THE NOVA SCOTIA MEDICAL BULLETIN

"... to comfort always"

In this issue of the Bulletin and in the next, two respected general practitioners give personal accounts of their own illnesses. Although they write in the column "Around the Willow Tree" — a column designed to amuse rather than educate — this is not to minimize the significance of their contributions. While the adage "Physician, heal thyself" is known to us all, perhaps at times we forget its deep significance; and to be the complete physician it is essential that we achieve insight into illness and all that it entails, a process which perhaps is most truly done the hard way, through personal knowledge of illness and suffering. These lighthearted contributions are the bubbles on the surface of a draught of medicine, which has surely influenced their authors.

Some years ago The Lancet[1] published a collection of essays by doctors on their own disabilities. Some were most moving, creating as they did lasting impressions of the authors' courage and humanity. Such accounts, as well as the exemplary fortitude which we recognize in our own colleagues who come to suffer, do affect us profoundly in an intangible way which goes beyond words. What true insight these men and women must surely gain!

It is perhaps fortunate that we do not all have to swallow such bitter medicine in order to understand and empathize with our patients, for not all of us would be ideal patients. It is all the more important that from time to time we take stock of the ways in which we can alleviate suffering, particularly the ways in which we can lessen the discomforts of illness in hospital. For hospitalization is the entry to a stranger's land.

For those of us who work largely within hospitals, this is a constant challenge. One hears all too often standard complaints from patients, yet how few of us act on them. We recognize the impersonal environment of the hospital, but how few of us attempt a colourful and sympathetic physical environment, be it with mural, mosaic or music. We know that hospitals are strange and forbidding and frightening, yet how few of us remember to show tact and consideration in corridors, elevators, operating-rooms and recovery areas, where jostling and jocularity are not unknown and where quietude is too often absent. We realize that our patients complain about block-bookings for appointments with the inevitable wastage of time, but how few of us try to obviate this. We hear *ad nauseam* the criticism that doctors do not explain to their patients what is about to happen to them, and yet it is still common to find on the eve of surgery that patients really have not been told about an operation scheduled for the next morning.

Perhaps these are relatively insignificant causes for complaint when considered in the totality of medical practice, but they do add up to an impersonal whole, to an outlook which, with a little effort, could be ameliorated. Perhaps we are unwilling to change our routine in what is, for us, familiar territory and which sometimes must seem to be arranged primarily for our benefit rather than for our patients'. And yet what tremendous benefits could accrue from a change of attitude.

A particular need for a sympathetic attitude arises during the management of children in hospital, frequently a poignant concern and one which affects the emotional behaviour in later years. Fortunately the importance of liberal visiting time in children's hospitals has become widely recognized, as has the provision of in-hospital accommodation for mothers during the period of their children's hospitalization. It is commendable and of local interest that consideration has been given to this during the planning stages of the newest hospital in the Atlantic Provinces, The Izaak Walton Killam Children's Hospital in Halifax.

Is it too much to want to go even further? For there are intriguing examples which point the way for those who are concerned. One such is a recent study of the beneficial effect of the presence of the child's mother at induction of anaesthesia,[2] a study which has confirmed the personal impressions formed over the years by some anaesthetists and paediatricians. This is a relatively small area but it is one which underlines the value of perhaps an unorthodox approach to humanity in hospitals; there must be surely many others.

Progress is sometimes the apparently impossible breaking of barriers, often within ourselves. At least let us pose the questions and let us look beyond the horizons of our limited experience to find out how far we can follow the maxim, "To cure sometimes, to relieve often, to comfort always."

D.A.E.S. □

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Appreciation

Hugh Edgar Kelley

Edgar Kelley was born in Yarmouth in 1902, and there obtained his high school education. He graduated from Dalhousie University in 1922 with a B.Sc. degree. In 1926 he was awarded his medical degree, also from Dalhousie, and then served for a year as the Staff Anaesthetist at the Victoria General Hospital.

Following this, he served on the H.M.C.S. Nascopie for eighteen months during the first Hudson's Strait Expedition.

On his return from the North he settled in Middleton, November 1928 to do a General Practice.

In 1929 he married Anna Morris who became a devoted wife and Mother of two daughters, Eileen and Marsha.

With his death in April 1969, we in the Valley area lost one of the truly great general practitioners of our time. He gave long and faithful service to this area, and will always be remembered as a devoted family physician with a special interest in obstetrics.

During his practice in Middleton he delivered over 5000 babies with an incredible ease which astounded his colleagues.

Blessed with a phenomenal memory he was a constant source of reference for his patients and friends.

There are many, many anecdotes about Edgar Kelley that his friends could tell. Those of us who were close to him over the past years were privileged and we have been the better for it. He understood people, and loved them, and had a tremendous circle of friends. His kindness and thoughtfulness will be sadly missed, but never forgotten.

In his company, the world was a better place. □

The Physiology of the Pulmonary Circulation and Pulmonary Hypertension

JAMES H. HALDANE, F.R.C.P.(C), F.R.C.P. (Glasg.), M.R.C.P. (Edin.)

Halifax, N. S.

PHYSIOLOGY

Normal man, when oxidising 3,000 calories of food per day, inhales 600 litres of oxygen and some other gases and exhales 480 litres of carbon dioxide. Through the action of haemoglobin, oxygen is abstracted from the air into the pulmonary circulation and is delivered to the tissues at almost the same pressure as in the alveoli. The carbon dioxide produced daily in the tissues becomes carbonic acid in an amount equivalent to two litres of concentrated hydrochloric acid; this is eliminated as carbon dioxide through the lungs with very little change in the pH of the blood.

During normal breathing, the tension of oxygen (Po_2) in alveolar air is 100 mm. Hg and in the venous blood is 40 mm. Hg, the oxygen diffusing across this concentration gradient. The red cell remains in the pulmonary capillary circulation for only 0.8 seconds but, due to the high affinity of haemoglobin for oxygen, equilibration with alveolar oxygen is almost complete within this time. Each 1.0 g. of haemoglobin combines with 1.34 c.c. of oxygen, so that each 100 ml. of blood contains 20.1 c.c. of oxygen; a small amount of oxygen (0.3 c.c. per 100 ml. of blood) is transported in solution. The sigmoid character of the dissociation curve of oxyhaemoglobin is such that arterial haemoglobin is virtually saturated at a Po_2 as low as 80 mm. Hg. This factor is important in hypoventilation states in which the Po_2 level is decreased and carbon dioxide continues to enter the alveoli, causing increase in the Pco_2. Normally, the Pco_2 is 40 mm. Hg in the alveoli and 46 mm. Hg in the capillary blood; carbon dioxide diffuses across the alveolar capillary membrane and is eliminated in the expired air. The elimination of carbon dioxide is important in maintenance of acid-base balance: hypoventilation results in respiratory acidosis; and hyperventilation, with its resultant decrease in alveolar Pco_2, leads to respiratory alkalosis - which occurs also as a compensatory mechanism in metabolic acidosis. To maintain efficiency in these functions, adequate perfusion of the lungs is essential, as well as adequate ventilation.

Pulmonary Circulation in the Foetus

Angiography has shown that oxygenated blood from the placenta passes to the inferior vena cava via the ductus venosus, passing mainly through the foramen ovale to the left atrium, left ventricle, and aorta; and the supply from the superior vena cava passes almost entirely into the right ventricle and pulmonary artery. Most of the blood from the pulmonary artery passes through the ductus arteriosus to the aorta but some flows through the lungs, so that pressure in the pulmonary artery and in the aorta is equal. It seems probable that the ductus arteriosus remains open normally for three days after birth; however, because the aortic and pulmonary artery pressures are similar, flow within the ductus is slight and thrombosis occurs. By the third month of extra-uterine life 95% of the ducts have closed.

The arteries in the foetal lung are structurally similar to those in the sytemic circulation. The media of the elastic pulmonary artery and of the aorta are of similar thickness and those of the smaller pulmonary arteries are thick-walled and muscular, thereby giving rise to the high pulmonary artery pressure that is present in the foetus. This pressure falls to the adult level in the first month of extra-uterine life.

Pulmonary Circulation in the Adult

The pulmonary vasculature extends from the pulmonary valve to the orifices of the pulmonary veins in the wall of the left atrium. The arterial trunk arises from the right ventricle as a short tube, 3 cm. in diameter and 6 cm long; it passes upwards and backwards in front of, and then to the left of, the ascending aorta; in the concavity of the aortic arch it divides into a right and a left pulmonary artery. The left pulmonary artery is connected above to the concavity of the arch by the ligamentum arteriosum, which is the remains of the ductus arteriosus. The arteries branch repeatedly and accompany the branchings of the bronchial tree.

The *pulmonary arterioles* are less than 100 microns in external diameter. Their walls consist of an endothelial lining and a single elastic lamina, with no muscular media and almost no adventitia, and their structure is identical with that of a pulmonary venule. The *inter-alveolar septum* contains pulmonary capillaries with endothelial walls, a basement membrane, and pores which allow communication between alveoli. The alveolar capillaries

and lining cells are supported by many delicate reticular and collagen fibrils and by a few coarse elastic fibres which prevent over-expansion of the lung. The *pulmonary capillaries* form a very rich vascular network; only a small proportion of them are open at any one time. The *pulmonary venules* pass into the connective-tissue septa between secondary lobules to enter pulmonary veins distant from the bronchial tree. *Pulmonary blood vessels* are supplied by sympathetic and parasympathetic nerves.

The pulmonary lymphatic ducts are lined by endothelium and their walls are composed of elastic fibres and smooth muscle. The superficial ducts are in the pleura and the deep ones are around the bronchi, pulmonary blood vessels, and in the septa between the secondary lobules. The two systems anastomose in the pleura and at the hilum and drain into the tracheobronchial lymph nodes.

Pulmonary Haemodynamics

The basic physiological measurements are those of pressures in the blood vessels, blood flow, and blood volume in the lung.

