haemorrhage, and urine production correspondingly diminishes or ceases. The blood flow through the kidney is curtailed even more than indicated by the blood pressure because of a more severe vasoconstriction than in most other parts of the body. Glomerular filtration rate falls to zero at blood pressures of 65 mm. Hg. or less. Since there is still some blood flowing through the kidney this suggests the opening of arterio-venous shunts.

Probably the most common precipitating factor in acute renal failure is extensive surgery. Preoperative fasting and purgation result in dehydration, to which is added premedication and anaesthesia, which also depress renal blood flow. Surgical trauma alone results in vasoconstriction, and the

attending blood and fluid loss, and sequestration oedema, further diminish kidney perfusion. In several series the operation of resection of abdominal aortic aneurysm has been particularly potent in this regard. Abruptio-placenta and post-partem haemorrhage from all causes are also high on the list. Trauma, of course, is increasingly important as a cause of acute renal failure.

The human kidney can tolerate complete ischemia probably for no longer than 90 minutes without developing acute failure. With increasing frequency, especially as treatment is often instituted more rapidly, an intermediate stage of renal failure has been recognized.[11]

It has been given the unfortunate name of "high output renal failure," thus confusing it with the condition occurring in chronic kidney disease.

Moderate ischemia may result in cloudy swelling of distal tubular cells, and occasional proximal tubular cells, with concomitant partial interference with tubular function. The clinical effect is that of a loss of ability to concentrate urine, such that large amounts of dilute urine of low, though not fixed specific gravity, are excreted, in the face of a rising BUN and serum potassium. The kidney does retain a very limited ability to excrete sodium, potassium, and acid metabolites.

Clinically this condition is very easily overlooked, as the urinary output seems adequate. The very adequacy of the urine output is the diagnostic clue, however, as patients who are in shock, or who are dehydrated, should put out small quantities of high specific gravity urine. If this condition is unrecognized and surgery performed, acute renal failure is almost inevitable. The condition is ADH resistant and can be diagnosed by the pitressin test.

Complete recovery is fortunately the rule.

The effects of transfusion reactions are unusual. Urine flow stops, and measurement of PAH clearance becomes impracticable, yet the blood pressure and direct renal blood flow, except for a transient drop, remain unchanged. Thus, blood flows in and out of the kidney but performs little or no function, suggesting arterio-venous shunting.

Presumably direct tissue deposition is a most important site of plasma hemoglobin clearance, particularly in the renal cortical tubules. This deposition is the most likely cause of the renal damage that occurs. The action of myoglobin, from muscle breakdown, is probably similar.

Gastric Mucosa

Observations of patients dying from shock have revealed a remarkably high incidence of acute gastric ulcers, as high as 60% in some series.

Harjola and Sivula[12], in an experimental preparation in rabbits noted three kinds of changes: 1) gastric mucosa becomes pale in consequence of general vasconstriction; 2) several large diffuse patches appear and disappear completely after the restoration of blood volume; 3) small sharply defined spots appear at the beginning of blood letting. Their paleness is very intense. Hemorrhages ensue in these spots after restoration of blood volume.

The same areas always seem to be involved in repeated experiments. It has been suggested that these areas are in relation to the arterio-venous shunts which are present in large numbers in the stomach.

The Liver

The liver takes part in the generalized vasoconstrictor response to shock but the peculiarities of its blood supply result in some differences. The portal vein constricts to a greater degree than the hepatic artery, or the hepatic veins, in response to haemorrhage, or to infusions of adrenaline. The pathological lesions are characterized by congestion of the central hepatic veins and sinusoids, centrilobular necrosis with preservation of the reticulum framework in the necrotic zone, enlargement of the portal tracts by oedema, congestion, inflammatory reaction, and, not infrequently, portal haemorrhages. All these changes are found in patients dying of shock and in experimental animals the extent of the changes depends on the length and severity of the shock.

From experimental work it is probable that pooling of blood in the liver does not occur until late in shock, and in the early stages the congestion is reversible with treatment.

It has recently been demonstrated that surgical manipulation of the biliary tract, particularly crushing or stretching of the nerve plexus accompanying the hepatic artery, results in severe reflex vasoconstriction of the hepatic artery and also of the renal arteries. This latter effect is profound, resulting in marked diminution of renal blood flow and decrease or cessation of urine flow[13]. This effect can be prevented by infiltration of the hepatic nerve plexus or infiltration of the kidney nerve supply with local anaesthetics.

Endotoxic Shock

The most marked effect of endotoxic shock in man is on the kidney circulation. Blood containing endotoxin perfused into the kidney circulation causes a marked increase in renal resistance regardless of the central arterial pressure. This vasoconstriction can be prevented by incubating the mixture with ADP or aldosterone, or by the systemic injection of massive doses of cortisone. Sympathetic blockade or vasodilating agents are also effective.

This severe renal constriction is present even while the patient is peripherally dilated and is warm and dry at the skin level. It is my belief that in endotoxic shock, which is usually preceeded by a rigor and high temperature, that the warm flushed appearance of some patients is a central phenomenon due to stimulation of the vasomotor center by a pyrogenic agent, and the initial skin vasodilatation is a reflex to high temperature. One notes that this

warm shock is accompanied by oliguria and is soon followed by the classic shock picture of cold, clammy, mottled, pale, cyanotic extremities.

It should be brought out that the distinction between exo- and endotoxin is not precise. Crude lysates of gram positive staphlococci and streptococci have been shown to produce the physiological effects characteristic of gram negative shock, and clinically approximately one third of the cases of classically gram negative shock have blood cultures growing only gram positive organisms.

Generally, however, as shown by Kwaan, Bradley and Weil[14], gram positive infection is characterized by vasodilatation with lowered peripheral resistance, a mild drop in blood pressure and little interference with cardiac output. Urine flow is usually adequate, and metabolic derangements mild.

Gram negative shock is typically characterized by a marked increase in peripheral resistance, and a falling cardiac output and severe drop in blood pressure. Severe metabolic acidosis rapidly ensues.

The status of "pooling" as a significant factor in gram negative shock has not as yet been settled. Despite the severe splanchnic stagnation that occurs in the dog, measurements of blood volumes in man has not shown any great changes until the terminal stages of shock.

Kobold et al[15] found that when endotoxin was incubated with whole blood and blood fractions, histamine, serotonin and a vasoactive polypeptide-like substance were released. When endotoxin was incubated with tissue homogenates this also occurred and these substances could be isolated from the blood of animals in endotoxic shock. Removal of abdominal viscera delayed the release of histamine and the development of shock. This is not surprising, as the bowel, particularly the ileum has a high concentration of histamine.

The importance of this latter substance in septic shock is controversial, particularly as some of the features of histamine shock, notably the extreme bronchospasm, are absent.

Martin et al[16] have obtained the best results so far reported in treatment of experimental endotoxin shock with a regime which included a potent anti-histaminic agent, along with a vasodilator drug, fluid therapy and antiacid, antifribolysin and anticoagulant drugs.

Summary

The pathophysiology of shock and its effects on various organs has been discussed. The presently accepted theories and some of the controversial aspects have been mentioned. □

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PART II: THE CLINICAL MANAGEMENT OF SHOCK

Introduction

The clinical syndrome of shock, as encountered by the surgeon is seldom uncomplicated. Many factors combine to produce it, and it is most frequent in the elderly arteriosclerotic patient undergoing major surgery, where associated disease may be present in many systems. Unless all systems are supported, and all contributory factors treated, the condition may not respond. All too often, corrective measures are too few, and applied too late. Almost half of all the cases of shock encountered should have been anticipated, and the majority of these are preventable, notwithstanding the high mortality of shock in this group of patients.

In reviewing cases of shock, the commonest error was a failure to appreciate and correct the fluid deficit that may exist preoperatively in patients who are anorexic, anaemic or are suffering from weight loss, vomiting or diarrhoea. The second most common error was to give inadequate fluids in the postoperative period, and to give most of it in the form of glucose-water. There is no justification for restricting salt in the non-cardiac patient after operation; it should be given in the form of a balanced electrolyte solution such as lactated Ringer's Solution. The worst error is the inadequate evaluation of renal function both before and after operation. If the kidney cannot concentrate to a specific gravity of 1.020 or better in an overnight fasting specimen, major elective surgery must be postponed and a thorough investigation of renal function undertaken. Postoperatively, the measurement of specific gravity will reveal a high output failure syndrome, and measurement of urine volume will provide an early indication of impending shock. Lastly, the failure to detect chest complications following surgery, such as retained secretions or atalectasis, may allow arterial oxygen desaturation to lead to the development of shock.

Diagnosis

While the basic cause of shock is usually apparent, being either hypovolaemic, due to blood or fluid loss, cardiac, due to failure, infarction or arrythmia, or endotoxic, due to septicaemia, the diagnosis must include an assessment of the state of function of all the important body organs. It cannot be too strongly stressed that a thorough working knowledge of the underlying altered physiology is the key to the successful treatment of shock. This requires an initial assessment, through an accurate history and a thorough physical examination, paying special attention to the parameters listed in Table I., to provide a baseline. These parameters are recorded at frequent intervals to measure the response to treatment. In addition, many of the diagnostic tests listed in Table II. are frequently required to establish an accurate diagnosis, and may need to be repeated at intervals. These parameters and the results of the diagnostic tests must be evaluated as part of the total picture, since undue reliance placed on any one of them may lead to the wrong conclusion being drawn.

TABLE I
Parameters monitored in shock

Blood pressure
Pulse rate and character
Heart sounds and E.K.G.
Jugular venous pressure
Urinary output and specific gravity
Respiratory rate and depth
Chest signs including X-ray
Skin colour, evidence of vasoconstriction or sweating
Body temperature
Level of consciousness

TABLE II
Diagnostic Procedures in Shock

Central venous pressure
Hemoglobin and hematocrit
Serum electrolytes
Blood Urea nitrogen
Urinalysis
Blood gases and pH
Blood volume
Cardiac output
Pulmonary function studies
Blood lactate and pyruvate
Special determinations when indicated:
a. Blood, urine and stool cultures
b. Blood clotting factors
c. Serum calcium
d. Liver function tests

The Evaluation of Parameters

A fall in blood pressure has been traditionally synonymous with the term 'shock', but it must be remembered that patients may be in shock from a physiological point of view, with inadequate tissue perfusion, for hours or even days prior to the onset of severe hypotension. Again, a hypertensive patient may show 'normal' blood pressure levels and still be in shock, while, after treatment with vasodilators, some patients may run blood pressure levels as low as 70 mm Hg. systolic without any of the common manifestations of shock.

Although a rise in pulse rate commonly precedes hypotension in shock, bradycardia is common in the elderly patient with cardiogenic shock, and can also occur in hypovolaemic and septic shock. Pulse volume is commonly reduced in shock, but in some types of septic shock, with a high output state, the pulse may be full and bounding.

Auscultation of the heart may provide evidence of changes in cardiac murmurs, and when combined with the E.K.G. may confirm an arrythmia or the possible development of a myocardial infarction. However, S-T segment changes and inversion of the T wave are common in hypotension and do not necessarily indicate infarction.

The jugular venous pressure indicates the heart's ability to handle the venous return, and the volume of that return, but occasionally, the heart will fail precisely because of hypovolaemia or inadequate haemoglobin content of the blood, and the venous pressure may be falsely elevated. In septic shock, patients may show an elevated venous pressure in spite of an increased cardiac output.

Although the urine output is one of the most reliable indices of tissue perfusion, it may be unreliable in the presence of renal disease, or in the syndrome of high output renal failure.

The frequency and rapidity of occurrence of lung complications is greatly increased in shock. Bronchospasm, retained secretions, atelectasis, pneumonia and pulmonary oedema are common, especially after upper abdominal operations or in patients with preexisting chest disease. These are commonly associated with changes in the rate and depth of respiration, and in alterations in blood gases and pH, and may indicate the need for supplementary oxygen or respirator therapy.

Coolness, vasoconstriction and sweating are the indications in the skin of an excessive amount of circulating catecholamines, and indicate inadequate tissue perfusion, even in the presence of a normal blood pressure. However, for reasons which are at present not clear, in some cases of septic shock, the skin may be warm and dry. In this condition also, the body temperature may be elevated, in contrast to the usual finding of a falling temperature in hypovolaemic or cardiac shock.

In the absence of head injury, decreasing levels of consciousness are of serious prognostic significance, and indicate marked circulatory failure. Minor disturbances of consciousness, such as irritability, confusion, blurring of vision and tinnitus frequently occur in shock, and are usually reversible when tissue perfusion is restored.

Evaluation of Diagnostic Tests

The measurement of central venous pressure provides the same information as jugular venous pressure more conveniently, but it is liable to the same errors. A long radio-opaque catheter is inserted by a cut-down or percutaneously through the brachial vein to the superior vena cava, or through the saphenous vein to the inferior vena cava, and is connected to a water manometer. The catheter is kept patent by infusing a solution containing 1000 i.u. of heparin in each litre, and this route can be used for the administration of blood or fluid requirements. The manometer allows accurate measurement of the CVP to be carried out at frequent intervals by the nurse, but because of the danger of infection or thrombi, the catheter should be removed as soon as the cardiovascular dynamics are stable, and preferably within three days.

The haemoglobin and haematocrit provide valuable information of the extent of haemodilution in children and young adults, but are less reliable in the older patient, especially where extensive fluid losses or sequestration may lead to haemoconcentration. Similarly, the serum electrolytes must always be interpreted in the light of intravascular and extracellular fluid losses. A rising Blood Urea Nitrogen indicates the kidney's inability to excrete urea, either due to fluid or electrolyte shifts, or to primary kidney disease. Urinalysis will assist in determining which of these conditions is present.

Blood gases provide an important index of the efficiency of tissue respiration. Acidosis is the general rule in shock, and the healthy lung will compensate to some extent for the metabolic acidosis by hyperventilating to remove CO_2: thus pCO_2 will be lowered. If lung disease prevents this compensation, respiratory assistance with a respirator and tracheostomy should be considered.

Although blood volume determinations using I^{131} labelled albumin are subject to error, serial determinations are a more reliable index of changes in volume, and are more useful than determinations of cardiac output, which must at present be considered a research procedure. A similar status must be accorded most of the pulmonary function studies which are occasionally performed on shocked patients, and the determination of blood lactate and pyruvate levels, which are available in only a few research-oriented biochemical laboratories, in spite of their prognostic significance.

Under certain circumstances, many other laboratory tests may be indicated: the commonest of these are blood cultures, and cultures of other specimens; blood clotting factors, if a consumption coagulopathy or excessive fibrinolysins are suspected; serum calcium in cases of acute pancreatitis, and liver function tests where liver failure follows surgery and is associated with shock.

The Therapy of Shock

From the above physiological and diagnostic considerations, it will be seen that the management of the shocked patient requires the following:

Fluid Therapy

Fluid deficits must be replaced with the same type of fluid, using either blood, plasma, a balanced salt solution (lactated Ringer's Solution) or less commonly, glucose-water. Amounts must be adequate to compensate both for observed losses, and for presumed losses into swollen tissues.

Correction of Acid-Base Balance

Intravenous infusion of sodium bicarbonate solution to correct the acidosis that is almost invariably present should be calculated according to the formula:

Deficit = (Normal Bicarbonate - Actual Bicarbonate) x E.C.F.V.

where ECFV is the calculated volume of extracellular fluid. It should be remembered that this calculation underestimates requirements because it does not take into account

the intracellular acidosis which may be present.

Pulmonary Function

All patients in shock require supplementary oxygen, but if the pO_2 cannot be maintained by these measures, respiration must be assisted by endotracheal intubation or tracheostomy and positive pressure respiration.

Circulatory Support

Evidence of failure must be treated by digitalisation, but if a poor output continues, isoprenaline not only increases the force of myocardial contraction and tends to reduce venous pressure, but also causes mild vasodilatation through a reduction in peripheral resistance. One to five mg. in 500 c.c.'s of I.V. fluid run slowly will usually suffice to keep the blood pressure above 90 mm.Hg. systolic, but the drip rate should be adjusted to produce the maximum pressure rise with minimum increase in cardiac rate.

Treatment of Infection

Many cases of shock are complicated by infection, or may be entirely due to sepsis. Multiple cultures of blood, urine, stool, sputum and drainage sites should be taken before antibiotics are given. A broad spectrum antibiotic such as Kanamycin is given intravenously for immediate results, then changed if necessary to a more specific one when the results of cultures are available.

The peripheral vasoconstriction of septic shock can be reversed by the use of vasodilators such as isoprenaline, dibenzyline at the rate of 1 mg/15 Kg. body weight, or chlorpromazine in 5 mg. increments to a total dose of 25 mg. Antihistamines such as Benadryl, 50 mg. or Periactin, 80 mg. intravenously may be helpful, while large volumes of sodium bicarbonate solution and extensive volume replacement with lactated Ringer's Solution are essential.