Pressures. — The mean pressure in the pulmonary artery as measured by cardiac catheterisation is 15 mm. Hg (22/10 mm. Hg). The mean pulmonary arterial wedge pressure (*i.e.*, the pressure at which the catheter's advance into the lung is halted by impaction) is 7 to 15 mm Hg. It is usually comparable with but a few millimetres higher than, the pressure in the left atrium. Increase in the wedge pressure is present in cases of pulmonary arterial hypertension secondary to pulmonary venous hypertension.

Blood Flow. — The average normal pulmonary blood flow in adult man totals 3.5 to 4 litres per min. per square metre of body surface, varying, even in the same person, from 2 to 5 litres per min., and declining gradually from childhood to old age. It is increased during exercise, digestion pregnancy, anxiety, and fever. In upright subjects this pressure is 27% less than in those lying down and, because its mean pressure (15 mm. Hg) has to offset the hydrostatic pressure of a column of blood 15 cm. high, cannot adequately perfuse the lung apices. The uptake of radioactive carbon dioxide by the alveoli is approximately 20% at the lung bases and 2% at the left apex. It has been reported that the physiological dead space increases by an average of 83 ml. on standing (*i.e.*, that approximately one-seventh of the total number of alveoli become non-perfused), but this finding has been challenged.[1a] In mitral stenosis, the rate of perfusion is greater in the apices than in the lung bases, because of the greater severity of arterial disease in the lower lobes.

All methods to determine pulmonary blood flow depend basically upon the same principle. A known amount of tracer substance — which may be oxygen, nitrous oxide, or a dye — is introduced into the circulation and its concentration is measured upstream and downstream from the point of entry. The technique most commonly used (the *Fick method*) requires the measurement of three parameters: the oxygen content of systemic arterial blood (arterial needle); the oxygen content of blood in the pulmonary artery (cardiac catheterisation); and the rate of oxygen uptake by the lungs (calculated from analysis of a three-minute collection of expired air). With the *dye method* the contrast medium (Evans blue; Fox green) can be injected via a cardiac catheter into the superior vena cava, right atrium, or pulmonary artery. Blood is sampled continuously from a brachial artery and is drawn through a photo-electric densitometer. With *body plethysomgraphy*, which was introduced by Lee and Dubois,[2] the subject sits in an air-tight box and the pressure inside the box is measured by a sensitive manometer and is recorded graphically. Newer methods of measuring cardiac output and blood flow to the individual lobes of the lung employ radioactive xenon and krypton.

Blood Volume. — The pulmonary component approximates 10-20% (323-365 ml./m.^{2}) of total blood volume and is extremely susceptible to even slight differences in output of the two ventricles. Thus, if the stroke output of the left ventricle persistently exceeded that of the right ventricle by even 0.1 ml., the lungs would become exsanguinated within two hours. The controlling homeostatic mechanism is probably situated in the myocardium. Lammerant[3] measured pulmonary blood volume with a precordial counter which recorded the arrival of intravenously injected albumin-tagged ^{131}I at the right and left sides of the heart. This method records two peaks from which separate primary curves may be drawn, representing dilution curves of the right and left sides of the heart; the difference in mean circulation time of the two curves, which is the mean circulation time between the two sides of the heart, is multiplied by the cardiac output to give the pulmonary blood flow.

The volume of blood in the pulmonary capillaries is approximately 80 ml. in normal man. It may be calculated from measurements of the CO-diffusing capacity of the lungs at different concentrations of inspired oxygen.

Bronchial Circulation

Normally, the lung receives blood at low pressure through the pulmonary arteries and at high pressure through the bronchial arteries. The main or true bronchial arteries arise from the aortic arch, usually one inch distal to the left subclavian artery but occasionally from the intercostal, or internal mammary arteries. They pass along the posterior aspect of the bronchus, following the bronchial tree and closely adherent to its walls. There are two bronchial arteries to each bronchus; they branch and anastomose repeatedly to form one network around the bronchi and another supplying the submucosa

and have the structure of systemic arteries. The deep bronchial veins arise as tiny radicles in the walls of terminal bronchioles and form large trunks; they communicate freely with the pulmonary veins, which drain into the left atrium.

PULMONARY HYPERTENSION

Definition

Normal pulmonary artery pressure is 22/10 mm. Hg, and pulmonary hypertension exists when these levels are persistently exceeded.

Causes

I.—*Physiological adaptation.*

II.—*Quantitative differences in blood flow.*

(a) Pre-tricuspid shunt, due to any form of interatrial communication or to anomalous pulmonary venous drainage into the right atrium or superior vena cava. (b) Post-tricuspid shunt, due to large ventricular septal defects alone or associated with other congenital defects (*e.g.*, corrected or uncorrected transposition of the great arteries); persistent ostium primum; widely patent ductus arteriosus; a large aorta-pulmonary septal defect; ruptured aneurysm of a sinus of Valsalva; and persistent truncus arteriosus.

III.—*Prolonged elevation of left atrial pressure*, due to mitral stenosis (rheumatic or congenital); mitral incompetence (rheumatic or secondary to subacute bacterial endocarditis); cor triatriatum; compression of the pulmonary vein by a myoma of the left atrium or mediastinal tumour; or, occasionally, secondary to aortic stenosis, aortic incompetence, or systemic hypertension.

IV.—*Chronic lung disease.*

V.—*Recurrent pulmonary thrombo-emboli.*

VI.—*Congenital or acquired kyphoscoliosis.*

VII.—*Idiopathic pulmonary hypertension.*

VIII.—Other (rare) conditions.

Pathology

In cases of shunt-induced increase in flow, persistent elevation of the pulmonary artery pressure causes various types of progressive change in the structure of the small pulmonary arteries, from foetal-type arteries (in cases of post-tricuspid shunts) to medial hypertrophy, with cellular intimal proliferation, fibrous vascular occlusion due to intimal fibrosis, general dilatation with thinning of media and (rarely) necrotizing arteritis. At the same time, the elastic arteries (more than 1000 microns in diameter) become dilated, with thickened media and the depositon of atheroma. Atherosclerosis is characteristic of all conditions associated with pulmonary hypertension.

In cases of large post-tricuspid shunts, the pulmonary blood flow is increased from birth and the thick, foetal-type blood vessels respond to the mechanical stimulus by constriction, so that pulmonary hypertension is present at birth and the vicious circle of hypertension and constriction progresses to an advanced stage in early infancy. Pretricuspid shunts (*e.g.*, atrial septal defect) do not give rise to pulmonary hypertension until later in life, since the pulmonary blood flow is not greatly increased in early infancy. The direction of flow through an atrial septal defect depends upon the compliance or distensibility of the ventricles. Since the walls of both ventricles at birth are of equal thickness and, therefore, have a similar degree of compliance, blood does not tend to pass preferentially into the right ventricle. Thus, the walls of the pulmonary vessels undergo involution in the normal way and are thin-walled when left-to-right shunt occurs later.

When the hypertension is associated with intracardiac shunts the vascular resistance increases progressively until it reduces the shunt and finally, when pulmonary artery pressure exceeds systemic pressure, reverses it. These cases show marked changes in the structure of the pulmonary arteries. Lung biopsy may be helpful in confirming the degree of severity of advanced vascular changes.

Clinical Features

I: Physiological Pulmonary Hypertension.—Hypoxia causes constriction of the blood vessels in the lungs and raises pulmonary artery pressure, and in 1946, von Euler[4] suggested that it caused local lactic acidosis and thereby stimulated constriction of the arteriolar wall. Because of the low oxygen tension at high altitude, pulmonary hypertension develops in people who live in such areas and the foetal muscular pulmonary arteries do not undergo the usual rapid regression in those who are born there.

II: Quantitative Differences in Blood Flow.—There are three phases in the evolution of pulmonary hypertension secondary to intracardiac shunts, characterized by excessive, normal, and reduced pulmonary flow.

Increased Flow.—The two main groups of patients are infants and children with large post-tricuspid shunts (Group 1), and children and adults with large pre-tricuspid shunts (Group 2).

Group 1: The pulmonary vascular resistance is lower than that of the sytemic circulation, so that blood pours preferentially through the lungs. Infants fail to thrive and may die at this stage, although emergency surgery to put a constricting band around the pulmonary artery may save them until they are fit to undergo correction of the defect. The main symptom is dyspnoea. There is a precordial pansystolic murmur, a third heart sound, and a flow murmur during diastole. Roentgenography shows enlarged pulmonary arteries and fluoroscopy reveals their increased pulsation, and ECG shows left ventricular hypertrophy. In infants who survive there is progressive increase in pulmonary vascular resistance, thereby effecting reduction in blood flow.

Group 2: The adult with pre-tricuspid shunt but without advanced pulmonary hypertension is moderately breathless on effort but can do a day's work, and is very susceptible to respiratory infec-

tion. Cyanosis and finger-clubbing are absent. Outward movement of the chest wall to the left of the sternum is visible during systole. The second sound may be loud and palpable in the pulmonary area and is widely split due to delay in activation (right bundle branch block) and in emptying of the right ventricle; it is fixed, respiration having no effect on the two atria. A pulmonary systolic ejection sound, due to stretching of the wall of the dilated pulmonary trunk, may be heard, and the increased flow through the pulmonary artery gives rise to a pulmonary ejection murmur during systole. Enlargement of the pulmonary artery and its branches, the right atrium and right ventricle is apparent on the roentgenogram, and fluoroscopy shows hilar dance. ECG may reveal partial or complete right bundle branch block.

'Normal' Flow.—In patients with shunts, during this stage of equal pressures their condition may appear improved, with fewer attacks of breathlessness.

Diminished Flow.—The intracardiac shunt becomes predominantly right to left as pulmonary vascular resistance exceeds systemic resistance. This usually occurs before the end of the second decade in patients with post-tricuspid defects and in the third-fifth decade in those with pretricuspid defects. The main presenting complaint is dyspnoea on exertion. In about 30% of cases haemoptysis occurs from rupture of thin-walled branches of muscular pulmonary arteries and, rarely, an enlarged pulmonary artery may press on the recurrent laryngeal nerve and produce hoarseness and aphonia. In some cases, effort provokes retrosternal pain, not relieved by nitroglycerine, that may be due to ischaemia of the right ventricle, or may give rise to syncope. Reversal of the shunt results in cyanosis and, in severe cases, finger-clubbing. The legs, and sometimes the left arm, may become cyanosed in cases of patent ductus arteriosus.