Summary

The successful clinical management of shock depends upon a thorough understanding of the pathological physiology of the condition, and an accurate diagnosis of the state of function and reserves of all the major body systems. Although the mortality is high in this poor-risk group of patients, it can be substantially reduced by intelligent anticipation, and early, effective and adequate therapy. □

Basic Principles in the Treatment of Hand Injuries

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Hand injuries are very common, and even the minor injury can keep a patient off work for the same length of time as an abdominal operation. Only the best treatment can get the best results, and these can only be obtained at the first operation. Thus, it is vital to choose exactly the right procedure, and make sure it is carried out perfectly. For healing to occur in any tissue it must be rested; while the hand is being rested oedema and fibrin are binding down all the mobile structures, and if rest is enforced too long, nothing will move again in the hand. However, if the hand is mobilised before healing is complete, healing will be delayed and oedema will be made worse. In effect, every injury causes a race between the healing process in the tissues and the effects of oedema in stiffening the hand.

Skin

Let us consider the effects of injury in each of the individual structures of the hand in turn, starting with the skin. Primary closure is the rule for all hand injuries: the old golden rule that these injuries should not be closed if more than eight hours has elapsed since injury has gone with the use of adequate immobilisation and antibiotics. Closure should never be tight, and, since excess skin is not available in the hand, skin grafts, or local or more distant flaps may need to be used. The cross-finger pedicle flap, for example, is an ideal method for closing injuries with skin loss on the flexor surface of the fingers or thumb.

Only very rarely is primary closure contraindicated: examples are cases where the wound is obvi-

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ously contaminated, as in a crush injury or gunshot wound, and does not come to surgery until 24 or 48 hours after injury. Here, delayed primary closure is the method of choice. All dead and injured tissue must first be removed in a complete and thorough debridement, even though it may take an hour, and is best done under pneumatic tourniquet at a pressure of 250 mm Hg. After complete debridement, and only if primary closure proves impossible, the wound should be packed open with a vaseline dressing and rechecked in approximately 72 to 96 hours. If the debridement was adequate, the wound should be uninfected and starting to granulate, and can then be closed with a skin graft. In the very rare event that the wound shows signs of generalised infection, closure is, of course, not possible, and treatment by immobilisation and antibiotics must be continued until the infection dies down. Only then can secondary closure be carried out, with the aid of a skin graft. I must re-emphasise that the results in terms of final usefulness of the hand are much inferior to those of primary closure, due to the prolonged immobilisation. Again, if a primary closure becomes infected, the results will be disastrous, possibly worse than those of delayed primary or secondary closure. This underlines the imperative need for adequate initial debridement.

One further use of the delayed primary closure is in wounds with poor blood supply where the skin edges become necrotic three or four days after primary closure. The obviously necrotic skin edges can then be cut away, and the wound closed, preferably by using a local or distant pedicle flap. To wait for the skin edges to slough away in this type of case is pure folly, since irreparable stiffness will result.

To sum up, primary skin closure is the ideal method of treating all hand injuries, but wound debridement must be adequate to avoid infection. If the blood supply is imperilled by lack of skin, then skin must be brought from elsewhere, either as a free graft, or as a rotation, cross-finger or pedicle flap.

Tendon Injuries

Tendon injuries are difficult to assess preoperatively, and a tendon which has been cut three-quarters of the way through will move a digit adequately and often painlessly, while, because the hand is in a different position than at the time of injury, only the intact portion of the tendon is visible in the bottom of the wound. A useful rule is that if the deep fascia has been cut and there is any possibility of tendon injury, the hand must be explored under block or general anaesthesia using a tourniquet. Otherwise, tendon injuries are bound to be missed. If a tendon is found to be cut, the absolute rule is that, except where the injury is in the fibrous flexor sheaths of the fingers, primary suture must be carried out.

If a cut tendon is missed, or if primary suture is not done, the muscle will retract, making resuture impossible. Flexor tendons are difficult to repair, and unless they are repaired with perfect accuracy, adherence to surrounding tissues will occur, and there will be no movement. Tendons that have been cut in the fibrous flexor sheaths require a tendon graft, and because this may be required in many flexor tendon injuries, I think it is extremely unwise for a surgeon to attempt to repair them unless he has had quite a lot of experience. Extensor tendons are relatively easy to sew up, except for the occasional difficulty in finding the proximal end.

Nerve Injuries

Controversy continues as to whether these should be repaired primarily or secondarily. Personally, I think the primary repair is better, because the period of immobilisation is reduced, but they can also be repaired within a month or two of injury, so that there is no urgency about getting them done. Whatever method is used, nerve repairs give poor results, even with attention to detail. Under magnifying vision the nerve is partially resected to remove the growing axons, and the cut ends are carefully approximated, using fine sutures, in perfect alignment. By the time that a poor result shows up, it is too late to re-anastomose the nerve, so it is important to obtain a good apposition at the first attempt. As in the case of flexor tendons, I feel that nerve suture requires a lot of experience and perfect conditions to achieve even a modest result.

Skeletal Structures

It has been said that it is easier to treat a fractured femur than to obtain a successful result with a fracture of a proximal phalanx in the hand. Although this is an exaggeration, it underlines the difficulty in dealing with fractures in the hand. Unless alignment is close to perfect, adhesions develop, and the resulting imbalance produces deformities and stiffness. Closed fractures should be reduced under X-ray control to a position of perfect alignment, and if this cannot easily be obtained, there should be no hesitation in arranging for open reduction and alignment by internal fixation, using K wires. Even compound fractures should be treated by open K-wire fixation, as far as I am concerned, provided this is combined with adequate debridement to reduce the risk of infection.

Summary

Hand injuries are a serious cause of disability. Properly treated, with careful attention to every individual structure in the hand, rapid healing with minimal fibrosis will be obtained. The cardinal rules are primary closure of skin, primary suture of tendons, and primary opposition of cut nerves, provided adequate debridement is carried out. □

Anaesthesia For Acute Trauma

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The skill of the anaesthetist in making a rapid, accurate assessment of the patient's condition is never more necessary than when he is confronted with the problem of anaesthetising a patient who has recently been subjected to severe trauma. In addition to the difficulty of obtaining a history of pre-existing disease or previous medications which may affect the patient's reactions to anaesthesia, frequently there are difficulties in assessing the amount of previous blood loss, and rate of present loss, since this may be internal as well as external. Establishment or maintenance of an adequate airway and adequate ventilation may be technically difficult and can introduce a new set of hazards, threatening complications which must be carefully avoided or treated immediately.

The importance of introducing a large bore plastic cannula to establish an intravenous route *as soon as the patient is seen*, cannot be over-emphasized. At this time, the patient's veins are still visible: later, when vasoconstriction supervenes, the veins will disappear, and cut-down procedures may have to be used to obtain an intravenous route. When this needle is placed in position, blood should be withdrawn for grouping and cross-matching. The introduction of a central venous pressure cannula at this time will provide valuable information, monitoring the effects of previous and continuing blood loss. Because of the anaesthetist's special skill in making the most of the available veins, he should form part of the team that sees the patient on admission, since his assistance may also be required in obtaining control of an airway imperilled by oropharyngeal bleeding, facial fractures, or haematoma of the subglottic and laryngeal airways.

Once an intravenous route has been established, and airway control is secured, the anaesthetist can make a careful assessment of the patient's cardiac, circulatory and respiratory status, and attempt to secure information concerning the patient's previous medical history. Age, previous respiratory or cardiac disease are the factors which may most vitally affect the outcome of anaesthetic and operation, but one should not overlook the possibility of previous endocrine disorders such as diabetes, adrenal insufficiency (especially that associated with steroid therapy) or hypothyroidism, or the possibility of co-existing renal disease which may predict a post-operative renal failure if prolonged hypotension occurs, and may interfere with the excretion of some anaesthetic drugs.

The Time of Operation

The decision as to the time of operation is one in which the anaesthetist must be prepared to exercise considerable judgment. If there is evidence of steady haemorrhage that cannot be controlled by pressure, as in a case of a ruptured liver or spleen, or a torn mesenteric vessel, no amount of blood replacement will save the patient, and his only hope is immediate operation and ligation of the bleeding point, no matter how poor the condition of the patient. However, once the bleeding point is located and ligated, no further surgery is allowed until the patient has been adequately transfused with massive quantities (up to 3000 ccs) of lactated Ringer's solution, or normal saline or a plasma expander, such as 6% Dextran. In some cases where exsanguination is severe, it may be necessary to use unmatched Group O, Rh negative blood to provide an adequate amount of circulating haemoglobin. Considerable tact may be required to persuade the surgical team to stand idle during this time, but there is no doubt that it is in the patient's best interests, and firm insistance will usually prevail.

Experience with a succession of patients with this type of problem may persuade a surgeon that every severely injured patient must be rushed to the operating room, and here the anaesthetist must firmly resist all attempts to persuade him to go ahead with an anaesthetic until there is an adequate supply of cross-matched blood available, until there has been an opportunity to adequately assess the patient, and his blood volume, as indicated by blood pressure, pulse rate, skin color and central venous pressure, has been restored to normal. The main difference in approach is that there is no guarantee that there is one major source of bleeding, and surgical intervention will inevitably result in further blood loss in an already hypovolaemic patient, and is simply out of the question until the patient's condition is improved and blood is available.

There is one other indication for immediate anaesthesia and operation: this is the patient with a

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head injury who loses consciousness, and who shows signs of rapidly increasing intracranial pressure by the dilatation of one or both pupils. Not only does anaesthesia make surgical relief of the increased pressure possible, but the anaesthetist, in establishing an intravenous route, can inject hypertonic 50% dextrose solution and mannitol solution to reduce brain volume, and by the application of vigorous hyperventilation, can often reduce brain volume sufficiently rapidly that the dilated pupil will constrict, and the damaging additional pressure will be relieved until surgical relief can be accomplished.

There are many other considerations which may indicate a postponement of operation, or a choice of a regional or local technique as a substitute for general anaesthesia such as the presence of a full stomach, or the presence of co-existing disease of other systems. While it is impossible in a brief review article such as this to cover all of these possibilities, a few of the major problems will now be discussed.

The Full Stomach

Under normal circumstances, the stomach is empty within four hours of a meal, but injury, pain, haemorrhage, and pain-relieving drugs have been shown to cause pylorospasm and inhibit gastric emptying. If an accident has occurred within two hours of a meal or of drinking alcohol, or if there has been any oral intake after the accident, it must be assumed that the patient has a full stomach. It must be remembered that oropharyngeal injury will result in the swallowing of blood, and on occasion, the stomach may contain as much as two to three litres of swallowed blood. In these circumstances, the indications for operation must be weighed against the hazard of aspiration of vomitus. Where the injury is such that operation cannot be performed under a regional or local block technique, and the patient cannot be held until there is a reasonable likelihood that his stomach is empty, a special technique of inducing anaesthesia to avoid the hazard of aspiration must be employed.

If feasible, a stomach tube should be passed, and the stomach aspirated in the operating room, just prior to the induction of anaesthesia, with the patient first supine, and then turned onto his right side, since the portion of the stomach which lies to the right of the vertebral column may remain full unless this is done. The stomach tube is then withdrawn, since leaving it in position keeps the cardiac sphincter open, and favours regurgitation of stomach contents. If the patient's circulatory status permits, he is then elevated into a sitting position, and a free-running intravenous route is established. The anaesthetic machine is checked, a working laryngoscope must have been tested, and a cuffed endotracheal tube, also tested, must be ready for use. A large bore suction, such as a tonsil suction is placed on the anaesthetic machine and connected to a strong source of suction, and the patient is then asked to breathe oxygen for two minutes. An assistant is instructed to stand by, ready to compress the cricoid cartilage against the oesophagus as soon as the patient loses consciousness. Anaesthesia is then induced with a calculated dose of thiopentone, sufficient to produce brief unconsciousness, but not so large as to produce circulatory embarrassment, and this is immediately followed by a full paralysing dose of succinycholine (Anectine), usually 60 mg. In this way, the patient becomes unconscious and paralysed in the same instant, and can no longer vomit actively. He is then intubated deftly before passive regurgitation can take place, and the cuff of the endotracheal tube is inflated to prevent aspiration. Anaesthesia is then continued as indicated by the circumstances of the case, the patient being rapidly returned to the horizontal position.

Maxillo-Facial Injuries

This injury, graphically called the "facial smash", is characterised by disorganization of the facial bones, and loss of control over the airway. Bleeding into the oropharynx is often profuse; hypovolaemia and a stomach full of blood is often present when the patient arrives in hospital. The multiple fractures give rise to continued oozing, which is accentuated by surgical manipulation, and is difficult to control, so that blood *must* be available before operation.

Tissue swelling is often marked, giving rise to respiratory obstruction from the swollen tongue and pharyngeal tissues. It is often difficult to judge whether it will be possible to maintain an airway when the patient's oropharynx is relaxed by an anaesthetic, or whether endotracheal intubation will be possible in the presence of blood clot and free bleeding. A golden rule is never to induce anaesthesia in the presence of upper airway obstruction. A tracheotomy should first be performed under local anaesthesia, and a cuffed tracheostomy or endotracheal tube introduced through the stoma so that the trachea can be isolated from the oropharynx by inflating the cuff. Once a safe airway is established, anaesthesia can then be induced and continued in the conventional way.

Chest Injuries

In open chest injuries, where the affected lung collapses, and the mediastinum swings away from the affected side because of air at atmospheric pressure entering through the open chest wall, positive pressure ventilation is often life-saving. Because hypoxia in open pneumothorax induces hyperventilation, it is often difficult to carry out effective mouth-to-mouth respiration, and at the site of the accident, the most effective aid is the hand resuscitator with a self-inflating bag, which allows venti-

lation of large volumes via a mask or endotracheal tube. The opening in the chest should be closed as rapidly and effectively as possible by laying the victim on the affected side, or applying occlusive pressure with the palm of the hand or adhesive strapping.

In closed chest injuries, where fractured ribs may perforate the lung and give rise to a pneumothorax, positive pressure ventilation may be extremely dangerous, since it may force more air into the pneumothorax, the tear in the lung acting like a valve. Tension rapidly builds up on the affected side, pushing the heart over to the sound side and interfering with the return of blood to the heart. The patient's condition rapidly deteriorates, and unless the pressure is relieved by inserting a needle into the affected side, the patient will die within a few minutes. If positive pressure appears essential in the presence of a pneumothorax, a chest tube should first be inserted in the second or third interspace, using a trocar and cannula, and connected to an under-water seal.

If a flail chest is present, where one segment of the thorax is floating free of bony attachment, respiration will be inefficient because this free segment is sucked in on inspiration, and pushed out on expiration. A severe flail chest injury requires positive pressure ventilation via a tracheostomy and respirator for up to three weeks following injury, and if these facilities are not available at the hospital nearest the site of the accident, the patient should be intubated with a cuffed endotracheal tube, and manual artificial ventilation should be instituted with a hand resuscitator bag and maintained while the patient is transferred to an institution that has the necessary equipment to deal with long term respiratory care. Delay in transfer may result in the development of intractable atalectasis and pneumonia which may seriously affect the patient's chances of survival.

Co-existing Disease

There is no doubt that survival from serious injury is more frequent in the young and healthy, and that any additional disease, no matter how trivial, may be the one factor that proves to be too much for the patient and results in his death. Attention to detail, uncovering evidence of renal impairment, hepatic involvement, metabolic disorders such as diabetes, myxoedema and adrenal insufficiency, whether primary or secondary to steroid replacement therapy, before operation, may prove to be major factors governing the ultimate survival of the patient. Of particular importance is the existence of respiratory or cardiac disease. Restrictive or obstructive pulmonary disease may show itself under anaesthesia as decreased compliance, with or without increased secretions, and may lead the anaesthetist to advise prolonged intubation or even tracheostomy, coupled with postoperative respiratory assistance until the patient demonstrates an adequate respiratory reserve, enabling such aids to be discontinued.

A history of a previous myocardial infarction, or evidence, when the patient is connected to an electrocardiographic monitor in the operating room, of ischaemic changes, is an important indication that these patients will tolerate anaesthesia very poorly. Any episode of hypotension may imperil an already restricted myocardial blood supply, resulting in a sharp reduction of cardiac output under anaesthesia. In addition to taking an E.K.G. before operation, the anaesthetist should be prepared to deal with hypotension through the use of vasopressors and light anaesthesia, combined with blood volume replacement as indicated by the central venous pressure levels. Arrhythmias occurring under anaesthesia may also seriously impair cardiac output, and should be corrected by the appropriate pharmacological and physiological measures. Adequate ventilation to remove carbon dioxide, with an adequate concentration of oxygen, combined with lightening of the anaesthetic level will abolish many arrhythmias, but persistent supraventricular tachycardias may indicate the judicious use of neostigmine or other cholinergic agents, while atrio-ventricular block will usually respond to atropine or to an isoproterenol drip. Ventricular arrhythmias indicate vagal stimulation in the presence of endogenous or injected adrenalin, and may precede the serious arrhythmias of ventricular tachycardia and ventricular fibrillation. They should be treated immediately with the intravenous injection of 60 – 90 mg of lidocaine (Xylocaine) or the use of 2 – 4 mg. of propanalol (Inderal) by the same route.

In addition to dealing with arrhythmias, the anaesthetist must be prepared to deal with acute left ventricular failure and pulmonary oedema through the usual methods of positive pressure and venesection, and occasionally through the use of a short acting ganglion blocking drug such as trimetaphan (Arfonad), given by intermittent injection or as an intravenous infusion. Digitalisation to deal with impending right heart failure, associated with tachycardia and a low output, may occasionally be necessary.