The enlarged right ventricle causes a systolic heave situated to the left of the sternum, and there may be a large 'a' wave in the jugular venous pulse. The second sound in the pulmonary area is loud and palpable and may be closely split, and an ejection click may be audible during systole. Although the pulmonary systolic flow murmur may disappear, a Steell diastolic murmur, due to incompetence of the pulmonary valve, remains.

Roentgenography may reveal diminished vascular markings in the periphery of the lung fields. The pulmonary arteries and right vencticle are enlarged, as is the left ventricle also in cases of post-tricuspid shunt. The tracheobronchial tree may be distorted by the enlarged pulmonary arteries.

Pulmonary hypertension due to intracardiac shunts should be treated surgically, by the closure of septal defects, before the vascular changes become irreversible.

III: Prolonged Increase in the Left Atrial Pressure (which may reach 40 mm. Hg) results in pulmonary venous hypertension.

In the early stages the lung is congested and the capillaries are distended and may rupture; macrophages ingest the haemosiderin and often become grouped subpleurally to produce the picture of pulmonary haemosiderosis. It is believed by some that the bleeding originates from small distended pulmonary veins and by others that it occurs from bronchopulmonary anastomoses. There is a gradual change from haemosiderosis to siderofibrosis, with iron impregnation of elastica and reticulin in the lung and resultant foreign-body giant-cell reaction. The alveolar spaces and walls are oedematous; the oedema coagulate becomes organised, and leads to the formation of fibrosis and thickening of the walls and ossification of the spaces.

In cases of mitral stenosis the pulmonary blood flow is decreased and exercise causes increases in the wedge pressure and left atrial pressure.

Limitation in the cardiac-output response to exercise may prevent the left atrial pressure reaching levels that would cause pulmonary oedema. The chest roentgenogram may show Kerley's lines, the horizontal markings at the lung bases which are said to be caused by co-existent oedema of septal connective tissue and distension of the lymph ducts and which are apparent when the mean left atrial pressure exceeds 24 mm. Hg. Increases in the pulmonary artery and pulmonary venous pressure are disproportionate, thereby creating a protective mechanism which prevents elevation of the pulmonary capillary pressure above the plasma's osmotic pressure.

IV: Chronic Lung Disease.—When pulmonary emphysema is present, the abnormalities in structure of the muscular pulmonary arteries are of limited importance in altering flow and pressure. The pulmonary artery pressure may be normal in emphysema but usually is raised during infection or in congestive heart failure. The capillaries are distributed irregularly in the alveolar walls, and chronic lung disease may result in a decrease in their numbers in many areas and dilatation elsewhere; also, mechanical forces may distend one part of the lung and compress adjacent parenchyma and blood vessels, resulting in much variation in structure in different areas. The ventilation/perfusion ratio (normal, 4/5) is disturbed, the degree of alveolar perfusion varying from normal to nil.

The most characteristic vascular lung lesion in pulmonary emphysema is intimal proliferation, which is restricted to and may block thin-walled bronchiolar arterioles. The total area of the pulmonary vascular bed must be reduced by more than 50% before the pulmonary artery pressure becomes significantly elevated. When interstitial fibrosis develops, some pulmonary capillaries are closed off.

In the Hamman-Rich syndrome, whether acute or chronic, the alveolar capillary membrane is greatly thickened as a result of enlargement of the lining alveolar epithelial cells, the formation of a hyaline membrane, and oedema and fibrin deposits in the alveolar walls. There is diffuse, progressive interstitial proliferation of fibrous tissue within the lung.

Other causes of interstitial fibrosis are sarcoidosis, tuberculosis, berylliosis, asbestosis (and other diseases of occupation), irradiation of the lungs, scleroderma, xanthomatosis, eosinophilic granuloma, and lymphangitic carcinomatosis.

Acute, transient thickening of the alveolar capillary membrane occurs in cases of inflammatory pulmonary oedema following the inhalation of irritant gases, and in pulmonary congestion complicating left ventricular failure or mitral stenosis.

Massive pulmonary fibrosis occurs due to the prolonged inhalation of coal dust or silica, or of other dusts encountered in industry, chiefly those containing iron, talc, or gypsum. The capillaries and muscular pulmonary arteries are destroyed by fibrous tissue and the elastic pulmonary arteries become obstructed at the edge of the fibrous mass. In all of the pneumoconioses there is a great proliferation of fibroblasts in the alveolar walls, with destruction of pulmonary arteries.

V: Recurrent Pulmonary Thrombo-Embolism.—Massive pulmonary embolism from a thrombus in the femoral or pelvic veins or, in a small percentage of cases, the right side of the heart, usually leads rapidly to death. However, a patient may survive acute impaction of a large embolus in a tertiary branch of the pulmonary tree; the embolus becomes thrombosed and recanalised but eventually causes pulmonary hypertension and congestive cardiac failure. Smaller emboli lead to pulmonary infarction but sometimes are recurrent and silent, and showers of small, even minute emboli may pass into the pulmonary circulation and produce elevation of the pulmonary artery pressure. The small embolus becomes invaded with polymorphs, lymphocytes, plasma cells and fibroblasts, and eventually is replaced by fibrous tissue; this may shrink and appear finally as a thickening of the intima. There is widespread obstruction of the pulmonary vascular bed, with medial thickening of the small pulmonary arteries. Mean pulmonary artery pressure may be as high as 60-70 mm Hg, with a normal wedge pressure. The arterial oxygen saturation is only slightly diminished and small emboli have no effect on P_{CO_2}, but end-tidal P_{CO_2} may be 15 mm. Hg lower than arterial P_{CO_2} when a large embolus is present.

The symptoms and signs are those of pulmonary hypertension, with progressive dyspnoea and an unproductive cough, and syncope may ensue during exertion. Haemoptysis and pleuritic pain seldom occur. The heart signs are those of pulmonary hypertension. Usually there are no abnormal physical signs in the chest and no roentgenographic signs of past or present pulmonary infarction. Although lung scan may indicate the diagnosis in cases of recent or massive emboli, it is not helpful in the syndrome of pulmonary hypertension resulting from repeated small emboli. Similarly, the ECG shows only right ventricular hypertrophy and 'p' pulmonale.

These patients should be given anticoagulant therapy, in an attempt to halt progress of the disease.

VI: Congenital or Acquired Kyphoscoliosis alters the shape of the thoracic cage and invariably decreases both total lung volume and vital capacity; these latter values may be only one-third the predicted value. Airway resistance and diffusing capacity are normal. Ventilation/perfusion ratios are disturbed[1b] and in most cases inert-gas distribution is impaired. The main effect of these changes is the production of mild hypercapnia and a moderate degree of arterial desaturation. Patients with kyphoscoliosis usually have dyspnoea on exertion for years before any abnormality of blood gases is found at rest.

VII: Idiopathic Pulmonary Hypertension.—This condition was first suspected in 1891 when Romberg reported a case of right ventricular hypertrophy and pulmonary arterial sclerosis found at necropsy, with no apparent causal lesion. Clinically, these patients have the signs and symptoms of severe pulmonary hypertension with decreased pulmonary blood flow. The arterial changes are similar to those found with congenital cardiac shunts, and roentgenography shows enlargement of the pulmonary artery with relatively clear peripheral lung fields. The diagnosis may be made by finding a high pulmonary artery pressure on catheterisation and by the exclusion of other causes. The disease occurs predominantly in females.

The cause is unknown. In some cases it is first detected immediately after pregnancy and then may be due to amniotic emboli in the pulmonary circulation. Repeated pulmonary thrombo-emboli are commonly thought to constitute the prime etiological factor in the majority of cases; reactive intimal fibrosis over multiple foci of medial aplasia or hyperplasia is another possibility, and some have suggested that the pulmonary vascular resistance is abnormally elevated from birth, even though clinically the condition appears to start in late childhood or early adult life. The pulmonary hypertension is intractable and leads to congestive cardiac failure and death within three years of onset. Treatment with anticoagulants has been advocated because of the possibility that thrombo-emboli are concerned in the aetiology.

VIII: Other (Rare) Conditions.—Pulmonary hypertension has been reported in association with pulmonary schistosomiasis, rheumatoid arth-

toid arthritis, progressive systemic sclerosis, Hand–Schüller–Christian disease, and sickle-cell anaemia.[5]

SUMMARY

The lungs require adequate perfusion with blood to carry out their main function (gaseous exchange). The foetal circulation is described and the factors affecting pulmonary circulation in the adult are examined, including cardio-pulmonary resistances, arterial blood flow, blood pressure, and blood volume. The bronchial circulation is described briefly.

The causes of pulmonary hypertension, and the pathology and clinical features are discussed. □

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PREVENTION OF Rh DISEASE
Nova Scotia 1968 - 1969

Women are Rh-sensitized by small transplacental bleedings. The size of the hemorrhage is proportional to the degree of sensitization.

Since we know that antibodies form because the Rh Positive cells of the foetus enter the blood of the mother, it had to be shown that the Rh Immune Globulin, manufactured or extracted from plasma of women who had high antibody titres, destroyed Rh Positive cells which had leaked across the placenta into the mother's blood stream immediately after the termination of pregnancy.

Method of Proof

1/10 ml. of Rh Positive cells was injected intravenously into Rh male volunteers. This was followed by an injection of Chromium 51 as a tracer for the Rh Positive cells. 0.5 ml. Rh Immune Globulin was then injected intramuscularly, and it was demonstrated that the Rh Positive cells were destroyed. Therefore, it was proven that the Immune Globulin could prevent sensitization.

The Clinical Application:

There are about 15,000 births in Nova Scotia per year, of whom 900 are at risk *from* Rh Disease. If the criteria are broadened to include ectopics and abortions, the total number of women in the Province who qualify for Rh Immune Globulin would be about 1,200.

1. Any Rh Negative woman with no demonstrable antibodies, and with an Rh Positive mate, who gives birth to an Rh Positive baby which is Coomb's Negative, should be given 1 ml. Rh Immune Globulin, intramuscularly, within 72 hours of delivery.
2. Any Rh Negative woman with Rh Positive mate, who suffers an abortion or ectopic pregnancy and showing no demonstrable antibodies in her blood, should have 1 ml. Rh Immune Globulin within 72 hours of the abortion or ectopic.
3. A repeat injection should be given after each succeeding pregnancy.
4. A follow-up with blood sampling for presence of antibodies is done at 6 weeks and 6 months following the injection.
5. It is therefore possible to eradicate this disease if the public and medical profession take advantage of this scientific breakthrough.
6. Supplies of the Rh Immune Globulin might therefore be expected to diminish and finally cease if the source were confined to those donors who had been severly affected. A new source is, in actual fact, now being created. Sterilized and/or post menopausal women are having their Rh antibody titres enhanced by the injection of Rh Positive cells so that the supply of the Immune Globulin will be protected.
7. Since March 15, 1969 the supplies of Rh Immune Globulin have been stored with the Red Cross Depot in Halifax, and any hospital in Nova Scotia can simply contact the Red Cross for a supply of phials to serve the needs of their area.
8. Since the supplies of Rh Immune Globulin became available in Nova Scotia last June, approximately 600 women have been injected and therefore protected from Rh Disease with their next pregnancy.
9. May we hope that all women "at risk" regarding the development of Rh sensitization are injected post partum, or following abortion, or ectopic gestation, and thus be protected.

Rh Committee of The
Medical Society of Nova Scotia
5821 University Ave.
Halifax, N. S.

Phone 422-6501, ext. 241 □

Management of Pancreatic Trauma

M. A. Naqvi, M.D., F.R.C.S.(C), D. S. Nathanson, M.D., C.M., F.A.C.S. and
J. A. Roach, M.D., C.M.

New Waterford, N. S.

Traumatic pancreatic injury accounts for 2% of all abdominal trauma and there is a high incidence of multiple organ injuries associated with pancreatic trauma. The management of pancreatic injuries is difficult due to anatomical inaccessibility of the organ.[1,2] Recently, at the New Waterford Consolidated Hospital, we encountered a case of pancreatic injury due to blunt abdominal trauma who underwent exploratory laparotomy and drainage of the pancreas with successful recovery.

Case Report

G.M. A twelve-year-old boy was admitted to the surgical service of the New Waterford Consolidated Hospital on December 29, 1968, because of severe abdominal pain and vomiting of three days duration. Pain was localized in the epigastrium and later on became generalized. The patient gave a history of being struck in the abdomen with a hockey stick one day prior to the onset of the pain. He denied constipation, diarrhea, weight loss, anorexia or similar episodes of abdominal pain.

Physical examination revealed a young boy of average height and weight, who was cold and clammy. Blood pressure was 110/80 mm. hg., pulse rate was 120 per minute. Temperature was 101.6 degrees F. Abdominal examination revealed marked tenderness in the entire abdomen, more marked in the epigastrium and right upper quadrant. Rebound tenderness was present with muscle guarding. Bowel sounds were present but of low pitch. Liver dullness was not impaired. The rectum was empty and there was no tenderness on rectal examination. The remainder of the examination was noncontributory.

Laboratory studies disclosed the following values - Hemoglobin 10.8 grams, White Blood Cell Count 10,000/cu. mm., Urine was dark yellow with specific gravity 1.018 and was negative for albumin and sugar. Abdominal X-rays showed some elevation of the right hemidiaphragm, there was no evidence of free air, a serum amylase was 260 units (normal 50-150). A repeat serum amylase obtained within a few hours was 1200 units. A diagnosis of pancreatic injury was made and patient was operated upon. At operation approximately 500 c.c. of free blood was present in the peritoneal cavity, there was a large hematoma in the region of the head of pancreas extending into the duodenum, mesentery of small bowel and retroperitoneal space. Pancreatic head was boggy and edematous. Pancreatic capsule could not be visualized due to hematoma formation. The duodenum was mobilized and there was no evidence of any injury to the duodenum. The pancreatic region and lesser sac were drained with sump tubes and peritoneal cavity was closed. In the post-operative period there was considerable drainage from the sump tubes which contained high levels of serum amylase. The patient was placed on nasogastric suction with adequate fluid and electrolyte replacement. His serum amylase continued to drop and returned to normal levels on the seventh post-operative day. His sump tube drainage and gastric drainage gradually decreased. A gastrointestinal series in the early post-operative period revealed extrinsic duodenal obstruction with wide duodenal loop and subsequent follow up X-rays failed to reveal any evidence of obstruction. The patient made a successful recovery and discharged home on the 24th hospital day.

Discussion

Trevers, in 1827 reported the first case of rupture of the pancreas[3]. Since that time, the literature has been abundant on this subject. Pancreatic injuries have been classified as operative due to surgical operation and nonoperative due to penetrating and blunt abdominal trauma. Various series have reported a preponderance of blunt trauma over penetrating injuries[4]. The mechanism involved in blunt trauma is a crushing blow in the abdomen with relaxed abdominal wall muscles resulting in compression of the pancreas against vertebral bodies,[5,6] or a light blow to the abdomen with sustained doubling up action and an accentuated flexion of the trunk[7].

The clinical picture produced by this injury depends upon the severity of haemorrhage, extent of the damage to the pancreas and its duct. Pain, vomiting, abdominal distension and ileus will become prominent with pancreatic damage and discharge of pancreatic secretion into the peritoneal cavity[1]. A preoperative diagnosis is difficult. In the series reported by Thompson and Hinshaw, 84% of the patients were diagnosed during operation[1,7,8]. Elevated serum amylase is the most important diagnostic measure and high level in the abdominal paracentesis in a patient with blunt trauma is an indication for laparotomy[9]. According to Cleveland, the most important factor in diagnosis is an awareness on the part of the examining surgeon that traumatic pancreatitis may exist in any patient who has suffered severe trauma to the abdomen[10].

The principle of therapy in pancreatic injury involves early surgical exploration, adequate sump drainage, control of haemorrhage and definitive pancreatic surgery depending upon the extent of damage. Contusion of the pancreas without laceration, including moderate to marked hematoma formation, require drainage of the lesser sac and pancreatic area. The surgeon's aim at the time of operation is to preserve pancreatic tissue and to provide some form of drainage to the pancreatic secretions[11]. Drainage leading to the exterior is recommended routinely even for minimal trauma. When damage is limited to the tail of pancreas, disrupted tissue may be resected[12]. Doubilet and Mulholand recommended repair of the main duct of Wirsung, and also advised sphincterotomy in conjunction with primary repair of the duct in an effort to prevent build up of pressure within the ruptured duct from the resistance of sphincter[13]. Others have recommended Roux-En-Y Pancreaticojejunostomy in cases where the duct has been divided within the head and the body of the gland[1,8,14]. Mule has anastomosed both proximal and distal cut ends separately into the proximal jejunum with good result[7]. In the series reported by Werschky[11], 53% of the cases were treated by simple drainage, 28% had repair of the capsule and 12% underwent caudal pancreatectomy.

The common complications of pancreatic injury are fistulae, pseudocyst, pancreatitis, abscess, secondary haemorrhages and exacerbation of pre existing diabetes. Pancreatic fistula is the most common complication and the incidence is twice as high in blunt trauma as it is in penetrating injuries. Fistulae are more common in injuries of head and body than the tail, and are also frequent after simple drainage of the pancreas. Most of the fistulae are minor and close spontaneously[11]. Pseudocyst develops due to smaller leaks which remain localized, and are more common after conservative management[15].

The mortality in pancreatic injuries has dropped since the period of World War II from 56% to about 13-39%. At present, the mortality ranges between 20-30% in various series.[13] Mortality from penetrating wounds is higher than with blunt trauma and lower in patients with simple drainage than in those who have had definitive surgery[6,11].

Summary

A patient with traumatic pancreatitis due to blunt abdominal trauma was treated at the New Waterford Consolidated Hospital by simple drainage of the pancreas with successful recovery. The literature has been reviewed, the principles of management in traumatic pancreatitis involve early surgical exploration, adequate sump drainage, control of haemorrhage and definitive pancreatic surgery depending upon the extent of the damage. □

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Allopurinol in the Treatment of Gout

J. F. L. Woodbury, M.D., C.R.C.P.(C), F.A.C.P.;
D. M. Mehta, M.D., F.R.C.P.(C)*; W. I. Morse, M.D., F.R.C.P.(C)

Halifax, N. S.

The introduction of allopurinol has made possible the control of many cases of gout that are resistant to adequate treatment with urinosuric drugs. The action of this drug is more gradual and prolonged, and cases which require very large doses of probenecid (Benemid) or sulphinpyrazone (Anturan), for example, may be controlled by relatively small doses of allopurinol (Zyloprim).

However, it should be realized that allopurinol constitutes only one part of the treatment of gout. It is important that the attacks of crystal-induced inflammation in joints, bursae or tendons be treated with anti-inflammation drugs such as colchicine or phenylbutazone and that treatment of the defective metabolism of uric acid be considered as a long-term problem. Drugs that effect reduction in levels of serum uric acid may initiate attacks of acute synovitis when they are first given. Although bouts of inflammation are reduced in frequency or eventually eliminated, such a state of affairs is achieved only after weeks or months of maintenance of normal levels of serum uric acid. The practitioner who undertakes the long-term therapy of gout should be prepared (a) to decide whether correction of hyperuricaemia is necessary, (b) to decide whether the form of treatment should be uricosuric or with xanthine oxidase inhibitor (allopurinol), and (c) to monitor the efficacy of the treatment programme by estimation of serum uric acid levels at regular, frequent intervals (fortnightly until a satisfactory level is achieved, and then quarterly). This question was discussed in greater detail by one of the authors in 1968.[1]

The discovery of allopurinol has had important theoretical implications also, in relation to the mechanisms of development of primary hyperuricaemia. It has been established that the purines which are eventually excreted, chiefly as uric acid, are synthesized *in vivo*, and that many patients with gout synthesize an abnormal quantity. Thus, it is agreed that overproduction of purines and uric acid plays a part in the causation of gout, and that in these cases the disease is amenable to suppression by allopurinol. Less widely accepted is the view that renal dysfunction may contribute to the development of hyperuricaemia. Understanding of the role of the kidney in the development of hyperuricaemia has always been clouded by the fact that the kidney could be secondarily damaged by the excretion of unduly large amounts of uric acid; in recent years, however, several workers have adduced evidence of selective renal dysfunction in some patients with gout but without overproduction of uric acid. As a result, most former proponents of the theory of overproduction as the sole mechanism now concede that some patients have gout as a result of a renal function defect without overproduction and others have the renal defect in addition to overproduction. Before the advent of allopurinol, the treatment of gout was based upon increasing the renal excretion of uric acid, thereby demanding still more of the kidneys. The action of the xanthine oxidase inhibitor, allopurinol, in preventing the oxidation of hypoxanthines and xanthines to uric acid, permits the physician to present the kidneys with a smaller quantity of purines to excrete and with a purine load distributed among uric acid, xanthine, and hypoxanthine.