Summary

Faced with the many problems of acute traumatic accidents, the anaesthetist can bring to the team a particular assortment of technical skills and medical judgment which will contribute to the good handling and survival of the patient. Through his minute-to-minute assessment of the patient's condition, and his readiness to treat any of the acute problems which may occur during anaesthesia and operation, and his ability to provide adequate pain relief and adequate ventilation in the postoperative period, the anaesthetist strives to restore the patient's disordered physiology towards normal while the surgeon is repairing the mechanical effects of injury. □

Panoramic Radiology

A Diagnostic Tool Useful For Diagnosing Temporomandibular Joint Disturbances

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In the past it has been difficult to visualize the entire maxillo-mandibular region on a single X-ray film without superimposition of structures which make the region difficult to view. This is particularly true of the temporomandibular articulation.

A tomogram of the temporomandibular joint, corrected for the various angulations which the ramus and the head of the condyle of the mandible, makes with the glenoid fossa, is still the best tool to reveal subtle bony changes in the joint. Its usefulness is reduced because of its extremely limited field of coverage. For this reason fractures of the neck of the condyle can be missed. Similarly, when interference with the growth centre produces a discrepancy in size between the rami of the mandible, the tomograph is unlikely to reveal it.

A Panoramic X-ray has the advantage of reproducing the teeth and supporting structures, the maxillary region extending to the superior third of the orbits, the entire mandible extending to the temporomandibular joint region, all on one radiogram. For this reason the panoramic X-ray is an indispensable tool in diagnosing disorders of the temporomandibular joint resulting from trauma and developmental failure.

Panoramic radiography utilizes the principle of curved-surface laminography in which anatomic structures in a selected plane are recorded, while the intervening structures are so blurred that they are not discernable on the film.

There is some degree of inherent distortion in all panoramic radiographs because a fixed beam-film relationship is utilized to project structures which vary in the same individual and between individuals. In addition there is a 7-12 per cent over-all enlargement of the radiographic image because of the varying object-film distances and the fixed anode-film distance.

In spite of this, a panoramic film reproduces all the structures seen, in a correct relationship one to the other. It is this fact that makes it such a useful diagnostic aid.

The machine, the Panorex (Figure I) is simple to operate. The patient remains stationary and the tube, (A) and cassette, (B) rotate around the patient's head about the axis point (C). The movement is complete in 22 seconds.

The film produced is depicted in Figure 2. With variations in technic a radiogram of the temporomandibular joint is readily produced, (Figure 3.)

Figure 2 depicts an abnormal left (L) condyle. Such an abnormality is usually due to either trauma or to interference with the growth centre. If inter-

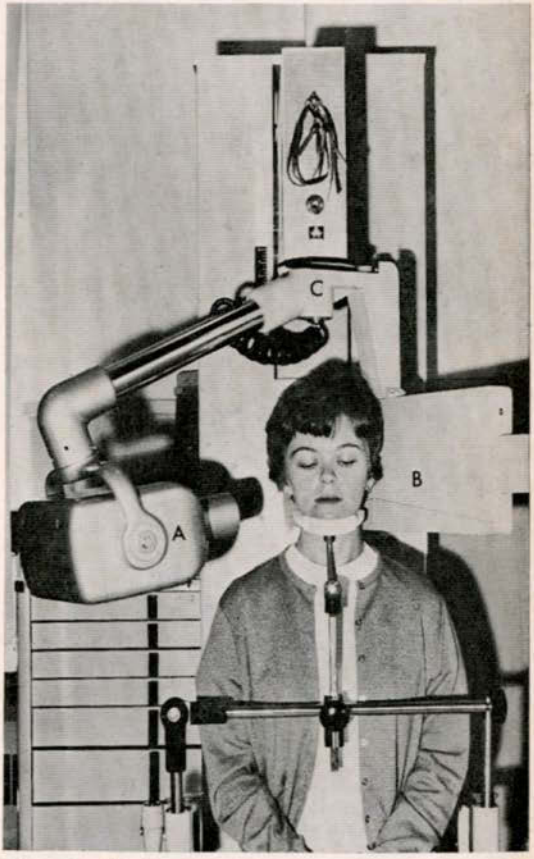

Figure 1. The Panorex Panoramic Radiographic Machine A: Tube, B: Cassette, C: Axis of rotation.

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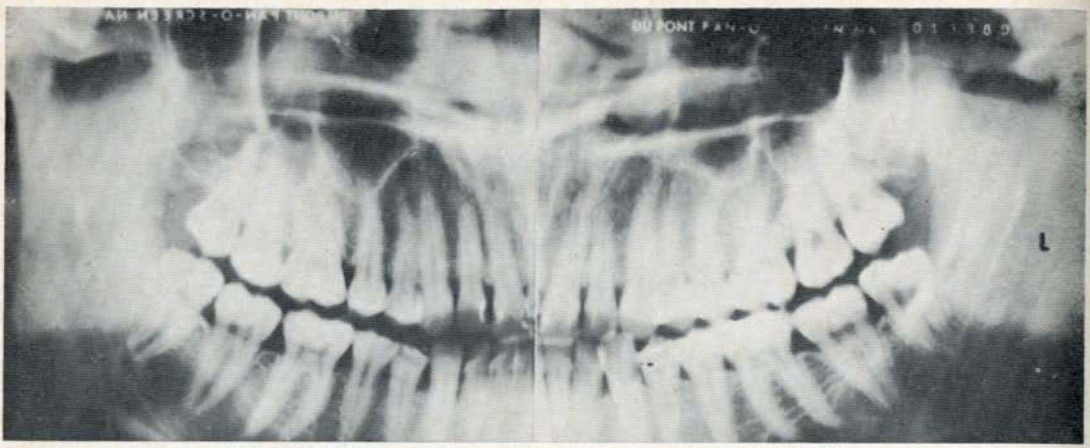

Figure 2. Panoramic radiograph showing abnormality of left Condyle.

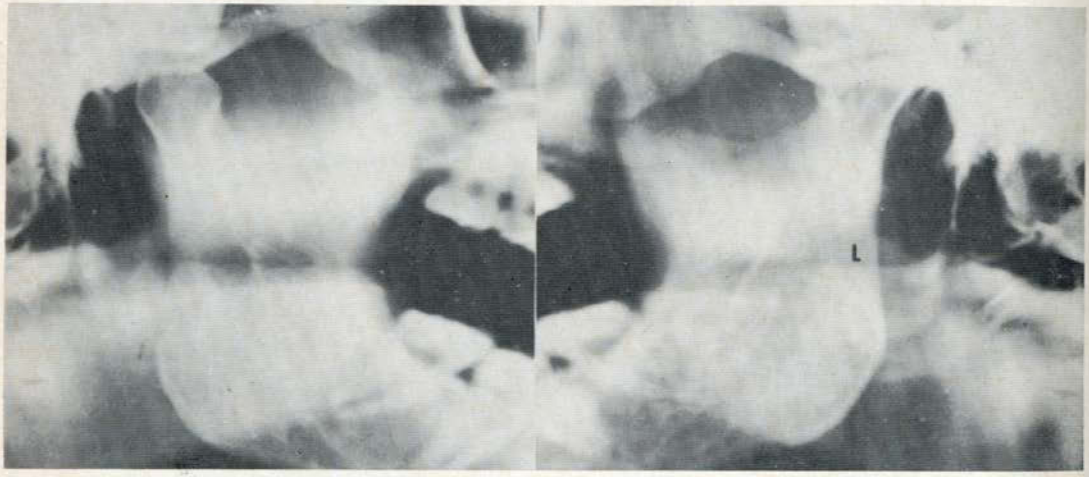

Figure 3. Technical adjustments give excellent visualisation of the temporo-mandibular joints.

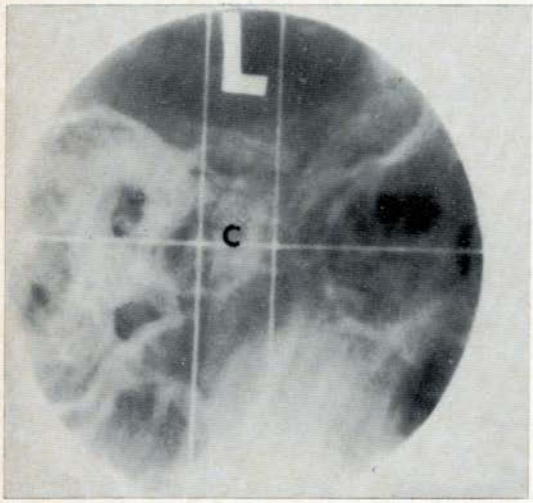

Figure 4. A standard temporo-mandibulor joint radiograph at approximately the same magnification, shows much less detail.

ference with the growth centre takes place at an early age the head and neck of the condyle, the ramus and the coronoid process of the mandible are all affected. The earlier the age the more severe the deformity.

Both temporomandibular joints must act together in function. If there is a lack of growth of the osseous components or if there is damage to the head and neck of the condyle due to trauma, the musculature and the articulation must compensate, thus joint pain and dysfunction often results.

Figure 3 is the same joint using a special "joint technic."

Figure 4 depicts the ordinary joint radiogram, produced by means of a "joint board" and a standard X-ray machine.

Figure 5 shows a fracture of the neck of the left condyle, A. This fracture was overlooked in two series of routine skull radiograms.

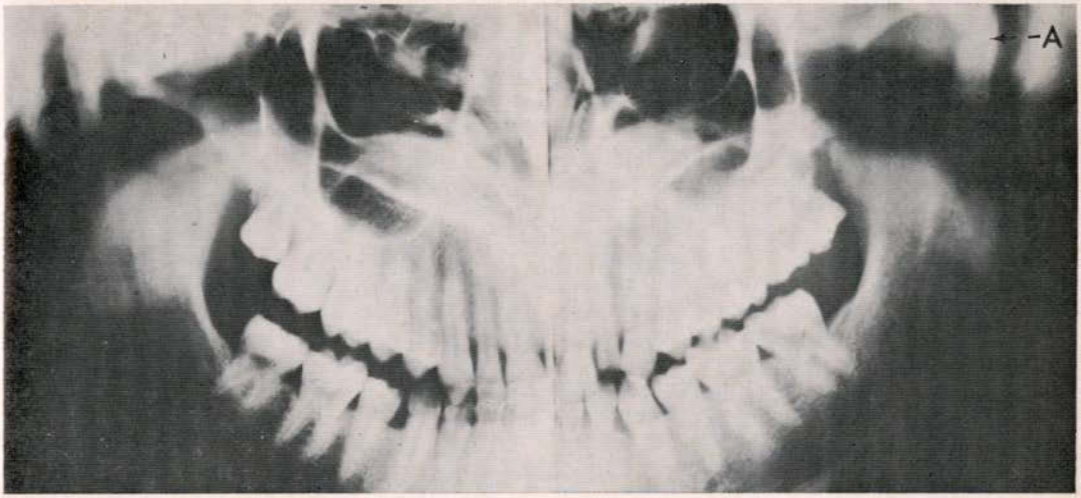

Figure 5. Panoramic Radiograph showing fracture of the left Condyle (A). □

Eye Injuries

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Trauma to and around the eye is very common and the family physician is often unsure of its significance and management. This article will attempt to outline the more common injuries and their initial treatment. Most such injuries are of a minor nature and can be readily handled by the family physician but he must be able to recognize the more serious ones and know when to refer the patient.

Foreign Bodies

Superficial foreign bodies in the eye are by far the most frequent form of minor trauma that we see. It is important that a careful assessment be made of the patient's vision before a foreign body is removed, or before any ocular treatment is begun. The old axiom that "a blind eye gets injured" is all too frequently true and it is surprising how often a person is unaware of poor vision in an eye, particularly if it has been poor since childhood. It is very easy after a minor injury to blame the defective sight on the injury or its treatment.

The examiner will need a good light and often some magnification to see small corneal foreign bodies. These foreign bodies are easily removed under topical anaesthesia; the best instrument to use is a sterile disposable needle. Metallic foreign bodies, particularly if they have been on the cornea for a day or two, will leave a doughnut shaped rust ring following removal. The cornea must be curetted to remove the rust or else the eye may be chronically irritated. Following removal of a foreign body a topical antibiotic should be instilled and the eye firmly patched for 24 hours to facilitate healing of the damaged epithelium.

Conjunctival foreign bodies can usually be wiped away with a cotton-tipped applicator without an anaesthetic. If the patient has the sensation that a foreign-body is present in the eye and nothing can be found, the upper lid should be everted; the foreign body will often be found under the lid a few millimetres from the lid-margin. A clue to the

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presence of a foreign body in this location may be obtained by instilling a drop of fluorescein in the eye and observing the vertical scratches on the cornea made by the foreign body under the moving lid.

Simple abrasions to the cornea produce the same symptoms as a foreign body. If no foreign body can be found, fluorescein on the cornea will stain any abrasion present a brilliant green (a wetted fluorescein impregnated filter-paper strip should always be used, since liquid fluorescein in a bottle is an excellent culture medium and a potential source of infection). Corneal abrasions are similarly treated with a topical antibiotic and a firm bandage for 24-48 hours until the abrasion is healed.

A final note is that if the patient's symptoms persist and nothing can be found, he should be referred, since many foreign bodies are very small, may be transparent, or may be hidden in the fornices of the conjunctiva. In any injury, if severe blepharospasm prevents adequate examination, a topical anaesthetic in the eye will usually allow the lids to be separated and the injury evaluated.

Chemical Injuries

Alkaline burns are the most important chemical injuries to the eye; for example, wet cement, lime, or plaster splashed in the eye can cause serious trouble unless treated quickly and properly. The eye should be copiously irrigated with water or saline, making sure that all particles are washed out and that the irrigation is carried under the lids. A 1,000 c.c. intravenous bottle using the tubing to direct the flow is most effective. After the initial treatment, all alkaline burns should be referred to an ophthalmologist since the alkali readily penetrates the cornea and can cause continuing damage.

Acid tends to burn the skin and conjunctiva but usually is less damaging to the cornea and hence is less threatening to vision. Similar treatment by irrigation should be pursued and if the cornea is not damaged there will probably be no subsequent corneal scarring. If the conjunctiva is burned, particularly in the lower fornix, adhesions or symblepharon may form with healing. Antibiotic ointments, should be used and the eye repeatedly examined for developing adhesions.

Thermal and Ionizing Radiation Burns

Thermal burns usually involve the eyelids more than the eye itself and the treatment is the same as with any burn, bearing in mind that subsequent contractures can cause exposure of the eye. Surgery to the eyelids may be required later. If the eye itself is burned the results may be visually disastrous since the corneal stroma tends to slough and healing will cause scarring and vascularization.

Flash burns, usually as a result of welding without a mask, are very common, very painful, but usually of no permanent consequence. These injuries are due to the short ultraviolet rays which do not penetrate more deeply than the cornea but cause sloughing of the corneal epithelium. The symptoms come on 8-12 hours after exposure and are quite severe, consisting of pain, redness, lacrimation, and blepharospasm. Due to this time-delay the patient is often in agony during the night. Treatment consists of bandages to both eyes, analgesics, reassurance, and time. Topical anaesthetics should not be given for home use. Usually in 24 hours the corneal epithelium has replaced itself and the patient is comfortable with no visual damage.

Snow blindness is due to the same process but is usually less severe.

Eclipse blindness is caused by infra-red rays which pass through the eye and permanently damage the macula. There is no treatment for this.

Blunt Injuries to the Eye

Eyelids and Orbit

Since the skin of the eyelids is loose and thin, hematomas readily occur producing the typical "black eye". The lids may be swollen shut: it is important to force them open with lid retractors so that the eye may be assessed for injury. If no further damage is found, cold compresses and time will usually cause a return to normal appearance and function.

Fractures of the orbit are fairly common and can be easily missed, particularly when only the floor of the orbit is involved, producing the so–called "blow-out" fracture. In this type of fracture the thin bony floor gives way; some of the orbital contents fall into the maxillary antrum and are trapped. Initially there may be proptosis instead of enophthalmos due to hemorrhage into the orbit. The ocular movements must be assessed; diplopia on gazing upwards is a particularly common sign due to limitation of upward movement of one eye. There may be anaesthesia of the cheek in the distribution of the infraorbital nerve. Routine skull and facial X-rays often fail to show these fractures and special views must be requested. It is important that orbital–floor fractures be repaired early if normal function is to be restored. Associated fractures of the orbital margin are usually fairly obvious. Finally, if much swelling is present, repeated examinations will be necessary.

Conjunctiva and Cornea:

Subconjunctival hemorrhage readily results from blunt trauma and may even occur spontaneously with sneezing or coughing. Further ocular damage should be looked for and if none is found the patient should be reassured that no treatment is necessary and that the hemorrhage will resorb in one to two weeks. These subconjunctival hemorrhages may go through the same colour changes as a skin bruise.

Blunt trauma may also produce corneal abrasions; these have already been discussed.

Intraocular Damage:

Blood in the anterior chamber or hyphema is the most frequent intraocular injury due to blunt

trauma. Usually the amount of blood is small but can be readily seen as a blood level at the bottom of the anterior chamber. If these hyphemas are left untreated, a considerable number will rebleed spontaneously two to three days later. This second bleed is often more extensive and may fill the anterior chamber producing a typical "8-ball" eye. The danger is that blood under pressure will cause a secondary glaucoma with blood penetrating the cornea, producing permanent corneal staining and visual impairment. All hyphemas, regardless of their size, should be treated with bed rest, binocular bandages, and sedation for 5 to 6 days. On this regime, the vast majority will resorb without ill effects. If a total hyphema or "8-ball" eye occurs, the anterior chamber will probably have to be washed out to prevent this corneal staining.

Blunt injury often produces a traumatic mydriasis and may lead one to suspect intracranial damage. This type of mydriasis is seldom complete and some pupillary reaction will usually remain. In most cases the pupil will return to normal after several weeks but occasionally, the pupil may remain partially dilated for life. Tears of the iris root may cause a distorted or oval pupil. This is permanent but usually of no great concern; however, rarely a secondary glaucoma may occur later.

Traumatic cataracts may result from blunt trauma. These usually develop over several hours or a few days and may require treatment later. The lens may be dislocated and if the dislocation is small the aid of a slit lamp may be required for diagnosis. In any case, vision will be disturbed due to the altered refractive power of the eye. Anterior dislocation may block the pupil and produce a secondary glaucoma.

Retinal and choroidal injury is also fairly common with blunt ocular trauma. Retinal hemorrhage of any size may occur and there may be blood in the vitreous. Deep retinal and choroidal hemorrhages appear dark grey or blue in colour, whereas the superficial hemorrhages are red. If they are located in the vicinity of the macula, vision will be markedly reduced. Macular edema is likewise common and again reduces vision; however this usually subsides within a few days and good vision is the usual result. This edema appears as a greyish-white, geographic, cloudy discoloration against the red – orange background of the normal fundus. Choroidal tears occurring concentrically above and below the optic disc may be evident after blunt trauma.

Retinal detachment can occur but it is usually a late sequel to trauma. Initial detachment associated with trauma usually reflects some prior vitreoretinal traction or retinal weakness. The detached retina is greyish in colour and has the appearance of billows extending into the vitreous.

Finally, blunt trauma can rupture the globe. When this occurs the eye will be soft, full of blood and there will usually be no sight.

Lacerations

Lacerations involving the skin of the eyelids are readily repaired with 4-0 or 5-0 silk. Because of the loose skin and natural skin folds, scarring is usually minimal. In any laceration, the function of the eyelid should be assessed, particularly that of the upper lid. If the upper lid is cut and there is a degree of ptosis then the levator muscle is usually damaged and must be repaired. Lacerations involving the lid margin must be accurately reapproximated since notching of the lid margin is particularly disfiguring. Usually the other eyelid is used as a splint in suturing lid margin lacerations. A special note should be made about lacerations involving the canaliculi. Even after meticulous repair a return of normal tear-drainage is seldom obtained. For this reason patients with this problem should be referred.

Conjunctival lacerations under 3 to 4 mm. in length will usually heal without suturing. Larger lacerations should be reapproximated using 6-0 plain cat gut sutures; magnification is essential to identify the edges of the lacerated conjunctiva.

Corneal lacerations may be partial or penetrating. Scarring will occur if the laceration extends deeper than the epithelial layer; if this is in the line of the visual axis some impairment of vision will occur. All penetrating injuries are a serious danger to vision. Since the eye is, as it were, suddenly deflated there is often a piece of iris incarcerated in the wound. The anterior chamber may or may not be flat depending on whether the eye is still losing fluid. These injuries must be repaired accurately and promptly. It must be remembered that infection is a potential hazard in any penetrating injury and systemic antibiotics should be started immediately. Once established, intraocular infection is difficult to treat and usually disastrous to vision. Bear in mind that with any penetrating eye injury, unless there is a clear-cut history to the contrary, the presence of an intraocular foreign body must be suspected: an X-ray may be most revealing.

Conclusions

It must be emphasized that all ocular injuries no matter how small may ultimately affect the function of the eye. Careful examination is essential before proclaiming that there is no significant damage. If there has been intraocular injury, it is wise not to make a firm prognosis concerning the patient's vision for at least a year. When it is necessary to refer the patient, the eye should be covered with a sterile bandage making sure the eyelids are closed under the bandage. □

Calcaneal Tuberosity Fractures

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Displaced horizontal fractures of the calcaneal tuberosity reduce the tuber joint (Böhler's) angle from the normal of 30 to 35 degrees to 20 degrees or less and may obliterate it altogether or be represented by a negative angle. The degree of proximal displacement of the tuberosity by the pull of the Achilles tendon determines the amount of relative lengthening of the tendon and with extreme proximal displacement of the fragment incapacity due to relative lengthening is the same as that of actual lengthening after rupture of the tendon. As a result of weakening of the calf muscle the patient cannot stand on tip-toe and loses the normal heel-and-toe spring when walking. This type of disability can be prevented.

Anatomy

A transverse crest divides the dorsal surface of the calcaneal tuberosity into two parts; these parts vary in size depending on the level of the crest. The Achilles tendon spreads out to insert above and below the crest and extends further proximally on the medial side.

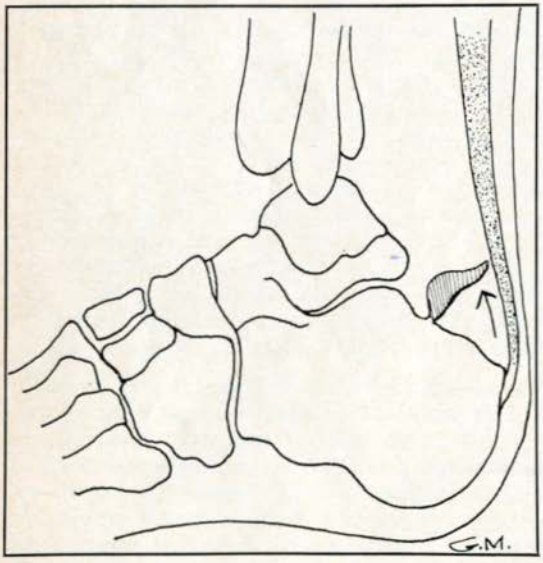

Figure 1. Beak fracture above the insertion of Achilles tendon. (Type I).

Classification and Treatment of Fractures of the Posterior End of the Calcaneal Tuberosity

These fractures are extra-articular and are arbitrarily divided here into Types I, II and III (see Böhler), although Protheroe has recently reported a few cases to support his argument that beak fractures of the calcaneus are a variant of the avulsion fracture and not a separate entity.

Type I

Beak fractures above the transverse crest of the calcaneal tuberosity and therefore above the insertion of the Achilles tendon (Fig. 1). If the foot is plantar flexed the Achilles tendon clears the fragment. In this position it can be reduced easily and reduction maintained without carrying out an open reduction.

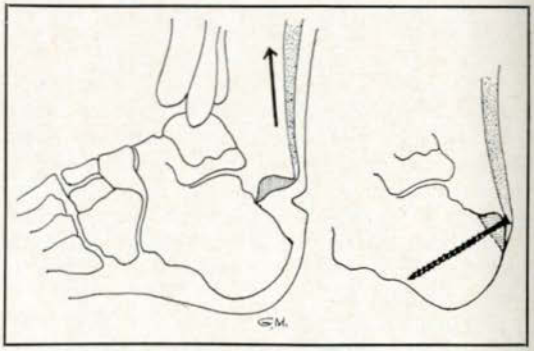

Figure 2. Beak fracture below insertion of Achilles tendon. (Type II).

Type II

Beak fractures below the transverse crest of the calcaneal tuberosity. The fragment is pulled up by the Achilles tendon (Fig. 2 and Fig. 4). The very elderly patient with reduced physical demands and impaired function may generally be treated by non-operative means particularly if there is only mild displacement of the fragment. However, operative reduction and some type of internal fixation is indicated in the younger patient particularly if the fragment is severely displaced or if there is a threat to the circulation of the skin overlying the displaced fragment. If there is a risk of pressure necrosis, then operative reduction and fixation is indicated even in the inactive, very elderly patient.

*From the Orthopaedic Section, Department of Surgery, Victoria General Hospital and Dalhousie Univerxity.

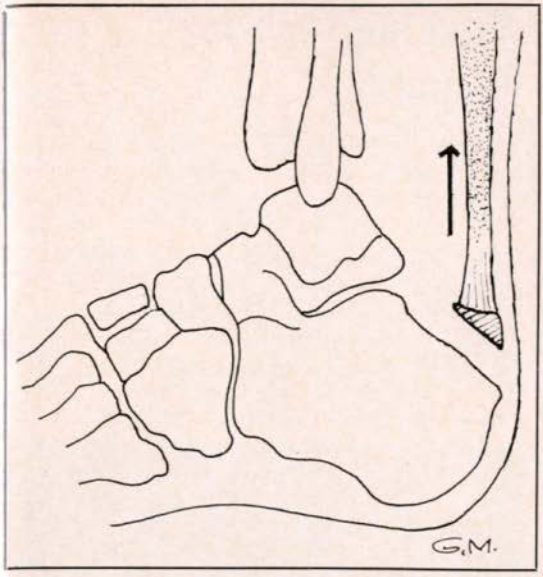

Figure 3. Avulsion fracture of the Calcaneal Tuberosity. (Type III).

Type III

Avulsion fractures of the calcaneal tuberosity below the level of the transverse crest (Fig. 3). The fragment is displaced proximally but is not tipped up. Obviously this has to be dealt with by open reduction and screw-fixation, since the disability is equivalent to that of a ruptured Achilles tendon. It has been pointed out that fractures of the posterior upper third of the calcaneal tuberosity usually occur between the ages of 50 to 70, whereas tears of the tendo Achilles usually occur between the ages of 30 to 50.

Illustrative Case

Patient C.S. aged 58 was admitted to hospital after she sustained the beak fracture of the left os calcis shown in Fig. 4. This fracture was the result of simply stepping down off a trolley. Open reduction was carried out using the incision shown in Fig. 5. and Fig. 6 demonstrates the X-ray appearances after reducing the fragment and fixing it with a screw. The post operative course has been essentially uneventful.

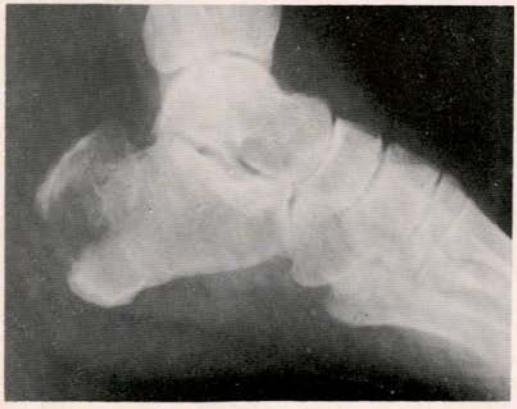

Figure 4. X-Ray of left us calcis showing beak-fracture. (Type II).

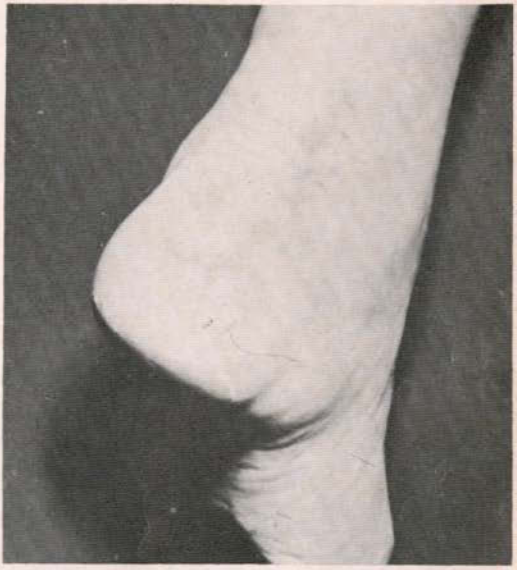

Figure 5. Incision used for open reduction.

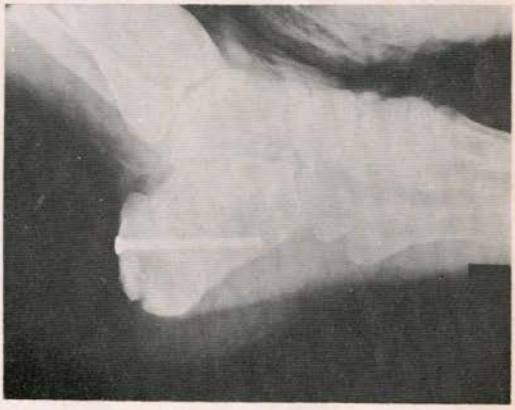

Figure 6. X-Ray appearance after open reduction and screwfixation.

Summary

Some aspects of calcaneal tuberosity fractures including a partial classification and indications for operative reduction together with a brief case report of a beak fracture have been presented. □

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Heel-Window in a Walking Cast for Calcaneal Fractures

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Early ambulation of patients with painful swollen heels following fractures of the os calcis can be facilitated by leaving an adequate window for the heel when applying a below-knee cast and a Böhler walking stirrup (Fig. 1). The writer was introduced to this technique at The Radcliffe Infirmary and has found it quite useful either as definitive treatment for selected fractures of the os calcis or as a temporary measure prior to carrying out a subtalar fusion.

Figure (1) shows the final appearance of the cast used for the definitive treatment of patient (H.H.) who had, approximately nine weeks earlier, sustained a fairly severe fracture of the os calcis including almost complete loss of Böhler's angle as the result of a seventeen foot fall off a staging. Figures (2) and (3) illustrate the appearance of the foot as well as the range of ankle dorsiflexion and plantar flexion immediately following plaster removal.

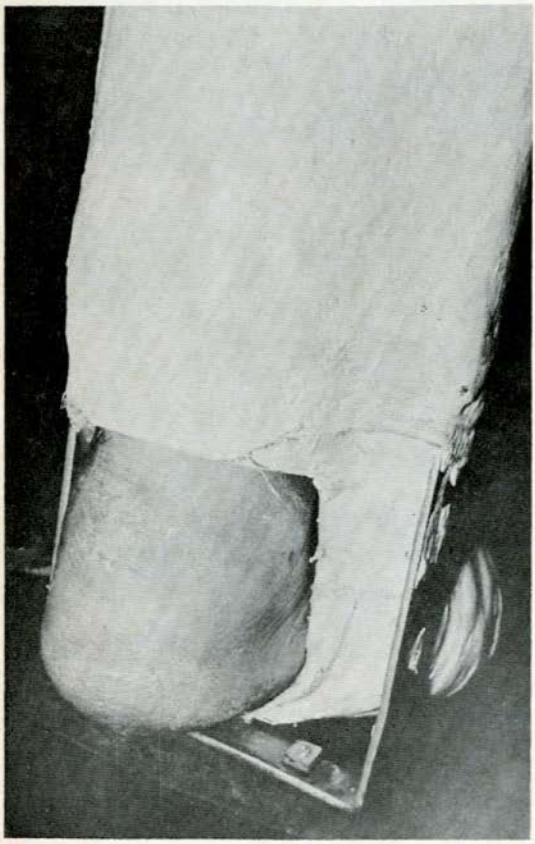

Figure 1. Below knee walking cast showing heel window and Bohler stirrup.

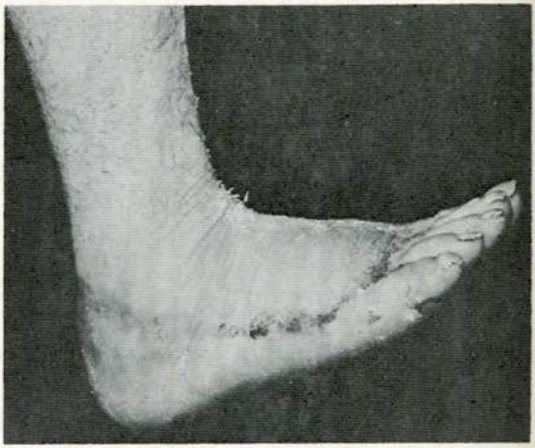

Figure 2. Appearance of foot and range of angle dorsiflexion immediately after removal of cast shown in Fig. (1).

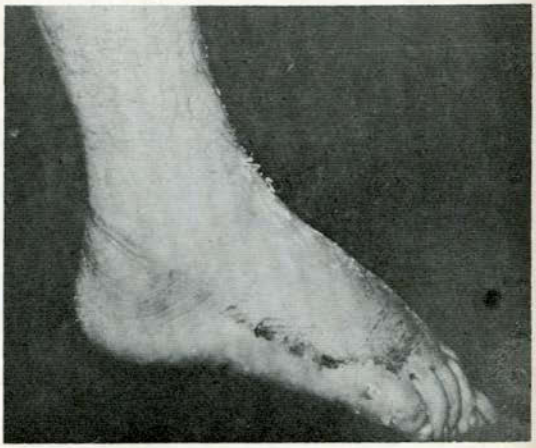

Figure 3. Appearance of foot and range of ankle plantar flexion immediately after removal of cast shown in Fig. (1).

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Geiser and Trueta[1] used animal experiments to show that osteoporosis of the os calcis developed soon after it was relieved from muscular compressing forces. All five series of their experiments causing bone rarefaction had one thing in common, namely inhibition of function of the calf muscles or making their function extremely inadequate. Plaster immobilization in a position of plantar flexion caused a quicker and more intensive bone rarefaction than immobilization in the position of 90 degrees or less; they also observed that faradic stimulation of immobilized calf muscles appeared to have a preventive action on bone rarefaction in the foot. In addition they questioned "whether in the inhibition of muscle contraction we do not have the greatest single responsible factor in establishing Sudeck's atrophy" and they related this to an earlier investigation by Barnes and Trueta[2] which showed how "the lack of muscle contraction reduces lymphatic flow and causes retention of tissue fluid".