When allopurinol first became available to us, we treated several patients in the clinical investigation unit of the Victoria General Hospital and observed the effects on uric-acid metabolism. This paper presents some case reports which exemplify our experience.

Control Studies

Serum uric acid values were determined in 100 normal subjects, not all of whom were fasting when specimens were taken (MacKenzie, A., and Dewar, J., unpublished), with an Auto Analyzer, by a modification of the colorimetric procedure reported by O'Sullivan, Francis, and Kantor,[2] who found high correlation with the uricase spectrophotometric manual procedure. (Their mean value was 0.31 mg. per 100 ml. higher with the automated method than with the uricase method, with slightly greater differences at higher levels of urate concentration.) Most of the 50 females were young laboratory workers, and the 50 males, most of whom were relatively young, were laboratory workers, medical students, or Armed Forces' personnel. These values (mean ± 2 S.D.) ranged from 2.7 to 5.6 mg. per 100 ml. in females and from 3.9 to 7.9 mg. per 100 ml. in males; however, using the same method, O'Sullivan *et al*[2] suggested that values above 6 mg. per 100 ml. in females and 7 mg. per 100 ml. in males usually are associated with a tendency to gouty arthritis.

From the Department of Medicine Victoria General Hospital and Dalhousie University,
*Clinical Fellow of the Canadian Arthritis and Rheumatism Society.

CASE REPORTS

Case 1:

This 65-year-old male had a 13-year history of recurrent acute gouty arthritis; he had had huge tophi, renal calculi, and mild chronic renal failure. Prior investigation had shown that suppression of the serum uric acid to normal levels required the administration of 1000 mg. of sulphinpyrazone per day; after a year of this treatment the tophi had nearly disappeared. His urate clearance without treatment was 3.6 ml/min.; while he received large doses of sulphinpyrazone it rose to 9 ml./min., and when allopurinol was substituted it fell to 4.2 ml./min. - almost the pre-treatment level.

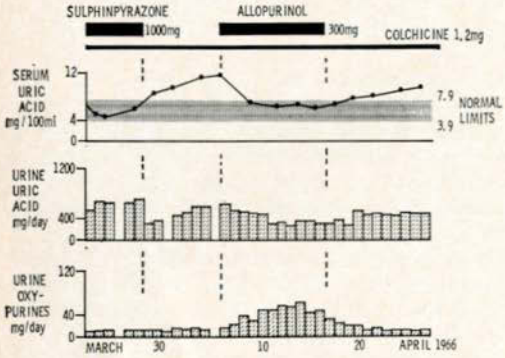

Fig. 1, Case 1
Comparison of effects of a large dose of sulphinpyrazone and a small dose of allopurinol in a 65-year-old man with chronic tophaceous gout and renal damage.

As shown in Fig. 1, after sulphinpyrazone was discontinued, the 24-hr excretion of uric acid dropped precipitously and then rose gradually to 640 mg. per day, a little above the upper limit of normal; concomitantly, the serum level of uric acid rose over a period of 9 days to 12.2 mg./100 ml. When allopurinol therapy was started, in a dosage of 100 mg. *t.i.d.*, oxypurine excretion increased and on the second and third days was 22 mg. per day, about double the usual values for this patient; at the same time the urinary output of urate decreased. The rise in oxypurine output and the fall in uric acid output continued until the latter totalled 300 mg. or less per 24 hr. When allopurinol was discontinued, oxypurine output decreased until reaching baseline levels on the sixth day and then was maintained, and the urinary output of urate reached a maximum of 530 mg. per 24 hr at 4 days and then levelled off at approximately 450 mg. per 24 hr during the next 11 days.

This case is notable because of the very high dose of uricosuric agents necessary to produce satisfactory results and the easy control by a moderate dose of allopurinol. It is presumed that the size of the necessary dose of uricosuric drugs is due to impaired renal function.

Table I. Mean total daily uric acid excretion in case 1 (male, aged 65 years)

Therapy	Dose (daily)	Uric acid excreted (mg.)
Colchicine	1.2 mg.	504
Sulphinpyrazone	1000 mg.	698
Allopurinol	300 mg.	427
Sulphinpyrazone and allopurinol	500 mg. 300 mg.	551

This patient's urinary output of urate during various therapeutic regimens is shown in Table I. It increased from within normal limits by 40% on uricosuric drugs alone, fell 15% below the control values with allopurinol alone, and rose to 10% above control with a combination of allopurinol and sulphinpyrazone. (Uric acid excretion while the patient was receiving both allopurinol and sulphinpyrazone probably was higher than shown, since comparison with urinary creatinine output indicated that parts of two 24-hour collections were lost.)

Table II. Mean total daily urinal Excretion of oxypurine in case 1 (male, aged 65 years)

Therapy	Dose (daily)	Oxypurine excreted (mg.)
Colchicine	1.2 mg.	11
Sulphinpyrazone	1000 mg.	10
Allopurinol (1966)	300 mg.	41
Sulphinpyrazone and allopurinol (1967)	500 mg. 300 mg.	30.7*

*See case report

It has been reported by Ogryzlo and associates[3] that uricosuric agents given during allopurinol therapy appear to inhibit to a varying degree the urinary excretion of oxypurines, resulting in slightly reduced excretion during the periods of combined therapy. Table II shows that mean daily excretion of oxypurine while this patient received allopurinol alone was 41 mg; when he returned for further evaluation a year later, and sulphinpyrazone was added to his therapy, the mean raw figure was 30.7 mg., but adjustment for probable urinary losses increased this to 37 mg. per day.

Case 2:

The next case illustrates a less striking response to allopurinol (Fig. 2). This 36-year-old male was known to have had attacks of acute gouty arthritis over the preceding 11 years and an episode of renal colic in 1957. Small tophi had developed recently. Co-operation with treatment on an out-patient basis had always been poor and he was admitted to hospital 19th May 1966. At the time of his admission the blood urea nitrogen was 25 mg. per 100 ml.

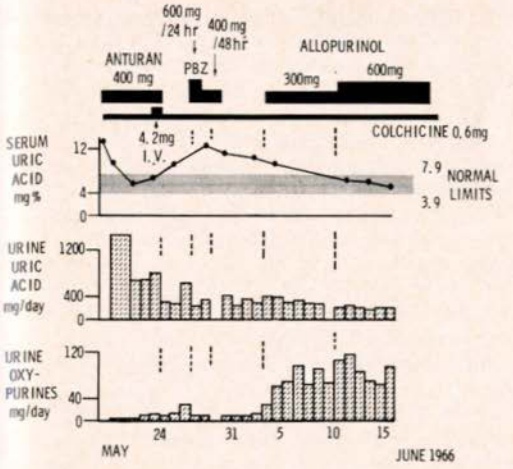

Fig. 2, Case 2
Slow but satisfactory response to allopurinol after cessation of uricosuric therapy in a 36-year-old male normo-exceretor of uric acid.

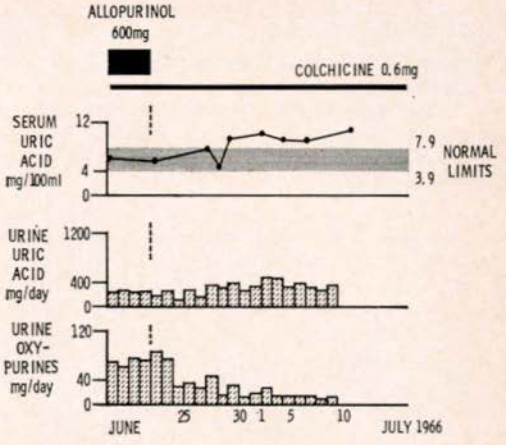

Fig. 3, Case 2
Gradual return to elevated serum levels, and to usual output of uric acid and oxypurines, after discontinuance of allopurinol therapy.

and a few granular casts were found in his urine. Although ostensibly he was taking sulphinpyrazone (Anturan, 400 mg. per day), the serum uric acid was 13.3 mg. per 100 ml., which was similar to values obtained over the preceding decade — during most of which time he had admitted to not taking medication. The administration of sulphinpyrazone in hospital was followed immediately by a precipitous fall in serum uric acid to 5.7 mg. per 100 ml. and a spectacular rise in urate output. When allopurinol was started (300 mg. per day), on 4th June, the expected rise in oxypurine excretion was immediately evident; decrease in the 24-hour output of urinary uric acid was less obvious but there was suggestive evidence of immediate response. Discontinuance of allopurinol was reflected in a change towards pretreatment values in serum and urine uric acid levels and oxypurine excretion within 3-6 days (Fig. 3).

After the patient was discharged from hospital, ostensibly on allopurinol, abnormal serum uric acid levels were consistently observed, presumably because he did not take his medication. Note that this man is a normo-exceretor, and a moderately high dose of allopurinol appeared necessary to suppression of urate output below average normal value.