These experimental observations suggest that there is some physiological basis for the use of the modified walking plaster described here.

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1. **Geiser, M. and Trueta, J.** "Muscle Action, Bone Rarefaction and Bone Formation," *Journal of Bone andJoint Surgery*, 40-B: 282, 1958.
2. **Barnes, J. M. and Trueta, J.** "Absorption of Bacteria, Toxins and Snake Venoms from the Tissues," *Lancet*, 1, 623, 1941.

Pump Bumps or Knobbly Heels

B. F. Miller, M.D., M. Ch. Orth. F.R.C.S., (Edin.)* and A. J. Buhr, M.D.*

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The lateral side of the postero-superior aspect of the calcaneal tuberosity may be abnormally large or may enlarge because of pressure (Fig. 1). Most people with this enlargement are asymptomatic, but occasional patients may become so disabled that they are unable to wear shoes. Young women who wear shoes with high heels or pumps are especially apt to develop Tendo Achilles bursitis or "pump bumps."[1] Other descriptive terms have been used including "calcaneus altus" and "high-prow heels". One of our graduates from the Caribbean told us that these patients were described as having "cucumber-heels" on the island from which she came.

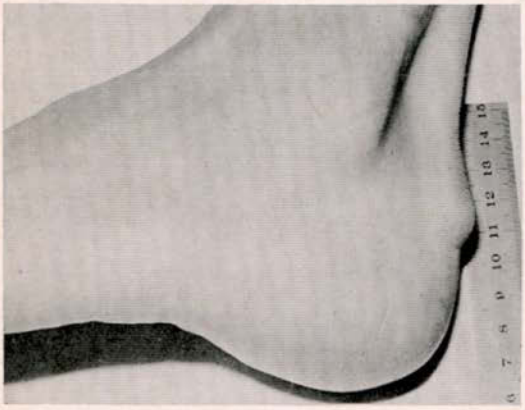

Figure 1. Typical "pump bump" or "knobbly heel".

Most patients who develop bursitis of the retro-calcaneal bursa or the subcutaneous bursa (Fig. 2) can be helped by conservative measures designed to relieve friction between the shoe counter

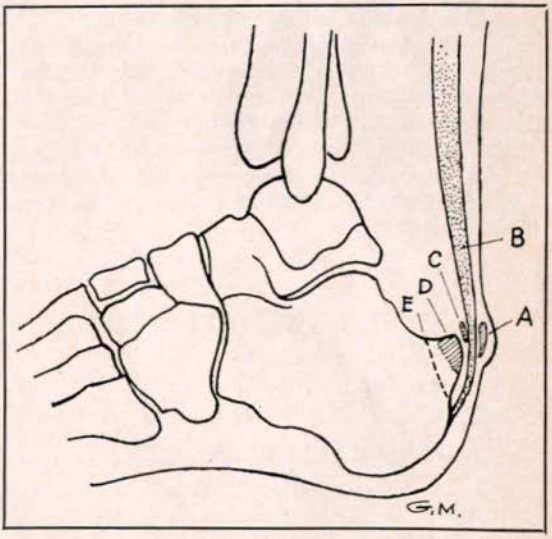

Figure 2. Schematic diagram showing anatomy of a "pump bump". A. Subcutanemus bursa. B. Achilles Tendon. C. Retrocalcaneal Bursa. D. "High-prow heel". E. Approximate level of resection.

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and the heel. Instructions to the patient to avoid shoes with closely-contoured heel counters, use of heel pads to raise the heel out of the shoe and thereby avoid pressure, or wearing a "heel-protector" made of latex or other appropriate material may provide relief. Properly fitted shoes with particular attention to the height of the heel will generally solve the problem.

Occasionally one may have to remove an enlarged painful bursa. Excision of the underlying prominence is sometimes necessary.

This brief report was stimulated by the recurrent problems of an eleven year old girl (D.R.) who having previously had a partial removal of the bony prominence on one heel through a lateral approach, continued to have disabling symptoms referable to both heels. Both bony prominences were therefore dealt with using the technique described by Fowler and Philip.[2]

With the patient prone, a horse-shoe shaped incision with the convexity upward was made around the back of the heel at a level just above the heel-counter. The skin flaps were elevated and a vertical incision, two inches in length was made through the Achilles tendon, ending at a point one-quarter of an inch above the bone. The tendon was then incised in an obliquely downward and outward direction, each limb being about three-quarters of an inch long and forming an inverted Y-incision (Fig. 3). The tendon flaps were retracted, the bursa excised and the bony prominence removed by means of an osteotome (Fig. 2). The tendon was then repaired and the skin closed. Plaster casts were applied with the ankle in moderate equinus.

In this particular patient the casts were not removed for six weeks, although Fowler and Philip suggest removal at three weeks at which time the heel of the shoe is raised one-half inch and then gradually lowered during the next two weeks.

The surgical technique described above has been used by us on other occasions and the results have been satisfactory. □

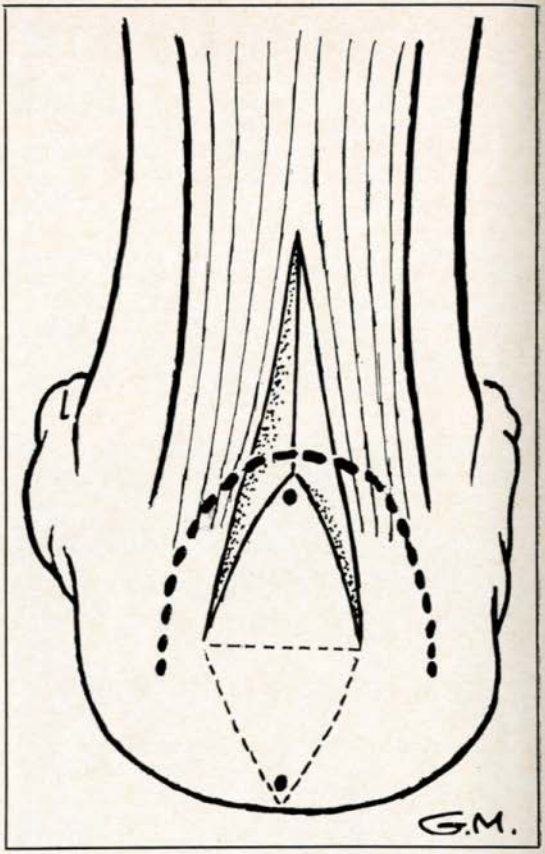

Figure 3. Surgical approach (After Fowler and Philip).

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The Initial Assessment of a Head Injury in Order to Establish the Future Course of Management

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Evaluation

Head injuries are a common problem faced frequently by most practitioners. Of the fatal automobile accidents, approximately 60% are associated with head injury. Ninety percent of all fatal motorcycle accidents are associated with a head injury. If it were possible to improve our management of head injuries, one might see how significantly this could lower the mortality figures associated with highway accidents.

The major problem for most practitioners in the handling of head injuries is to decide which injuries require specialized neurosurgical care and which do not. It is the purpose of this article to outline a few guide-lines which may be of assistance in making this decision.

The approach to a head injury problem should be just as organized and systematic as the approach to any other medical or surgical problem. A history can usually be obtained, if not from the patient, from an individual accompanying the patient. It is important to know when the accident occurred and the patient's conscious level following the accident. A deterioration in the patient's level of consciousness is, in itself, an indication of a dynamic intracranial lesion, such as a blood clot. Some patients with an extradural hematoma have a history of loss of consciousness immediately following the accident, followed by a lucid interval, prior to a progressive deterioration in level of consciousness. This is a classical history, but is by no means present in all cases of extradural hematoma. Most patients with a head injury as the result of an automobile accident, have multiple injuries. Therefore, during the initial examination of these patients, it is imperative to make an assessment of the patient's general condition so as not to overlook other injuries. On completion of this general assessment, one then directs one's attention to the head injury and the seriousness of this injury is evaluated.

First and foremost, attention should be focused on the patient's airway. The importance of establishing and maintaining an adequate airway cannot be over emphasized. Hypoxia or anoxia superimposed on an already damaged brain, may turn a reversible brain injury into an irreversible one. An adequate airway can usually be maintained in most unconscious people by posturing the patient in the semi-prone position and by inserting an oral airway.

However, if an adequate airway is not obtained in this way, steps must be taken to provide one, by passing an endotracheal tube or in some cases by performing a tracheostomy.

After providing a good airway, any obvious gross external haemorrhage is controlled. A quick evaluation of the chest is performed to rule out associated chest injuries, such as flail chest or pneumothorax.

If clinical shock is present, as indicated by a rapid pulse and low blood pressure, it is due to a lesion other than the head injury in almost all cases. Treatment of shock is started and an attempt is made to find a cause of the shock which is usually related to hidden haemorrhage from chest or abdominal injury or from multiple fractures.

If fractures of extremities are present they should be splinted. In all unconscious patients, one should palpate the spinal column from occiput to sacrum looking for tender regions or malalignments which might indicate an underlying spinal fracture.

Once these steps of providing an adequate airway, starting the treatment of shock if present, arresting any obvious external haemorrhage and splinting extremity fractures have been taken, one can then assess the central nervous system injury.

For obvious reasons, a detailed neurological examination cannot be performed where the patient is in a semi-conscious and usually restless state. However, it is usually possible to make a brief assessment which should include:

1. Level of consciousness.
2. Size and reaction of pupils.
3. Evaluation of possible weakness of one side of the body as compared to the other.
4. Presence or absence of extensor Plantar responses.
5. Evaluation of the vital signs, e.g. pulse, blood pressure and respirations.

The vital signs and neurological assessment should be repeated at regular intervals, the frequency of which would depend on the state of the patient. A slowing pulse and rising systolic blood pressure with widening of the pulse pressure indi-

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cates a serious intracranial complication such as a blood clot.

Further Management

This initial assessment of a patient with a head injury, carried out in an organized fashion as outlined, takes only a few minutes and is most important. Depending on the findings, it is then possible to decide on the further course of management.

At this stage one should ask oneself three questions:

1. Is there a decreasing level of consciousness?
2. Is there evidence of increased intracranial pressure as demonstrated by the vital signs, e.g. a slow pulse and high systolic blood pressure?
3. Are there any localizing signs such as weakness on one side of the body as compared with the other, a dilated pupil on one side, or an extensor plantar response on one side?

Depending on the answers to these three questions, head-injury patients can be divided into three groups. The further course of management will depend on the group to which the patient belongs.

Group I – In this group the answer to all three questions is "yes", that is, there is a decreasing level of consciousness, there is evidence of increased intracranial pressure as demonstrated by the vital signs and there are localizing signs present. Patients in this group have a dynamic intracranial lesion, such as a blood clot and they require emergency neurosurgical intervention. These patients are an acute emergency and require immediate transfer to a centre where neurosurgical intervention can be performed.

Prior to transfer they should be given 50 ccs of 50% glucose in water intravenously, or preferably, 500 cc. of 20% mannitol solution. These agents will shrink the brain, and therefore, decrease the intracranial pressure and allow time for transfer in most cases. Because of the massive urinary output with these agents, an indwelling catheter should be inserted prior to transfer.

Group II – In this group the answer to one or perhaps two of these questions would be "yes", however, not to all three, and patients in this group will probably require surgical intervention and therefore, should be transferred to a centre where surgical intervention could be carried out if it became necessary. However, these cases are not the acute emergencies found in Group I and time can be taken to have X-rays performed, etc. These are the cases which after arrival at a Neurosurgical Centre would have further investigative procedures carried out, and, in particular, arteriography prior to going ahead with any surgical therapy.

Group III – In this group the answer to all three questions is "no", that is, the patient's level of consciousness, although he may be unconscious, is not changing. His blood pressure and pulse are remaining stable with no indication of increased intracranial pressure. There are no localizing signs. These cases certainly make up the majority of all head injuries. Patients in this group should have skull X-rays and if there is no evidence of a depressed skull fracture, they do not require transfer to a Neurosurgical Centre. These patients should have regular assessment of their vital signs at intervals, depending on the seriousness of the case, because some of these patients will show evidence of intracranial complications at a later date which then might make it necessary for transfer to an appropriate centre. If during the management of these patients, their condition deteriorates, frequently one becomes most concerned over the possibility of an incracranial complication whereas in fact, the patient may be deteriorating because of complications developing in other areas, such as in the chest, abdomen etc. One should therefore re-evaluate the patient's general condition as well as consider possible intracranial complications. The details of the conservative management of head injury will not be outlined in this discussion, but in essence, these are conservative measures directed towards providing an environment which is conducive to self-healing of the injured brain. There is no specific therapy for the brain injury itself.

Lumbar puncture in head-injured individuals is often considered and I mention it here only to condemn it. The procedure itself, in a restless, uncooperative individual, is difficult, and traumatic taps contaminated with blood are frequent. Regardless of this, the presence or absence of blood in the cerebral spinal fluid in head-injured individuals is no indication of the seriousness of the injury. In those cases where the spinal pressure is greater than normal, the lumbar puncture itself has subjected the patient to an added risk. It is, therefore, recommended that lumbar punctures not be performed in patients suffering from a head injury.

Where problems or difficulties arise in the management of head-injured individuals, neurosurgical telephone consultation can be obtained and this practice should be encouraged. □

Tracheostomy Technique and Care

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Indications

The operation of tracheostomy has been familiar to surgeons for many years. Long lists are available of the indications for the operation, but these can be reduced to *three broad categories:*

1. Upper airway obstruction:
   most commonly maxillo-facial injury in adults, or acute upper airway infection in infants and children.
2. Acute ventilatory failure:
   examples being major chest injury such as flail chest; severe limitation of ventilation by the pain and debility of major abdominal or thoracic surgery; depression of the central respiratory centres by head injury or drug overdosage.
3. Management of airway secretions:
   this problem usually existing in combination with one of the first two indications.

The decision to perform a tracheostomy fundamentally depends upon *the recognition or anticipation of acute respiratory failure.* This diagnosis is not difficult if one is familiar with the clinical features of respiratory failure, and especially if facilities for measuring blood gases and acid-base balance are readily at hand. Several excellent monographs are available on acid-base and respiratory physiology.[1,2]

An important consideration in deciding upon tracheostomy is the alternative of *prolonged orotracheal or nasotracheal intubation*[3]. It is generally agreed that this alternative is satisfactory for 48 hours. In practice this usually means that if the expected duration of respiratory assistance is under 48 hours, orotracheal or nasotracheal intubation will suffice. These conditions are usually seen in patients having major surgery, or in drug overdosage. The patient requires the same details of care concerning cuff deflation, sterile suction, etc., as outlined below for tracheostomy care.

Surgical Technique[4]

Tracheostomy is performed in the operating room, with an endotracheal tube in place, and under general anaesthesia. Patients occasionally require intubation in the emergency department or elsewhere, but are then taken to the operating room. In transit, ventilation may be assisted with a self-inflating bag ventilator, or respirator with a portable air supply. The operations of laryngotomy and emergency tracheostomy are rarely considered. Their use is still conceivable, as in acute upper airway obstruction in infants, or maxillo-facial injuries in adults; even in these situations we have found preoperative tracheal intubation or bronchoscopy possible, under local anaesthesia. Bronchoscopy may immediately precede intubation for tracheostomy, and a nasogastric tube is often passed before induction of anaesthesia, particularly in accident victims. A defibrillator is available, and the electrocardiogram usually monitored.

Position of Patient: The patient is supine, with the chin, thyroid carilage and suprasternal notch in the same vertical line. The head is extended about 45 degrees, to bring 4 or 5 tracheal rings above the suprasternal notch, and to diminish movement of the trachea with respiration.

Exposure of Trachea: A transverse skin incision, 4 cms in length, is made a fingerbreadth above the suprasternal notch. This is deepened through the superficial fascia and platysma. The wound edges are retracted to allow vertical, midline opening of the deep fascia between the strap muscles; this opening should extend upwards to the cricoid. The strap muscles are now retracted laterally. The position of the trachea is checked frequently by palpation.

The thyroid isthmus is divided, at least in part. This facilitates exposure of the trachea, and also allows the tracheostomy tube to lie in a more natural position. After opening the pretracheal fascia above the isthmus, two hemostats are passed vertically from above. The isthmus is divided, and ligated with 2-0 chromic sutures. Only absorbable suture material is used beneath the skin. The clamps on the thyroid may be rolled gently outwards before they are removed, to expose more tracheal surface.

The Tracheal Opening: The cricoid and first tracheal cartilages must not be damaged, for fear of later sub-glottic stenosis. An opening in the fifth tracheal caritlage or lower may cause the tube to pull out of the trachea when the head and neck are flexed at the end of surgery; in addition, the tube is more likely to lie against the carina or in a major bronchus.

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Therefore the rings to use are 2 and 3, or 3 and 4. It has been customary to make a vertical, cruciate or 'T' incision in the trachea, or to cut an oval window. The technique we employ is shown in Figure 1(a).