Case 3:

This woman, who was 76 years old, had been subject to severe prolonged attacks of polyarticular gouty arthritis with a tendency to chronicity for 6 years. The blood urea nitrogen was 25 mg. per 100 ml., and creatinine clearance was 30 ml. per min.; uricosuric therapy failed to maintain satisfactory control of the serum uric acid level. The response to allopurinol (300 mg. per day) was excellent although the level of creatinine clearance did not change (Fig. 4). This patient was discharged from hospital in October 1966 and has continued on the same dosage of allopurinol. Urinary urate

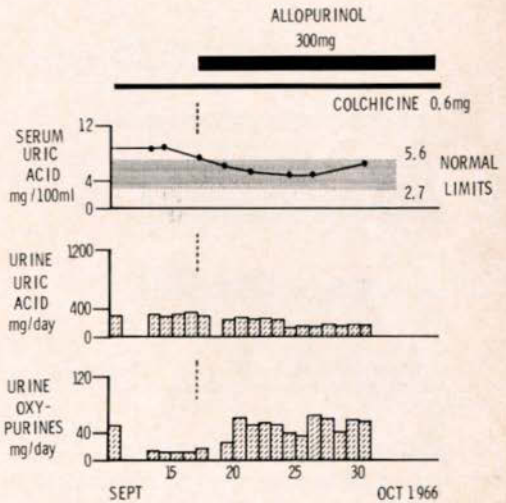

Fig. 4, Case 3
Good response to allopurinol in 76-year-old female with gout and impaired renal function.
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output in May 1967 was 309 mg. in 24 hr. - no lower than some of the established control values - and total oxypurine output has shown further increase, to 75 mg. per day. She has had no further attacks of gouty arthritis.

Case 4:

The second female patient was in advanced renal failure when referred to our study. Allopurinol was started in a dose of 300 mg. per day but had to be discontinued because of the development of a general pruritic macular eruption, fever, malaise, and swelling of the joints with distension by fluid which contained urate crystals. Two attempts

were made to re-institute allopurinol therapy in a dosage of only 100 mg. per day, but the same symptoms and signs developed and the treatment had to be abandoned.

Several authors[4-6] haver emarked that adverse reactions to allopurinol, especially rash, occur most often in patients with a significant degree of renal decompensation.

Case 5:

Response was unsatisfactory in a 39-year-old male also, in whom the simultaneous administration of sulphinpyrazone, phenylbutazone and colchicine had failed to achieve consistent control of his disease - probably due partly to failure of the patient to co-operate. He was thought to be addicted to alcohol and barbiturates and to have other personality problems. Open biopsy of the liver had demonstrated early cirrhosis, but results of liver function tests were normal when he came under our observations; glucose tolerance was slightly impaired. Uric acid crystals were found within and outside leucocytes in the synovial fluid.

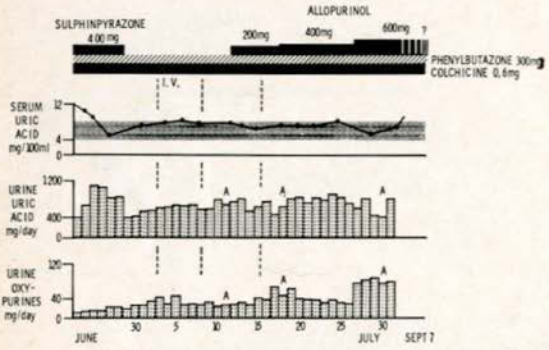

Fig. 5, Case 5
Unsatisfactory control of gouty arthritis despite some increase in urinary excretion of oxypurines in response to allopurinol.

Frequent attacks of acute gouty arthritis rendered it necessary to administer phenylbutazone as well as colchicine during studies of the patient's response to allopurinol. When sulphinpyrazone was withdrawn (Fig. 5), urinary output of oxypurine rose slightly; it increased further when allopurinol was given and reached its greatest height with increase in the daily dose to 600 mg. Even with this large dose of allopurinol the mean urinary output of urates fell very little below control value. The serum uric acid levels were elevated in this patient, also, after his discharge from hospital.

Discussion

The rate of production of purines and uric acid is governed largely by the recycling of the nucleotides of guanine and hypoxanthine. Kelley and associates,[6] who reported the identification of an enzyme, hypoxanthine-guanine phosphoribosyltransferase (HGPRT), necessary for the conversion of guanine and hypoxanthine to nucleotides, stated that absence of this enzyme leads to overproduction of urate; patients who are deficient in HGPRT respond poorly to allopurinol.[8]

Wyngaarden and his associates observed that some patients do not respond particularly well to allopurinol,[9,10] and suggested that this was due to a very high degree of renal clearance of hypoxanthine and xanthine.[11] They thought that this occurred most often in patients who had tophaceous gout with some degree of renal damage, but one of our patients (Case 5) who did not respond had no tophi and his creatinine clearance was 100 ml. per min.

Several authors have noted that the increase in urinary output of oxypurine which occurs when allopurinol is given is not as great as the decrease in urinary uric acid excretion. (To make the comparison between total purine excretions the data should be expressed in terms of millimoles per day; this was not done in our cases.) Using the raw figures in terms of milligrams per day (Table III) it can be seen that the total purine excretion appears lower on allopurinol: Yü and Gutman[12] have commented that this discrepancy is more striking in over-excretors of uric acid. Of the 6 patients whose data comprise this table, one had a mean 24-hour urate excretion of 654 and another excreted 615 mg. per day, both values being at the upper limit of normal. Pomales *et al*[13] believe that the discrepancy is due to the fact that the allopurinol protects hypoxanthine and xanthine from oxidation. The evidence for this is that if either is administered alone, almost all is excreted in the urine in the form of oxypurine and other end-products and little or none appears to be used for nucleic-acid synthesis; when allopurinol is administered concomitantly with labelled hypoxanthine or xanthine, however, the isotopic tracers are incorporated into nucleic acid adenine and guanine.

TABLE III. Daily urinary excretion of purines: aggregate of mean in six patients

Purines	Patients	Amount of purines excreted (mg./day)	
Uric acid	Untreated	2937	
Uric acid	Treated with allopurinol		2526
Oxypurines	Untreated	110	
Oxypurines	Treated with allopurinol		339
		3047	2865

Rundles *et al*[5] stated that the administration of allopurinol did not result in reduction in alcohol tolerance or impairment of iron absorption, storage, or incorporation into hemoglobin in any of their patients, but that a pruritic maculopapular skin eruption with fever, malaise and aching developed in 2-3% and disappeared without residua within a few days of cessation of allopurinol therapy.

In common with others we have found that the urate clearance rates are not significantly changed by allopurinol therapy. The mean clearance of urate was 4.2 ml./min. in our patients and 4.8 ml/min. in those of Ogryzlo and colleagues,[3] who reported finding a mean value of 7.1 ml./min. in 23 normal subjects. Similarly, the creatinine clearance rates of our patients did not change during allopurinol therapy, the mean value being 68.3 ml./min.

Summary

Allopurinol was administered in combination with colchicine or other agents to 10 patients known to have gout. An adverse reaction was encountered in only one patient, in whom rash, fever and malaise developed. The drug effectively reduced serum levels of uric acid and urinary output of urate when given in a dose of 300 mg./day in seven patients and 600 mg./day in two. Five illustrative cases are reported.

Allopurinol prevents the oxidation of purines to uric acid and permits their excretion in three different species of molecules, each of independent solubility. It is of great value in the treatment of patients with hyperuricaemia not controllable by uricosuric drugs, in those who cannot tolerate these agents, and in patients whose hyperuricaemia has produced renal damage or lithiasis.

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NEW MEMBERS

The Physicians listed below have joined The Medical Society of Nova Scotia between May 1 and June 30, 1969. A most cordial welcome is extended from the Society.

F. A. Dunsworth, M.D.,
President.

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DR. A. C. H. CROWE	Truro	DR. KAREN SAMPLE	Halifax
DR. E. K. HYSLOP	Vancouver, B.C.	DR. R. E. SCOTT	Halifax
DR. D. F. LARGE	Liverpool	DR. M. A. SMITH	Sydney
DR. G. P. LeBRUN	Halifax	DR. D. C. STEEVES	Liverpool
DR. C. W. McCORMICK	Middleton	DR. M. H. TAN	Halifax
DR. J. M. MacKEIGAN	Halifax		

Case Presentation - Poison Control Centre

E. A. Day, M.D.

The Children's Hospital
Halifax, N. S.

Case 1

A 3 year old child was referred to The Children's Hospital with a diagnosis of suspect meningitis. He had been treated for several days for an upper respiratory infection with tonsillitis and otitis media; and had then developed diarrhoea and vomiting, for which he received 5 mg. Stemetil IM. The same day, definite neck stiffness was noted, and the child became alternatively lethargic or very restless.

On arrival at The Children's Hospital, signs of meningeal irritation were present, with slight nuchal rigidity and a positive Brudzinski sign. These were puzzling because a short time later they were no longer present, but re-appeared after several hours. Signs of dystonia also developed, indicating extrapyramidal involvement.

Examination of the cerebrospinal fluid was normal.

Benadryl 15 mg. intravenously, six hourly for 3 doses was given with immediate marked improvement.

Case 2

A 6 year old boy developed severe intermittent vomiting, unresponsive to gravol. His physician then prescribed one-half of a Stelazine suppository q8h.

Two days later the vomiting had stopped, but the boy suddenly developed opisthotonos with head turned to the left. This passed off in a few minutes but re-occurrred every ten to fifteen minutes.

On admission to hospital he was drowsy but conscious. During examination an attack occurred; immediate relaxation resulted from the administration of 1 mg. of Cogentin intravenously.

For the next two days he was treated with Benadryl 10 mg. orally and was then discharged, with no neurological deficit.

Comment:

Stemetil and Stelazine are both phenothiazines used occasionally as anti-emetics in childhood. This group of drugs produces changes at all levels of the cerebrospinal axis, and also exerts significant effects upon organ systems throughout the body. The effects illustrated in the two cases presented are extrapyramidal ones, and can be considered extensions of the ordinary pharmacological actions on the central nervous system.