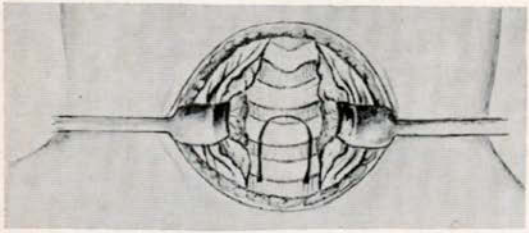

Figure 1(a): Outline of tracheal incision to create an inverted U-shaped flap.

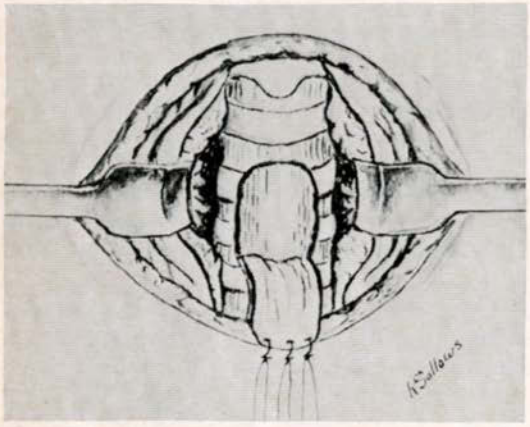

Figure 1(b): Shows the flap attached to the lower skin margin.

A transverse incision is made, between the first and second tracheal cartilages. This extends for about two-thirds of the width of trachea as seen face on, and and is made with a No. 15 scalpel blade. Downward vertical extensions, across one or usually two cartilages, are then made from each end of the transverse incision using scissors, the trachea meanwhile being steadied with a hook. This inverted U-shaped flap of trachea is then sewn to the lower skin margin, using two 3-0 silk sutures with non-cutting needles. They should be tied gently to avoid crushing tissue, with their ends being left long and taped to the skin over manubrium to facilitate later removal Figure 1.(b). If an oval window is cut, its lower margin can be sewn to the lower skin margin. The size of the tracheal opening must be large enough to accept an adequate sized tracheostomy tube.

Inserting the Tracheostomy Tube: A decision is made as to whether a cuffed or non-cuffed tube is required; several types of each are available. Cuffs are tested just before use. The anaesthetist withdraws his endotracheal tube, until its distal end is at the upper margin of the tracheal opening. The surgeon aspirates the trachea, and then inserts the tracheostomy tube. The anaesthesiologist makes connection to the tracheostomy tube as the cuff is inflated, and auscultates both lungs. The oral endotracheal tube is then removed completely. The tracheostomy tube tapes are tied in a knot at the side of the neck, after the head and neck have been put in a neutral position. Skin sutures are not used, to avoid interstitial emphysema and infection.

Aftercare

It is most important that tracheostomy patients be cared for in a specialized area, by nurses familiar with the details of suction technique, respirator connections etc. In larger hospitals, this area will be the Intensive Care Unit, while in smaller hospitals the Recovery Room may be used.

Tracheostomy Tubes:

1. Cuffed tubes:

The cuffs are deflated for 5 minutes every hour, and suction carried out. If a double-cuffed tube is used, the cuffs are alternated each hour. The patient is manually ventilated for several "maximum inflations" each hour. Cuffs are only inflated to the point where air leak is just stopped. Too great an inflation of the cuff may cause cuff tracheitis, which may in turn lead to tracheal stenosis or even tracheo-esophageal fistula. Cuffs are not inflated in patients not on respirators and not in danger of aspiration.

2. Metal tubes:

The flange should be movable on the tube, if the end of the tube is not to chafe the tracheal wall during neck movements. Inner cannulae, if used, should be cleaned at least every 8 hours.

3. Tube Changes:

These may be done whenever necessary with a flap type tracheostomy, but are routinely carried out every 2 to 4 days. The first tube change, when a tracheal flap has not been created, is hazardous before 5 to 7 days have elapsed, as the track will not have stabilized significantly before that time, and the tissues may fall together over the opening. When tubes are changed, adequate sterility, lighting and assistance are necessary, and a tracheostomy spreader, manual bag ventilator, laryngoscope and endotracheal tubes should be available nearby.

4. Tube Sterilization:

Tracheostomy tubes are sterilized in the Central Supply Room. They are packaged individually in paper, with sizes indicated.

Wound Care and Dressings

Dry gauze is used, with an underlayer of non-adhesive material. Vaseline is not used. Foam rubber is added as necessary, to prevent the tube reaching the carina. Dressings are changed twice on each 8 hour shift, and the wound cleansed with saline. Antibiotic sprays are not used unless the wound is obviously infected. Sutures attaching the tracheal flap to skin are removed at 5 days, to minimize chances of infection or epithelial union be-

tween the tracheal mucosa and skin. The flap does not fall inwards when the sutures are removed at this time.

Tracheobronchial Toilet:

1. A Regular Routine:

Coughing and breathing exercises, physiotherapy, position changes and inhalation therapy are carried out, tailored to each patient's requirements.

2. Humidification:

Continuous humidication of the patient's airways must be provided. Patients not on respirators have a humidifier connected via corrugated tubing to a Triox tracheostomy adapter*, which is positioned over the open tracheostomy tube. Water in the humidifier is usually warmed. Tracheal instillations are not used routinely.

3. Suction Technique:

Tracheostomy suction is performed hourly, or more often if necessary, using *Coudé*-tipped rubber catheters (14 Fr or 16 Fr). Suction pressure is only enough to remove the secretions, and is rarely over 150 mm Hg. The nursing routine is as follows: (a) Suction is turned on. (b) Nurse puts on mask. (c) She washes her hands. (d) A sterile-ended forceps is used to lift the proximal end of the suction catheter out of the tray of sterile water in which these catheters reside. (e) A sterile, disposable glove is put on right hand**. (f) With gloved hand, the catheter is connected to a "Y" connector, avoiding contamination of the glove. (g) With the left hand holding the "Y" connector, the right hand inserts the catheter into the tracheostomy tube, which is disconnected by an assistant. (h) The catheter is passed into the right main bronchus, the open end of the "Y" occluded, and the catheter rotated as it is withdrawn. Suction is never continuously applied for more than 5 to 10 seconds. (i) The procedure is repeated for the left main bronchus.

4. Care of Suction Catheters:

After use, the catheter is rinsed in sterile water. It is then placed in a tray of hydrogen peroxide for 10 minutes or more to loosen mucus; a syringe is used to flush peroxide through the catheter. The catheter is then soaked in Cidex† for 10 minutes, before being returned to the tray of sterile water, ready for use. Inner cannulae are similarly cleaned. A specially prepared tracheostomy care table, beside each patient's bed, has the various trays and solutions ready.

5. Bronchoscopy:

Thick mucus plugs may necessitate bronchoscopy which can easily be carried out through the tracheostome when the tube is temporarily removed. A local anaesthetic spray may be used if necessary.

6. Sputum Specimens:

Specimens for culture and sensitivity are sent at least twice weekly, and often daily, from all patients with a tracheostomy. The specimens are taken with Lukens 2 ml specimen collectors§.

Summary

The indications, technique, and aftercare of tracheostomy has been briefly reviewed. The procedure is safer in recent years because of two developments: (1) Performing the operation earlier, as a planned procedure in the operating theatre, with an endotracheal tube in place in most cases. (2) Caring for tracheostomy patients in specialized areas, by nurses competent in the details of management. □

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* – Triox Tracheostomy Adapter Set from Bennett Respiration Products, Santa Monica, California.

** – Dispos-A-Glove, from Arbrook Ltd., Somerville, New Jersey.

† – Aqueous Dialdehyde, from Arbrook Ltd., Somerville, New Jersey.

§ – Lukens 2 ml Specimen Collectors, from J. F. Hartz Co., Toronto, Ontario.

MEDICAL-LEGAL ENQUIRIES

IAN MAXWELL, M.B., Ch.B.

THE PROBLEM OF POLYCARE

Q: *I am concerned about the following type of situation. An elderly patient (say) sustains a fractured femur and comes under the care of an orthopedic surgeon. During the preliminary investigation and period of emergency care it becomes clear that she is suffering from hyperparathyroidism and anemia. An endocrinologist and a hematologist are called in. She develops urinary calculi and comes under the care of a urologist. One day she sustains a myocardial infarct; there is no cardiologist among the foregoing occupants of Mount Olympus. Whom do the nursing staff contact? If she dies without medical attention, against whom may her relatives bring suit for negligence?*

A. Even if somewhat overpainted, this is a picture of a situation which is by no means uncommon in the present age of micro-specialization.

It is my contention that when a physician accepts a patient, he enters into an implicit contract whereby he agrees to search out and treat if possible any disease from which the patient is suffering, or to see that this disease is so discovered and treated. It is quite possible that some ultra-specialists would disagree with me, but to suggest otherwise leads us into the farcical situation whereby a patient with breathlessness must perforce consult in succession a cardiologist, pulmonary physiologist, and hematologist before he learns from the gastroenterologist that he is overeating.

It may well be that the physician who is first consulted by the patient does not consider himself competent to prescribe treatment outside the field of his narrow specialty, so, as in the case cited by the questioner, he properly calls another doctor into consultation. In such cases it is wise practice for the three parties to agree mutually at the outset what the responsibilities and function of the consultant will be, either by the physician who requests the consultation indicating in writing in his request what he wishes of the consultant; by the consultant indicating in his written report what his responsibility for continuing care will be, if any; by word-of-mouth discussion between the two doctors in the presence of the patient; or preferably by all three of these safeguards. Many consultants wisely decline to prescribe for referred patients, but instead simply recommend to the attending physician what in their opinion his next step should be. In this way there is no doubt in the mind of anyone which of the two doctors is responsible for the actual care. Even this practice is not foolproof. On more than one occasion doctors have referred a patient for minor surgery, but during the hospital admission for this the investigative work-up has revealed a hidden cancer. The surgeon has dutifully reported this situation to the referring physician, but the patient has gone untreated for months, as each doctor has assumed that the other is treating the condition.

A question may arise regarding the liability of the attending physician if he acts on poor advice from the consultant. This would appear to be a situation of shared responsibility. *A priori* the consultant can be held to account for the advice he gives at the time of his examination and, unless such advice is manifestly foolish, the attending physician is absolved from responsibility if he follows it. If, despite the advice of the consultant, the patient's health deteriorates and the attending physician persists in the treatment, he may well be held to account for his actions and the consultant held not responsible if he has not been kept informed of the subsequent course of the patient.

It is customary in most large general hospitals for the house staff to assume emergency care of private patients if the attending physician is not immediately available, but, as we have indicated previously[1], the residents or interns who provide this emergency treatment are not entering into a contractual agreement with the patient and the attending physician remains responsible for their actions.

Which of the doctors is attending the Questioner's patient at the time of her death? In the absence of strong evidence to the contrary, one would assume that the orthopedic surgeon remains the attending physician. He

is the doctor whom the patient has consulted and by accepting the patient, he agrees to continue providing medical care until there has been mutual agreement to terminate the relationship. If he dissolves the contract unilaterally against the wishes of the patient, or without even notifying her that he has done so, he may be liable at law for abandoment, whereas if he has not dissolved the contract, but merely assumed that some other person will be looking after his patient, he may, at least, be charged with lack of diligence or even negligence.

In his wording of the problem, the questioner, however, leaves a minor loophole. It is just possible that the patient has come under the urologist for surgical care. Although in some parts of the country the referring physician assumes the post-operative care, in most centres now, and certainly in Nova Scotia, it is the usual convention for the operating surgeon to care for the patient throughout this period. It is possible, therefore, that the contract between the orthopedic surgeon and the patient was temporarily in abeyance at the time she suffered her heart attack.

If it were to teach no other lesson, this case illustrates well the undesirability and indeed danger of lay persons by-passing the general practitioner and consulting a specialist directly. No one could really blame the unfortunate orthopedic surgeon for not considering himself competent to treat the multiple ailments which were outlined in the problem, but by accepting the patient, he has automatically assumed responsibility for her medical care. □

References

1. **Maxwell, Ian:** "Responsibility for the Acts of Another—Part III (Interns and Residents)", *N. S. Med. Bull.* **47:** 68, 1968.

You are invited to contribute questions to our **Medical Legal Enquiries.**

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Please send completed form to: – Ian Maxwell, M.D.
Medical-Legal Liaison Committee
Department of Pathology
Halifax Infirmary
Queen Street
HALIFAX, Nova Scotia

The Medical Society and its Insurance Programs

EDWARD B. GRANTMYRE, M. D.

Chairman, Committee on Insurance

The Medical Society of Nova Scotia has three group insurance programs available for its members. These are Group Life, Disability Insurance and Office Overhead Expense Protection. All these plans are carried by large well known insurance companies and some years ago achieved the minimum numbers for group benefits. The relative cost of any of these programs is low, due to the size of the group and the fact that the Medical Society does some of the bookkeeping. As there have been significant increases in benefits, as well as some changes in types of coverage available, your insurance committee feels that a brief review of each plan is indicated.

Disability Insurance Program

A physician, when disabled by accident or illness suffers unavoidable loss of most or all of his income. A substitute source of income is necessary to carry him through such a period of financial emergency.

A long term disability income plan carried by Mutual of Omaha is available to all members. This policy will pay a maximum of $1,000.00 per month for a life-time from the 1st day of a disabling accident. Sickness benefits are payable up to five years from the 1st day of hospitalization, or from the 16th day of an illness at home. A sickness benefit extension rider is available so that benefits from a disabling illness may continue to age 65. This rider includes 3 months partial disability benefits for sickness, to enable a physician recuperating from a coronary occlusion, for example, to do a portion of his usual practice and still receive some disability benefits. The basic policy also includes a $10,000 accidental death benefit. The physician chooses the amount of monthly benefit (up to $1,000.00) for which he would like to be insured and the basic policy costs approximately $45.00 per year per $100.00 under age 50. The average policy cost diminishes from $49.50 for $100.00 monthly benefit to $360.00 for 1,000.00 monthly benefit (that is, $36.00 per hundred) so there is a definite saving in buying larger amounts. The sickness benefit extension rider may be added to this policy, at a cost under age 50 of $4.20 per $100.00 of benefit.

This year, besides extending the maximum coverage to $1,000.00, Mutual of Omaha have added different waiting period policies for sickness. It is now possible for a physician to purchase disability insurance with a 61 day, or even a 91 day "waiting-period." These are significantly cheaper than the policy with a 16 day sickness (or 1st day of hospitalization) "waiting-period" and each physician must decide for himself which of the three policies best suits his needs. These longer "waiting-period" policies before payment of benefits, have been made available to accommodate those salaried physicians who are partially protected by their employer's disability programs. They may also appeal to self-employed physicians who feel they would be able to manage short-term disability from their own resources. There are now 410 members of the society enrolled in the disability income plan.

Office Overhead Expense Protection

Many physicians, when disabled, are burdened with the additional cost of their offices, and this program has been made available to cover this eventuality. It is also underwritten by Mutual of Omaha and is available in amounts from $200.00 to $800.00.

The policy pays for rent, electricity, heat, utilities, salaries of employees, taxes, equipment depreciation and such other fixed expenses as are normal and customary in the conduct and operation of the insured's office. Benefits are paid for as long as 18 months, beginning after a 30 day elimination period up to the amount for which the policy is issued, but cannot exceed the average monthly amount of expenses incurred during the 6 month period prior to the disability.

Premiums are deductible as a business expense, and the premium cost, unlike the Group Life and the Disability Programs, does not increase after policy issue because of increase in age. The cost to a physician joining the program who is under 40 years of age is $20.00 per $100.00 of monthly benefit.

Group Life

This program of level term convertible insurance is carried with North American Life Assurance Company. Level term means that protection remains constant while premiums increase with increasing age. The convertible aspect of the program enables a member to obtain from the insuring company regular whole life or other policies at any time up to age 65 to almost the same amount of the term policy without medical examination.

The main advantage of this type of policy other than the convertible aspect is the high protection at low rates ($3.00 per $1,000.00 per year under 40 years of age) for the young physician when he needs it most. The disadvantage of this specific policy is that as you get older the cost increases significantly ($20.00 per $1,000.00 per year at age 65) until at age 75 it expires altogether. This is a pure protection policy, not unlike the fire insurance on your house, and hence has no cash or savings value. This type of insurance should be used only as a means of protecting your dependents during a period when an estate of sufficient size is being established by other means.

GROUP LIFE INSURANCE PLAN

INTRODUCED IN 1951

SPONSORED BY

THE MEDICAL SOCIETY OF NOVA SCOTIA

UNDERWRITTEN BY

NORTH AMERICAN LIFE  ASSURANCE COMPANY

EFFECTIVE OCTOBER 1 1969 5 UNITS NOW AVAILABLE

FEATURES

Term Insurance to age 75
Liberal settement options
Waiver of premium if disability occurs prior to age 60
Conversion privileges
Assignment of benefits (where permitted by law)
Up to five units now available

ELIGIBILITY

A member in good standing with the Society who is a physician duly qualified and licensed in the Province of Nova Scotia, resident in Canada is eligible provided he is not over 60½ years of age.

SCHEDULE OF INSURANCE AND ANNUAL PREMIUMS
Each unit of insurance has a value of $14,000. This includes a bonus of 40% in effect from 1 October 1967 and until further notice to the Society.

Insurance age at beginning of each Policy Year	Unit Value	Annual Premium
21–30	$14,000.	$ 32.00
31–40	14,000.	42.00
41–50	14,000.	70.00
51–55	14,000.	124.00
56–60	14,000.	188.00
*61–72	14,000.	286.00

*Insurance decreases by 10% and a like amount each year thereafter starting October 1st nearest your 66th birthday, until it expires on the policy anniversary nearest age 75.