Bizarre motor effects are common with high doses of the phenothiazines, in particular, a parkinsonian syndrome, dystonia, and dyskinesia and akathisia. Dyskinetic manifestations may be confused with convulsive seizures, but the latter may occur in any case.

Stelazine is a long acting drug, and a single 4 mg. suppository should provide symptomatic control from ten to twelve hours in an adult; it is not recommended for use in children under 12 years of age, except in hospital.

Stemetil injection is indicated exclusively for severe cases. The manufacturer's dosage recommendations should be closely adhered to and the physician should be alert to the possibility of dystonic reactions occurring in children. Infants and children are particularly sensitive to phenothiazines, and toxic symptoms simulating tetanus, meningitis, or frank dystonia with bizarre posturing do not seem to be dose-dependent, but may be idiosyncratic in nature.

Intravenous or intramuscular Benadryl, in doses of 5 - 10 mg., or Cogentin in doses of 1 mg. per dose, not only produce dramatic improvement but are of value diagnostically.

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Renal Tubular Necrosis and Pulmonary Edema

Reprinted from the Canadian Medical Association Journal, Vol 93, page 1365. Dec. 25th, 1965

A 30-year-old white married woman, who was pregnant for the first time and had an expected date of confinement of March 23, 1960, first consulted a physician for prenatal care when she was four months pregnant. On general physical examination at that time she was in good health and her hemoglobin (Hb.) was 12 g. %. The physician prescribed a low-salt diet and 15 grains of ferrous sulfate daily. She visited her physician when five months pregnant, at which time she felt well. During the next two months the physician made three home visits because the patient had upper abdominal pain, nausea and vomiting. On radiographic investigation on January 15, 1960, her gallbladder was abnormal and a fat-free diet was advised. She did not return for additional prenatal visits, and her diet for the remainder of her pregnancy consisted mainly of crackers and milk.

The patient was admitted to hospital on March 29, 1960, with uterine contractions every three minutes. At this time she was pale but was otherwise well. Two hours after admission a healthy 7 lb. 4 oz. female infant was delivered spontaneously. The patient did not receive any sedation; however, a few "whiffs" of trichlorethylene (Trilene) were given with the delivery. The placenta was delivered immediately. The uterus contracted well after 0.5 mg. of ergometrine maleate (Ergometrine) was given intravenously five minutes after the placenta was delivered. No vaginal lacerations had occurred and the blood loss was estimated to be 250 c.c. A Hb. estimation was not done.

Eight hours after delivery the nurse noted that the patient was bleeding briskly *per vaginam* and was passing several blood clots. The uterus was soft but contracted well following uterine massage and a second dose of 0.5 mg. of ergometrine maleate intramuscularly. Approximately 16 hours post partum the uterus again became soft and vaginal bleeding recurred. The patient's blood pressure dropped and an intravenous infusion of 5% glucose in water was begun. The bleeding persisted despite a third dose of 0.5 mg. of ergometrine maleate intramuscularly.

About 18 hours post partum the attending physician returned to the hospital. The blood pressure at this time was 84/64 mm. Hg and the intravenous infusion had stopped running. A femoral venipuncture was performed to obtain blood for cross-matching and an ankle cut-down was done. During the next three hours 1500 c.c. of Group O Rh-negative blood was administered and the Hb., determined for the first time 22 hours post partum, was 6 g. %. The vaginal bleeding continued and 28 hours after delivery another 1000 c.c. of cross-matched whole blood was given intravenously under positive pressure.

Consultations with an internist and an obstetrician were held 31 hours post partum. The internist reported, "The shock is a problem and over-hydration is not a worry unless anuria persists after the blood pressure is maintained." An ampoule of calcium gluconate and 100 mg. of hydrocortisone. (Solu-Cortef) were given intravenously. The obstetrician recommended exploration of the birth canal, and this was carried out 36 hours post partum. A few pieces of necrotic placental tissue were removed and the uterus and the vagina were packed with several lengths of three-inch gauze.

During the next 24 hours, 4000 c.c. of cross-matched whole blood and 1000 c.c. of plasma were given. The systolic blood pressure remained less than 100 mm. Hg and the total urinary output for the first 48 hours post partum was less than one-half ounce.

On the third day 1000 c.c. of whole blood and 1500 c.c. of 5% glucose in water were administered intravenously. The systolic blood pressure was 80 mm. Hg, the Hb. was 7 g. % and during that day the urinary output was less than 5 c.c. The uterine and vaginal packing was removed and there was no further bleeding.

During the fourth day, 2400 c.c. of intravenous fluid, consisting of 1000 c.c. of whole blood, 250 c.c. of one-sixth molar lactate and 1150 c.c. of 5% glucose in water, was given.

On the fifth day, the blood urea nitrogen (BUN) was 57 mg. %, the serum potassium was 5.64 mEq./l., the serum sodium was 137 mEq./l., the serum chloride was 87 mEq./l., and the CO_2 combining power was 48 vol. %. The Hb. was 8.5 g. %, the blood pressure was 100/70 mm. Hg and the pulse rate was 60 to 80 per minute.

On the sixth day, the patient began taking oral fluids. The blood pressure and the pulse rate were normal, and the serum potassium was 5.24 mEq./l. The total urinary output during the fourth, fifth and sixth postpartum days was less than 1 oz.

On the ninth day, the Hb. was 8.6 g. %, the BUN was 78 mg. % and the urinary output was 1 oz.

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.

On the tenth day the serum potassium was 3.5 mEq./l., the BUN was 108 mg. %, and the urinary output was 2 oz. The patient became comatose and had several liquid stools that evening; the following morning she had several generalized convulsions and died.

A complete autopsy was performed. The cause of death was renal tubular necrosis and pulmonary edema.

Decision of Committee on Maternal Welfare

The conclusions reached by the Provincial Committee on Maternal Welfare after a review of the case were as follows: "This was a preventable direct maternal death with combined preventable factors. There was a patient factor in that the patient did not avail herself of adequate prenatal care. The preventable professional factors are as follows: The patient's anemia was not recognized when she was admitted to hospital in labour. There was delay both in the control and treatment of the postpartum hemorrhage. The patient developed renal anoxia due to postpartum hemorrhage and the preexisting anemia with resultant renal tubular necrosis, anuria and death 11 days after the delivery. The patient's circulation was overloaded with intravenous fluid in an effort to maintain the blood pressure and this resulted in pulmonary edema. This maternal mortality has been considered to be ideally 'preventable' under the terms of reference of the Provincial Committee on Maternal Welfare and there is no implication of any negligence."

Discussion

This patient had inadequate dietary intake for the last two or three months of her pregnancy because of food intolerance due to chronic gallbladder disease. Her hemoglobin was 12 g. % when she was four months pregnant; however, the Hb. was not determined again until 22 hours post partum. The Hb. should have been determined on admission or immediately after delivery and before the first postpartum hemorrhage which occurred eight hours after the delivery.

Iron supplement was prescribed for this patient when she was four months pregnant. It is wise not only to prescribe iron therapy during the prenatal course but also to question the patient regularly during her prenatal visits to determine whether she is taking the medication as described. Repeated Hb. determinations should be done during the prenatal course. If, despite iron supplement, the Hb. remains less than 10 g. %, additional hematological investigation is mandatory. This should include complete blood count, serum iron levels and possibly bone marrow puncture. On occasion parenteral iron therapy is necessary if the patient is unable to tolerate oral iron. Oral folic acid, 5 mg. three times daily, is recommended if megaloblastic anemia of pregnancy is diagnosed following bone marrow puncture.

When delayed postpartum hemorrhage occurs, it is sound obstetrical practice to return the patient to the case room immediately and examine the entire birth canal in order to identify and treat the cause of the postpartum hemorrhage. In this case the patient was not taken back to the case room post partum for such an examination until 31 hours later and the physician relied on ergometrine maleate and uterine massage to control the postpartum hemorrhage. It is quite possible that if such an examination had been performed immediately following the initial hemorrhage (eight hours post partum), this patient might have survived.

Once delayed postpartum hemorrhage occurs, immediate blood replacement is mandatory. An intravenous drip, consisting of 20 units of oxytocin (Syntocinon or Pitocin) in 500 c.c. of 5% glucose in water given at the rate of 30 drops or more per minute, is also recommended while the patient is being returned to the case room for exploration of the birth canal.

Because of the shock and severe anemia (the postpartum hemorrhage aggravated the existing anemia), this patient developed renal anoxia which resulted in renal tubular necrosis and anuria. Despite the presence of anuria her circulatory system was overloaded in an attempt to maintain the blood pressure; this resulted in pulmonary edema.

Summary

A maternal mortality was reviewed by the Provincial Committee on Medical Welfare. The cause of death was renal tubular necrosis and pulmonary edema. The preventable factors are discussed. □

Exposure to Cold Environment and Rhinovirus Common Cold

Volunteers without antibody to a rhinovirus strain were subjected to a cold environment before and after inoculation with the viral strain. No effect on the illness was demonstrated, despite popular belief that cold has an adverse effect on the common cold.

Although in animals exposure to cold has been shown to increase both the frequency and severity of viral infections, such exposure has not been demonstrated to increase the susceptibility of man to infection with common-cold viruses. Studies published to date, however, have been among volunteers whose antibody status to the infecting virus was not known.

For the present study 44 volunteers found to be free of antibody to rhinovirus Type 15 were inoculated with purified stocks of this strain. Of these, 27 were exposed to cold at the time of inoculation and during incubation, illness, and recovery. The remaining 17, who served as controls, were not exposed to cold.

To test the normal reaction to the cold devices used in the study, five additional volunteers, wearing light cotton shorts and undershirts, spent one and one-half hours in rooms with temperatures of 4° and 10°C (centigrade). Since the cold caused shivering which led to elevation of body temperature, adjustments were made to lower body temperature. These consisted of immersion in a water bath at 32°C for four to six hours and of wearing wool jackets in the 10°C room.

The subjective opinion of the volunteers was that the experience in the 4°C room was uncomfortable and comparable to the short-term chilling commonly encountered in nature. In contrast, when placed in a tank with 32°C water three days later, they found the experience comfortable.