The premium increases to the amounts shown on attainment of the ages indicated.

Please make your cheque payable to the North American Life Assurance Co. for the amount shown in the table below according to the month in which you apply and your age bracket (maximum age for entry into the plan is 60½ years). Next premium due date is Oct. 1st.

Please note that the premium shown is calculated from the month following the date of the application. Therefore, make your cheque for the amount shown in the column for the month in which you make the application.

When you have completed this application send it to: North American Life Assurance Co., 105 Adelaide St. W., Toronto 1, Ontario, Association Section, together with your first premium. If a medical exam is needed it will be at no cost to you.

	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.
Ages 21-30	$ 32.00	$ 29.33	$ 26.67	$ 24.00	$ 21.33	$ 18.67	$16.00	$13.33	$10.67	$ 8.00	$ 5.33	$ 2.67
Ages 31-40	42.00	38.50	35.00	31.50	28.00	24.50	21.00	17.50	14.00	10.50	7.00	3.50
Ages 41-50	70.00	64.17	58.33	52.50	46.67	40.83	35.00	29.17	23.33	17.50	11.67	5.83
Ages 51-55	124.00	113.67	103.33	93.00	82.67	72.33	62.00	51.67	41.33	31.00	20.67	10.33
Ages 56-60	188.00	172.33	156.67	141.00	125.33	109.67	94.00	78.33	62.67	47.00	31.33	15.67

The above table is the required premium for one unit. Applications for more than one unit should be increased proportionately.

THIS IS NOT A CONTRACT

This year the amount of insurance available has been raised to $70,000.00 per physician. This represents a sizable increase from previously, and is available in units of $14,000.00 up to five units, or $70,000.00. This increase in coverage was felt to be necessary due to the decreasing value of the purchasing power of the dollar in the past ten years, and we, as a committee, were very pleased at the co-operation of the insurance carrier in making this available.

At present there are 260 members of the Society insured for various amounts, totaling a volume of over $6,000,000. The participation in this group has been increasing rather slowly considering the need and value of this type of coverage.

Conclusion

An outline of the various group insurance programs negotiated by The Medical Society of Nova Scotia has been made in an attempt to acquaint members with the plans available to them. Premium rates quoted are those in effect as of December 30, 1969. In a short article such as this, it will be appreciated that many minor details have been excluded but it is felt by the Insurance Committee that the fundamental features of the three plans have been presented. Any member wishing further information on any of these programs should contact Mrs. Clahane at the Medical Society office in Halifax, or representatives of the insurance companies carrying the specific policies. □

Of Recovering Drowned Persons

"Inflation of the lungs, which may be more conveniently effected by blowing into one of the nostrils, than by introducing air into the mouth. For the former purpose, it is necessary to be provided with a wooden pipe, fitted at one extremity for filling the nostril, and at the other for being blown into by a healthy person's mouth, or for receiving the muzzle of a pair of common bellows, by which the operation may be longer continued. At first, however, it will always be more proper to introduce the warm breath from the lungs of a living person, than to commence with cold atmospheric air. During this operation, the other nostril and the mouth should be closed by an assistant, while a third person gently presses the chest with his hands, as soon as the lungs are observed to be inflated. . ."

So says Mr. Culpeper in his famous *Herbal* - and he goes on to say a lot more, making "the kiss of life" a somewhat complicated procedure. For instance, the requirements he lists for "recovering suspended animation from drowning" are as follows:

"small bottle of rectified spirit of wine.
" " " " white wine vinegar.
" " " " sweet oil.
" " " " white French brandy.
" " " " volatil. sal ammoniac.
" " " " vitriolic ether.
" " " " mustard seed.

A machine for injecting the smoke of tobacco.

A leather tube, together with a pair of bellows, for inflating the lungs.

Another tube of leather, for introducing medicines into the stomach.

A small syringe for clearing the throat of mucus.

Three woollen covers, or blankets.

Four brushes, and six woollen cloths, for performing friction.

Several emetics.

Two lancets for blood-letting.

One pound of tobacco.

A roller and cushion, to be used in venesection.

Two quills, a sponge, and some lint.

A pocket-knife.

An apparatus for striking fire.

Camomile and elder-flowers.

Common salt — and a printed copy of rules and directions for treating the drowned."

While someone is rushing around looking for the book of instructions, it might be more profitable to take up another of Mr. Culpeper's suggestions — that "the body be carried to a brewhouse, and covered with warm grains for three or four hours" — since he obviously has the doctor's welfare in mind when he adds: ". . . these expedients generally require medical assistance". What with the white French brandy and the brewhouse, it might be worth while setting up as a full-time "recoverer of drowned persons"! □

Elizabeth Henry

Shock Associated with Uterine Inversion and Hemorrhage

*Reprinted from The Canadian Medical Association Journal, April 23, 1966, Vol. 94, Page 914**

A 21-year-old married white woman pregnant for the first time was admitted to hospital in labour at term. She had made an unknown number of office visits during the present pregnancy to a physician who had since left the community. The present physician had seen her on only one occasion, a few days before the onset of labour. At this office visit her blood pressure and urine were normal. A complete physical examination was performed and the notation was made that her general state of nutrition was poor. Her weight gain, hemoglobin (Hb.) Rh factor, blood group and serology were not recorded. She had received a vitamin and iron supplement during her pregnancy.

During her labour, which lasted 22 hours, she received 25 mg. of anileridine phosphate (Leritine) on two occasions and 500 mg. of glutethimide (Doriden). At 10:15 p.m. on October 21, 1960, she was given trichloroethylene (Trilene) by the nurse supervisor during her contractions. This was replaced by open ether and at 10:35 p.m. the patient was delivered of a healthy male child weighing 7 lb. 1 oz. in the left occiput anterior position by low forceps after an episiotomy was performed.

At 10:55 p.m. the attending physician expelled the placenta using a type of Crede maneuver. An inverted uterus was immediately recognized and replaced. The replacement required approximately two minutes. It was estimated that the patient lost 400 to 500 c.c. of blood during the uterine inversion and replacement. The patient was given 0.2 mg. of ergometrine maleate (Ergometrine) intravenously at 11:00 p.m. and 1000 c.c. of 5% glucose and saline was started intravenously. At 11:10 p.m. the systolic blood pressure was 70 mm. Hg. and the pulse rate was 130 per minute; 100 mg. of meperidine hydrochloride (Demerol) was then given intramuscularly. There was no excessive bleeding at this time.

The patient was transferred by ambulance to a larger hospital 30 miles away because she was in shock. The attending physician accompanied her in the ambulance, and the intravenous administration of 5% glucose and saline was continued during the transfer.

The patient was admitted to the second hospital two hours post partum, at 12.30 a.m. on October 22. She was examined by a consultant general surgeon, who wrote: "The patient is unconscious and lying in a pool of blood. She is pulseless and the blood pressure is unobtainable." A pelvic examination was performed; the uterus was well contracted and in its normal position. A blood transfusion of 500 c.c. was started at 2:00 a.m. Between 2:00 a.m. and 4:00 a.m. she received 250 c.c. of blood. The transfusion was then discontinued because the intravenous needle had been dislodged from the vein and the blood was running interstitially.

At 2:00 a.m. she was given oxygen and 1000 c.c. of saline subcutaneously. At 3:00 a.m. her pulse was 68 per minute and of poor quality. Her blood pressure was not recorded at this time. At 3:30 a.m. the consultant was notified by telephone that the patient was bleeding. He ordered 10 units of oxytocin (Pitocin) intravenously and 0.2 mg. of ergometrine maleate intramuscularly. At 4:30 a.m. 5 units of oxytocin was given intravenously and 0.2 mg. of ergometrine maleate was given intramuscularly. At this time the nurse noted that the patient was bleeding moderately and her pulse was imperceptible. She had not voided since the delivery. At 6:30 a.m. another 1000 c.c. of normal saline was given subcutaneously and this was repeated at 7:00 a.m. At 7:30 a.m. on October 22 the patient died, six hours after admission to the second hospital. No autopsy was performed.

Decision of Committee on Maternal Welfare

The conclusions reached by the Provincial Committee on Maternal Welfare after a review of this case were as follows: "This was a preventable direct maternal death. The cause of death was shock associated with an inverted uterus and hemorrhage. The preventable professional factors were the management of the third stage of labour and the transfer of the patient 30 miles to another hospital for treatment while she was in shock. There was also a preventable professional factor in the consultant's conduct; the shock was inadequately treated. This maternal death has been considered to be ideally 'preventable' under the terms of reference of the Provincial Maternal Welfare Committee and there is no implication of any negligence."

Discussion

Uterine inversion is a rare complication of delivery, occurring once in 5000 to 30,000 deliveries.

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*This series of articles arranged by an editorial subcommittee of the C.M.A. Committee on Maternal Welfare, and originally published in the Canadian Medical Association Journal, is being reproduced in the Bulletin at the request of the Medical Society of N. S. Committee on Maternal and Perinatal Health, by kind permission of the Editor of the Canadian Medical Association Journal.

The maternal mortality rate varies from 12% to 25%. The prompt recognition and the early replacement of an inverted uterus are associated with the best maternal prognosis. There is a progressive rise in mortality as recognition is delayed up to 48 hours, but if the patient with unrecognized uterine inversion survives beyond this time the mortality drops sharply.

In this case a preventable professional factor was associated with the technique and the amount of force used to deliver the placenta. A classical Crede maneuver to express the placenta—the uterus is squeezed with the hand of the attending physician to produce placental expulsion—should not be used in modern obstetrics.

The Brandt-Andrews method of handling the third stage of labour is recommended. The attending physician waits a few minutes following the delivery of the baby, to permit spontaneous separation of the placenta. Then with the left hand the umbilical cord is grasped near the vulva and the right hand is placed on the abdomen so that the palmar surface of the fingers is over the anterior surface of the uterus, approximately at the junction of the corpus and the lower uterine segment. By gently pressing backward and slightly upward, with the right hand, the corpus is pushed up into the abdomen. If the placenta has separated from the corpus, the cord in the left hand will not follow the upward movement of the uterus. To deliver the placenta and membranes, the operator stops pushing on the corpus with the right hand and exerts pressure over the pubis downward on the lower uterine segment toward the vulva. As this is being done, traction is exerted on the cord and it will bring forth the placenta and membranes. The placenta may also be delivered by continued gentle traction on the cord while the corpus is being pushed up and back.

If the placenta is still attached to the corpus or if it is being held back by a constriction of the cervix, this is manifested by an upward pull on the cord while the corpus is being pushed up and back. In such cases one must wait and repeat the maneuver a few minutes later, after the placenta has separated or the cervix has relaxed.

If the patient is bleeding excessively and placental separation has not occurred, it is better to do a manual removal than to attempt the Crede expression of the placenta. Uterine inversion may occasionally occur spontaneously but in most instances is attributable to the use of the Crede expression of the placenta or to vigorous cord traction. The patient reported here was under deep ether anesthesia during the third stage and uterine atony undoubtedly contributed to uterine inversion.

The attending physician is to be commended for his prompt recognition of the inverted uterus and its early successful replacement. The latter usually requires anesthesia and is best accomplished by inserting the hand in the vagina and with the fundus of the uterus in the palm of the hand exerting pressure with the fingers at the uterocervical junction and at the same time lifting the uterus up out of the pelvis. The tension thus applied to the uterine ligaments will usually replace the uterus in its normal position.

The presence of shock and hemorrhage, severe pain if the patient is awake, the absence of the uterus on abdominal palpation and the presence of a large round mass in the vagina should suggest the diagnosis immediately. At the completion of the third stage of labour, if the position of the uterus cannot be ascertained by abdominal examination because of guarding or obesity, a gentle pelvic examination should always be performed. The initial shock associated with an inverted uterus is due to the stretching of the peritoneum and the nerves of the broad ligaments and may disappear almost immediately following re-positioning. Profuse hemorrhage is the rule, particularly if the placenta has been removed. If the placenta is still attached replacement should be attempted without stripping it off the uterus, but if the mass is too bulky the placenta may have to be peeled off before the uterus can be re-inverted. Great care must be exercised to avoid puncturing the soft atonic uterine wall.

This patient was transferred 30 miles by ambulance in shock, and there was an interval of one and one-half hours between the replacement of the uterus and her admission to the second hospital. It was estimated by the attending physician that she had lost 400 to 500 c.c. of blood during the third stage of labour. However, she was "lying in a pool of blood" when she was seen by the consultant and the total blood loss must have been much greater than that. Once the consultant had established that the uterus was in its normal position and there were no genital tract lacerations to account for the continued vaginal bleeding, the shock should have been adequately treated.

Patients with postpartum uterine inversion who have had successful re-inversion of the uterus may die of shock without excessive blood loss. In retrospect this patient should have remained in the hospital where the delivery and the inversion occurred, and after the uterus was successfully re-inverted the entire birth canal including the uterine cavity should have been re-examined to establish the cause of the continued vaginal bleeding. After the examination the shock should have been adequately treated with massive blood replacement, vasopressor drugs, adrenocortical substances, antibiotics and oxygen. The blood could have been obtained from the second hospital instead of transferring the patient 30 miles in shock. □

Reference

1. **Greenhill, J. P.:** *Obstetrics, 12th ed.*, W. B. Saunders Company, Philadelphia, 1960, 0. 308.

Smoking, Sputum, and Lung Cancer

Chronic bronchitis, as evidenced by chronic cough and daily sputum production over a period of years, was the most significant symptom among men diagnosed as having lung cancer following a mass chest X-ray screening. The relationship between chronic sputum production and lung cancer among tobacco smokers was clear. No cancer was found among nonsmokers.

In connection with a mass miniature radiography screening, the relationship between lung cancer, persistent daily sputum production and tobacco smoking among men 40 years of age or older was investigated. The men were volunteers from the general public and industry.

The subjects were asked whether they smoked or had smoked and, if so, how much they smoked. They were also asked questions about raising phlegm. If sputum had been produced daily for five years, it was considered a symptom of chronic bronchitis. If abnormalities were observed on X-ray film, the individual was referred for further investigation, and six months later a follow-up was carried out. The chest physician who read the original film had no information on the smoking habits and sputum history of the subject.

Lung Cancer Proved

Only volunteers who had lesions confirmed as lung cancer by surgery, biopsy, post-mortem examination, or follow-up were admitted to the study.

Among the 21,579 men in the study, 33 cases of lung cancer were diagnosed. No case was found among nonsmokers. The highest number was among cigarette smokers.

The smoking categories and number of cases of cancer in each were:

Category (*Subjects*)	*Cases*
Nonsmokers (2,826)	0
Cigarette smokers (11,934)	23
Ex-smokers (4,516)	5
Pipe smokers (2,267)	5

The rates for lung cancer among the cigarette smokers rose from 1.35 per 1,000 persons for light smokers to 2.47 per 1,000 for heavy smokers. The rate for the group as a whole was 1.93 per 1,000. The rate for ex-smokers was 1.10 per 1,000. The majority of former smokers, including the five who had cancer, had smoked cigarettes.

Risk Declines for Ex-smokers

These data support findings of other studies that people who had stopped smoking had a lower risk of cancer than current smokers but a greater risk than nonsmokers. Four of the five former smokers who developed cancer had given up smoking only the year before the X-ray examination, which was not the case with the majority of the ex-smokers. It is possible that some of the ex-smokers had developed cancer while still smoking cigarettes.

The rate of cancer among the pipe smokers was high, 2.21 per 1,000. It may be that previous cigarette smokers were among the current pipe smokers.

In 25 of the 33 lung cancer cases, histologic examinations were made. In 14 persons, the cancer was squamous cell; in five, undifferentiated; and in four, oat cell. Two were adenocarcinomas.

Increased chronic bronchitis mortality with increasing amounts of tobacco smoked has been reported. Several studies indicate that chronic bronchitis and productive cough are both related quantitatively to smoking habits, and the frequency of persistent cough with phlegm has been found to be as high as 42 per cent in heavy cigarette smokers aged 55 to 64 years. In the present series, with its five-year minimum of sputum production, the percentages were lower, as was the average age.

Rates Related to Sputum

The rates of cancer per 1,000 subjects in relation to sputum were as follows: smokers without sputum, 1.30; smokers with sputum, 5.09; ex-smokers without sputum, 0.24; ex-smokers with sputum, 10.13; pipe smokers without sputum, 0.97; pipe smokers with sputum, 13.76.

Lung cancer rates for the subjects with persistent daily sputum are considerably higher than those without sputum in each smoking category. This relationship is also maintained among those with histologically proved cancer, 0.63 for those without sputum and 4.78 for those with sputum. The rates are significant in all categories. (Two cancers among cigarette smokers without sputum were adenocarcinomas, which do not appear to be associated with smoking.)

continued on page 209

J. Rimington, M.B., Ch.B. *British Medical Journal*, March 23,1968.
Reprinted from the Abstracts of the National Tuberculosis Association, November 1968.
Printed through cooperation of the Nova Scotia Tuberculosis Association.

Personal Interest Notes

Dr. D. C. Brown was one of twelve doctors awarded certificates in family medicine at the annual convention of the College of Family Physicians of Canada in Toronto in June. He heads the new department of Family Practice associated with Dalhousie University. Six other Nova Scotian doctors were presented with fellowships at the same convention in recognition of their contribution to the College of Family Practice or to their communities. They were: **Dr. F. Murray Fraser,** a Past-President of the College; **Dr. C. L. Gass,** Former Past-President and founding member of the College; **Dr. J. R. MacNeill** of Glace Bay; **Dr. H. C. Still,** past chairman of the College's Advanced Training Committee and member of The College's Board of Examiners; **Dr. H. I. MacGregor,** chairman of the College's Awards Committee and former member of the national executive, and **Dr. H. B. Whitman,** a Past-President of the Nova Scotia Chapter of the College.

Dr. Willard O'Brien was recently given a testimonial dinner on the occasion of his completing 50 years in general practice in the Wedgeport and Yarmouth areas. Presentations were made to him and Mrs. O'Brien by the more than 200 people attending the dinner.

Dr. Richard Hastings-James attended the 12th International Congress of Radiology in Toronto, where he presented a paper entitled "Calcification in Basal Ganglia".

Dr. and Mrs. D. F. MacInnis celebrated their 50th Wedding Anniversary with "Open House" reception at their home in Shubenacadie where they have resided for the past 50 years.

Dr. and Mrs. MacInnis, the former Ann Morrison, were married in the Methodist Church Gabarouse, C. B., on September 29th, 1919, by Rev. William Nightingale. The couple have two children, Ruth, Mrs. C. B. Smith, Halifax and Dr. Ross MacInnis, Shubenacadie. Congratulations are extended by members of the Medical Society.

Refresher Course in Surgery was presented in October under the Chairmanship of **Dr. G. W. Bethune.** Professor of Surgery, and coordinated by **Dr. B. J. Steele.** The Program was sponsored by the Division of Continuing Medical Education.

Dr. S. T. Norvell recently visited Brussels, Belgium, to take part in a symposium on the Use of Computers in Radiology. He presented a paper on "The Application of Computer Analysis to Lymph Node Dosimetry".

Dr. I. A. Karrel and Mrs. Karrel (Nee Helen Garson) were honored at a testimonial dinner given by the Jewish Community in St. John, for their contribution to the Jewish Community and to the Jewish National Fund, and also for services in the St. John community. Dr. Karrel is moving to take up practice in Toronto.

Dr. C. E. vanRooyen and **Dr. James F. Ross** are collaborating in a study of new methods of preserving skin for the treatment of severe burns. Dr. vanRooyen will shortly be attending the National Institute of Health of the U.S.A. and the U.S. Army Medical Centre, Walter Reed Hospital, to observe technical methods for setting up an emergency human skin bank for Nova Scotia.

Dr. Arthur Green, chief of staff, the Glace Bay General Hospital was recently honoured at a surprise function on the occasion of his 70th birthday. Appreciation was expressed for the "responsibility he has shown over the years to the medical staff, hospital staff and patients". Dr. Green joined the General Hospital staff in 1924.

Dr. C. M. Harlow has returned from Vancouver where he attended the annual conference of the North American Association on Alcohol.

Dr. C. H. L. Baker has resigned as Head of the Department of Anaesthesia at the Halifax Infirmary Hospital, Halifax. **Dr. John H. Feindel** has been appointed as the new Head of Department.

Our deepest sympathy goes to **Dr. J. G. Aldous,** Head of the Department of Pharmacology, and his family, on the death of his wife in a motor vehicle accident, October, 1969.

Dr. John Alexander Fraser Young, of Pictou died October 19, 1969 at the age of 58. Dr. Young took up practice in Pictou in 1937. During the 2nd World War he served with the Canadian Navy overseas; after his discharge with the rank of Major he returned to Pictou to resume his practice. He is credited as being one of those instrumental in getting Harris-Sutherland Memorial Hospital in Pictou, built. To his wife and family we extend our deepest sympathy. □

Since both productive cough and lung cancer are associated with cigarette smoking, it is possible that the high rate of lung cancer among the cigarette smokers with persistent daily sputum for five or more years is due to the association of both conditions with cigarette smoking. In view of this possibility the cigarette smokers have been divided into three consumption categories — light, medium, and heavy — but, irrespective of the amount smoked, the lung cancer rates for those with persistent daily sputum are considerably higher than for those without this symptom.

While a very slow-growing lung cancer could cause chronic cough, it is difficult to believe such a cancer could result in excessive sputum production over a long period of time. None of the volunteers with lung cancer who said they had chronic sputum production had such a respiratory disease as tuberculosis or bronchiectasis to account for the sputum. The reasonable assumption is that the sputum was due to chronic bronchitis.

Smoking Plus Cough

In other studies it has been found that the lung cancer mortality of smokers with a morning cough three years prior to death was three to five times greater than that of those without cough.

It seems clearly indicated that anti-smoking propaganda should be directed at the high-risk bronchitic group, and smokers with a chronic cough or sputum should understand that they are at considerable risk of lung cancer. They should be urged to stop smoking and to seek radiologic supervision.

Many might be sufficiently convinced of the seriousness of their situation to give up smoking. As for the others, early radiologic detection of lung cancer can only serve to improve the changes of a surgical cure, slight though that might be. □

News Flashes

NOVA SCOTIAN FIRST

The February 1969 issue of *Health Rays* contains an interesting account of the life and times of Dr. Edward William Archibald (1872-1945), who is thought to have performed, in 1912, the first thoracoplasty to be carried out in the western hemisphere. In July 1921, at the invitation of Dr. A. F. Miller, he came to Nova Scotia and carried out rib resection on a patient from the Nova Scotia Sanatorium. The operation, which was performed in the Victoria General Hospital, resulted in considerable improvement in the patient's condition. A roentgenogram taken at the time is reproduced in *Health Rays*.

LACROSSE — THE CANADIAN GAME

No sport played in Canada has a finer tradition than lacrosse. It deserves to be called "the Canadian game" because its roots are deep in our history.

For many years, before and after the turn of the century, lacrosse was a major sport in this country and the names of lacrosse "greats" were on every boy's tongue. It was played by both English- and French-speaking Canadians, and by the Indians who invented it, so it was truly a national game. Professional and amateur leagues flourished, and the game spread to England, Australia, and the United States; it is still a very popular game in girls' high schools throughout the British Isles.

As the twentieth century advanced, the popularity of lacrosse declined in North America. Now, it is making a comeback here. Lacrosse has much in its favour. It has been called "the fastest game on two feet"; it is a game of skill, not brute force (contrary to general belief), and the little fellow can beat his (or her) larger opponent by superior ability; it can be played from early spring in all kinds of weather; equipment is not expensive; and it is an exciting game to watch. The playing area can be an open field, a hockey box or arena, or even a large gymnasium.

Boys, who can be introduced to the game as early as 8 or 9, enjoy it tremendously. There is something about the stick-work and teamwork, catching and throwing the ball, eluding the opponent and shooting on goal that appeals to the best athletic spirit. As a conditioner, lacrosse is unsurpassed, and some athletic directors feel that this advantage alone justifies its inclusion in their sports curriculum. Because of the very fact that a stick is employed, the game develops self-control.

(Extract from *Lacrosse*, published by the Department of National Health and Welfare)

CANADIAN EXECUTIVE SERVICES OVERSEAS

The Canadian Medical Association advises that an urgent need exists for doctors to participate in the C.M.A. – C.E.S.O. program which is the scheme whereby C.M.A. provides volunteer doctors for the Caribbean for periods of one to two months. The University Hospital of the West Indies needs help! Contact the Canadian Medical Association for more information.

1867 Alta Vista Drive,
Ottawa 8,
Ontario.

IF YOU WANT TO STOP SMOKING, WHAT THEN?

Quitting smoking isn't easy. Some try and don't make it. But you may have noticed that more and more people are trying, and are making it. If you want to be one of the quitters, one thing is certain: it's up to you and no one else. But for some help along the way, here are a few tips from *So, I'm Living Dangerously*, issued by the Department of National Health and Welfare.

A good time to kick the habit is when there is a change in routine — a holiday, a few days in bed with a cold, a business trip — in fact, anything that feels like a fresh start. If you need to stop because of an illness or choose to stop on a holiday, why start again?

List your reasons for stopping and the benefits you expect to gain, so you can refer to them when you feel you might slip.

Stop carrying cigarettes. It will break the automatic habit of reaching for one.

Quit "cold" if you can. If you can't, cut down and consciously stop inhaling. Then gradually reduce until you quit altogether. This is easier when cigarettes are out of reach.

Let others know of your intention. Knowing that others are keeping an eye on you will help strengthen your resolution.

Team up with someone to make the break — especially a husband or wife, boy friend or girl friend. It helps to share your experience.

THE CANDIDATE

Personnel Manager: For this job we want a responsible girl.

Applicant: That's me. Wherever I've worked, if anything went wrong, they said I was responsible.

News Flashes

FORTHCOMING MEETINGS

Dalhousie University is offering the following short courses:

Drug Abuse	December 12 – 13, 1969
Psychiatry	February 5 – 6 – 7, 1970
Pathology	February 12 – 13, 1970

The American College of Physicians announces the following postgraduate courses:

Medicine and Law	January 2 – 4 –, 1970
Function and Dysfunction of the Gastrointestinal Tract	January 2 – 6, 1970
Practical Approach to Selected Clinical Problems	January 26 – 29, 1970
Gastroenterology for Teachers	February 23 – 27, 1970

The Second International Air Pollution Conference of the International Union of Air Pollution Prevention Associations will be held in Washington, D.C., from Dec. 6-11, 1970. The Program Committee invites submission of proposals to present papers at the Conference. Deadline, Jan. 31, 1970.
For more information on any of the above apply: The Nova Scotia Medical Bulletin Office.

MEDICAL STAFF CONFERENCES: CORRECTION

The Department of Therapeutic Radiology and Nuclear Medicine announces that the Nuclear Medicine Conferences are held at 2.00 p.m. in the Therapeutic Radiology Room, The Victoria General Hospital, Mondays to Fridays. Therapeutic Ward Rounds are on Thursdays, originating at 8.30 a.m. from the Department of Therapeutic Radiology, the Victoria General Hospital. The foregoing is a correction to the list of conferences, ward rounds and meetings published in the Nova Scotia Medical Bulletin, Vol. 48 August, 1969.

THE MODERN GENERATION

"Our adolescents now seem to love luxury. They have bad manners and contempt for authority. They show disrespect for adults and spend their time hanging around places gossiping with one another. . . They are ready to contradict their parents, monopolize the conversation in company, eat gluttonously and tyrannize their teachers." (Socrates — 5th Century B.C.)

COLLECTING COLLECTIVES

At a recent meeting of physicians, a group of editors inspired by James Lipton's *An Ecaltation of Larks* invented terms for strings of specialists. Ken Bussy of Lea and Febiger and Eunice Stevens of Hoeber books rivalled Lipton's "a conglomerate of geologists, a shrivel of critics, a persistence of parents" with "a patch of allergists, a bank of haematologists, a brood of obstetricians, a click of cardiologists".

An invitation to readers added a swirl of suggestions from California to Denmark: a rash of dermatologists, a herd of audiologists, a catch of epidemiologists, a pile of proctologists, a smear of gynaecologists, a cast of orthopedists, a mess of dietitians and a crunch of chiropractors. (M.D. of Canada, August 1969).

TO ALL PHYSICIANS

RE: Early and Continued Supervision of Your Pregnant Patients with Rh Incompatability

We continue to lose babies due to Erythroblastosis because their mothers were referred too late in their pregnancy for anything constructive (e.g. intrauterine transfusion or early induction of labour) to be done. This, we feel, is a tragedy and a judgment against our profession. In some of these cases, however, it is the patient's fault for not reporting to her doctor early in pregnancy.

May we respectfully suggest a continued "all out" effort in protecting these unborn? We owe it to ourselves—the Medical Profession of N. S.—and particularly to the unfortunate babies who are at high risk.

A Recommended Outline of Management

1) All pregnant women to be Rh tested - 5 c.c. of blood is all that is needed.
2) Any Rh negative multigravida to have her blood tested for antibodies every month.
3) Any Rh multigravida with Rh antibodies to have an Amniocentesis at the 28th week, or **earlier** if she has a previous history of stillbirths or neonatal deaths or previous babies requiring transfusion at birth.

We are here to help any way we can. Please use us. Phone us (collect) or write:

The Rh Committee
5821 University Ave.
Halifax, N.S.
Phone 422-6501, local 241.

News Flashes

THE GATES OF WRATH

Somewhere between the largest trailer combination and the smallest compact, we find a creature known as the tailgater. Tailgaters come in assorted shapes and sizes, — mostly repulsive. You find them everywhere — but mostly two feet from your rear bumper. Undertakers love them, drivers being tailed hate them and empty highways frustrate them.

He's your terror, your shadow, the cause of your cursing, your constant too close companion on the road. But when he finally turns off at a tavern, he's a soothing vacant space behind your vehicle, a toothache that's stopped hurting, a feeling of safety in the world. (American Society of Safety Engineers).

TALE WITHOUT END

Doctor: "How's the little boy who swallowed the half-dollar?"

Nurse: "No change yet". (Baltimore Sun)

PLAYTIME

"I do hope the weather is nice today," said one lady kangaroo to another: "I just hate it when the children have to play inside."

ONE MINUTE COMMERCIAL

A man got hungry during the night and went down to the Kitchen. As he opened the refrigerator door he was surprised to see a cute little rabbit leaning on its elbow, calmly smiling up at him.

"Hewwo", said the rabbit.

"Hello yourself. What on earth are you doing there?" asked the man.

"This is a Westinghouse, isn't it?" said the rabbit.

"Yes".

"Well, I'm Westing". (From Health Rays, June 1969.)

ADVERTISER'S INDEX

CANADIAN COUNCIL ON HOSPITAL ACCREDITATION

30 Bloor St. West, Toronto 5, Ontario

Early in 1970, the Canadian Council on Hospital Accreditation will expand its programme to encompass accreditation of Extended Care Centres across the country. Commenting on this wider scope of activities, Dr. R.S. Duggan, Board Chairman of the C.C.H.A. stated: "For sometime, Council has felt that the advantages and values of accreditation should be made available wherever personal health care services are offered. Our entry into this field is recognition of the growing proportion of care rendered by institutions and agencies other than acute general hospitals. We are indebted to the W.K. Kellogg Foundation for their encouragement and their support in the form of a substantial grant to prepare a programme and to implement it".

This new program, like the well established accreditation programme in the acute general hospital field, is a voluntary programme, open to institutions and agencies offering health care to patients whose stay is over an extended period of time. Hospitals for the chronically ill, convalescent hospitals, nursing homes, home care agencies and a variety of service organizations that span or include these kinds of care may become eligible on application. The common element of all extended care centres is adequate and necessary medical and nursing care with appropriate supportive services. Evaluation will recognize the differing emphasis of this broad range of health services from active restoration to essentially domiciliary care.

Work on this project has been underway since May 1968 when it began under the aegis of Dr. W.I. Taylor. Dr. Michel Gingras, now Medical Director of Jean Talon Hospital in Montreal, was Project Director and was ably assisted by Miss Nicole DuMouchel, Nursing Consultant of the Council. The invaluable assistance of many active practitioners in the extended care field made possible the development of STANDARDS in a relatively short period of time. In addition, an Accreditation Guide Book for Extended Care Centres has also been prepared for interpretation of these Standards. Both will be available before year end.

ELIGIBILITY

To be eligible for accreditation, a centre must meet the definition of an extended care (health) centre, must possess a current operating licence, have been in operation under the same ownership not less than six months prior to survey and submit an application form containing basic information to Council.

FORMAT OF ACCREDITATION

The accrediting process will follow essentially the same pattern as that of the acute general field. A survey date is assigned to the eligible applicant some 4 to 8 weeks before the actual survey visit. A survey report is prepared before the arrival of the surveyor (or survey team) to provide a background of basic and current information necessary for accreditation. An experienced nurse surveyor will take part in each survey and will be assisted in selected situations by a doctor or by an administrator. After appraisal

(over).....

of the completed report by the executive office and the Board, the centre will be notified of the accreditation status awarded. An attractive certificate indicating achievement of accreditation will be given for public display. Each participating institution will receive a report of the visit containing comments and appropriate recommendations. Accredited centres will be visited every three years unless some important issue requires earlier re-assessment. Provisionally accredited centres are re-surveyed in one year. Non-accredited may seek re-survey when they feel ready for re-assessment. (Field surveys could begin in late February or early March and will extend throughout the year.)

Fee-for-Survey

Organization and start-up costs will come from the Kellogg grant and from the Council. The on-going programme will be self-supporting from fee-for-survey charges to the Centres entering the programme. These will range from about $300.00 for the small Centres to near $500.00 for the larger ones.

There are some 3,000 Centres in Canada whose field of action is long term care. A modest target of at least 100 has been set for 1970 and present indications suggest that this total will be exceeded. The programme will be directed from the offices of the Canadian Council on Hospital Accreditation at 30 Bloor Street West, Toronto 5, Ontario.

L.O. Bradley, M.D.,
Executive Director

Definition of extended care centre:

An extended care centre, which may operate under voluntary, proprietary or governmental auspices, is one that provides the necessary nursing and medical care with other required services as well as personal assistance with the acts of daily living. These centres may be institutional such as nursing homes or extra institutional such as home care.

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