Inoculation of the 44 subjects was done by small particle aerosol in 28, by nasal instillations of virus-containing fluids in 16. There was no difference in clinical response to the two methods.

Inoculated After Exposure

In one experiment, nine of the 16 volunteers inoculated by nasal drops were infected at the end of exposure in the 4°C room. The other seven were not exposed to cold. Infection rates in both groups were similar and the illnesses were typical of the common cold. All the infected demonstrated early-high virus-shedding patterns. In each an eightfold or greater rise in serum neutralizing antibody developed five weeks after inoculation. Titers in both groups were comparable.

In following experiments six volunteers were placed in the 10°C room and seven in the cold bath on the morning after inoculation. Both exposed groups were compared to unexposed volunteers inoculated at the same time. Results were similar in all three groups. The frequency and severity of illness were not significantly greater in the group exposed to cold than in the unexposed volunteers. Furthermore, there was no increase in quantities of virus shed from the nasopharynx among volunteers exposed to cold. Antibody responses among those exposed were also similar to those not so exposed.

Clinical responses, virus-shedding patterns, and antibody responses were also similar in two volunteers placed in the water bath on the first or second day of illness and two others inoculated at the same time but not exposed to cold.

Illnesses Similar

In another experiment, six volunteers were placed in the 10°C room and seven in the water bath on the seventh or eighth day after inoculation. Illnesses in the two groups were similar.

When four volunteers were reexposed to cold in the water bath, nasal obstruction and discharge developed in three. In two the symptoms disappeared within eight hours; in the third, who also had headache and cough, the symptoms lasted for 24 hours. No symptoms were observed in the three volunteers first exposed to cold after recovery from illness. Virus-shedding patterns and mean antibody responses were similar in both groups.

Pneumococci and staphylococci as well as nonpathogenic species of bacteria were recovered from nose and throat swabs from both the volunteers exposed to cold and from those not exposed in each of the experiments.

No effect of exposure to cold on a common cold due to rhinovirus Type 15 that might account for the popular belief that cold was bad for a common cold was demonstrated. However, two possible mild effects of cold on rhinovirus illness were noted. One was a slight increase in nasal symptoms in three of seven volunteers exposed to cold during recovery, and the other was a slightly greater frequency of

R. Gordon Douglas, Jr., M.D.; Keith M. Lindgren, M.D.; and Robert B. Couch, M.D. *The New England Journal of Medicine*, October 3, 1968.
Reprinted from the Abstracts of the National Tuberculosis Association, February 1969.
Printed through cooperation of the Nova Scotia Tuberculosis Association.

illness among some subjects exposed to cold during the incubation period. In both, the difference between subjects exposed to cold and controls was minimal.

Failure to demonstrate a relationship between cold and the common cold could be because environmental temperature has no effect on the common cold; or because exposure for longer periods or to more severe conditions may be necessary to influence the course of common colds. It may be, too, that exposure to cold influences the course of other viral illnesses in man, but not rhinovirus illness.

It is also possible that infection with a wild rhinovirus and its tissue culture progeny would have a different result, as might exposure to a different strain of virus. However, rhinoviruses are the most frequent isolates from adults with colds and, with the possible exception of Type 1A, there are no predominant serotypes.

No Bacterial Complications

Exposure methods used in the study permitted testing of differing effects of cold. The 4°C room was analogous to short-term chilling of the type encountered in nature. The water bath was a comfortable procedure, but produced a fall in body temperature.

None of the volunteers had bacterial complications such as otitis media or pneumonia. The presence of potentially pathogenic organisms was not greater in the volunteers after infection and exposure to cold than in those after infection alone. In neither group were potential pathogens more frequent after infection than before inoculation. There was no evidence that respiratory viral infections predispose to pneumonia by qualitative changes in bacterial flora. More detailed bacteriologic studies will be needed to determine if significant quantitative changes occur in this regard. □

STATEMENT ON DRUG ABUSE

The following Statement relating to the use of Drugs was produced by the Alberta Medical Association. It is commended to the attention of all physicians.

1. Accumulated evidence indicates that hallucinogenic and mind altering drugs including L.S.D. (lysergic acid diethylamide), S.T.P. (dimethoxy methylamphetamine), Amphetamines, Marijuana, Barbiturates and related drugs, produce injurious mental and/or physical effects.

2. Each of these is a potent substance with mind altering properties and toxic potential when taken by humans. Toxicity is dose-related but adverse reactions depend also, in part, on the unique nature and personality of the user.

3. We recognize that the increasing use of marijuana is causing much legal, social and medical concern. Therefore special consideration has been given to marijuana.

4. The evidence from scientific observations in Western countries has not as yet been compiled in a controlled and scientifically valid fashion. To contend that the use of marijuana is therefore harmless is not warranted. There is definite evidence of toxicity. After careful perusal of much published evidence from the World Health Organization and other authoritative sources we are of the opinion that long term or chronic use of the drug produces psychic dependence.

5. In the short term or intermittent user of marijuana the effects of use are unpredictable. Documented reactions include depression, panic episodes and development of an amotivational syndrome.

6. We would suggest that the usage of the above-mentioned drugs be subject to continued legal control. We would also emphasize that illegal trafficking procedures be subject to continued stringent legal penalties. This does not mean that present regulations and legal restrictions could not be improved upon. □

Lung Cancer Detection by Chest X-Rays at - Six - Month Intervals

In a controlled study in England, cancer of the lung was detected in a resectable stage more frequently when chest X-ray surveys of the population at risk were made at intervals of six months than when the surveys were three years apart.

An investigation was carried out to test the hypothesis that the early detection of lung cancer by frequent routine chest roentgenograms of the population at risk might improve the prognosis of the disease.

In a three-year study, comparison was made between the lung cancer experience of two random population groups, one of which was offered more frequent roentgenographic examinations than the other. The subjects were men 40 years of age or older who were employed in industry.

Chest roentgenograms were available to the men in one group, the test group, every six months. Those in the control group had roentgenograms only at the beginning and end of the study. A total of 29,723 were enrolled in the test group, of whom 29,416 were followed up, and 25,311 in the control group, of whom 25,044 were followed up. Smoking habits in the two groups were almost identical. There were no apparent occupational hazards in the industrial firms where the subjects were employed that might be expected to have a bearing on the development of lung cancer.

On initial survey, 31 cases of lung cancer were detected in the test group. At the mass surveys at six-month intervals, 59 cases were detected; at the final survey of the group, six cases. Among the controls, 20 cases were detected at the beginning of the study and 76 during the three years.

Cancer Detected Elsewhere

At the end of the three years, 33 of the 59 patients whose cancer had been detected in the six-month surveys were alive and 26 had died. It was found that 36 patients in the test group and 59 in the control group, whose cancer had been discovered elsewhere, had died from lung cancer at some time between the first and last roentgenograms. Of the 36 in the test group, seven had died within six months of the initial chest film. The remainder had missed some or all of the six-month surveys. In the control group, three of the 59 deaths had occurred within six months of the initial examination.

When roentgenograms preceding the one which led to the diagnosis of lung cancer in the test group were reviewed, abnormalities that might have been related to the disease were noted for the first time in seven cases. Six of these were still suitable for resection.

In calculating the rate of detection of lung cancer, it was obvious that where the interval between the chest X-rays was approximately three years (the initial and final survey in the control group and the initial survey of the test group), the detection rates were almost identical.

However, where the interval was six months, as in the final survey of the test group, the rate of detection was considerably lower. This was attributed to the fact that many cases of lung cancer had already been discovered at the intermediate examinations of this group. The detection rate for the six-month surveys was remarkably constant. The mean annual incidence of lung cancer in the group was 0.9 per 1,000 examined.

Resectability

The resectability of the cancer found in the routine examinations every six months was compared with that of cases detected in the surveys at three-year intervals and with that detected by methods other than by mass X-ray surveys. Thirty-one (61 per cent) of 51 cases discovered by the initial survey of both groups were resectable as compared with 42 (65 per cent) of the 65 cases detected in those examined every six months.

Thus, the chance of resection is not materially influenced by the frequency of the X-ray examination, but since more cases will be discovered by examination at six-month intervals, the number of patients given this chance will increase. However, the rates for resectability of cancer discovered by the surveys were decidedly higher than cancer discovered by other means, 5-6 per cent compared with the test group; 18.7 per cent as compared with the controls.

The study has shown that, due to more frequent examination of the same population, 59 cases of lung cancer were discovered which would not have been found at that particular time had only three-year surveys been employed.

G. Z. Brett, M.D., "The Value of Lung Cancer Detection by Six-Monthly Chest Radiographs," *Thorax*, July, 1968 (23:414).
Reprinted from the Abstracts of the National Tuberculosis Association, January 1969.
Printed through cooperation Nova Scotia Tuberculosis Association.

The rate of resection in these cases was not significantly higher than in those discovered at the initial surveys of both the test and control groups conducted on conventional lines of mass radiography. This may be because the longer mass X-ray surveys are spaced out, the greater the number of patients with lung cancer who will have been diagnosed by other methods. Thus, the lung cancer remaining to be detected will have a relatively short period of roentgenographic detectability, probably not substantially different from that in the cancer detected in surveys at six-month intervals. This might account for the similarity of the resection figures.

In the design of the study the comparison of death rates from lung cancer between the test and control groups was of crucial importance. The lower lung cancer mortality observed in the test group might be interpreted as an indication that early diagnosis on the basis of roentgenographic examination every six months may have some effect on the future of the disease, but the difference in mortality between the groups was too small for this interpretation.

Evaluation

The conclusion to be drawn must be based on both the positive and equivocal results of the study. If the value of the roentgenographic examinations every six months is to be measured only by a significant reduction of mortality from lung cancer in a population at risk, then no definite evidence has emerged to justify a policy of frequent large-scale surveys of this kind. However, if the merit of early diagnosis and a better chance of resection for a larger number of cancer patients is linked with even a small reduction of mortality, there is, without detracting from the importance of prevention by a change in smoking habits, reason why men in the cancer age should have chest X-rays regularly. □

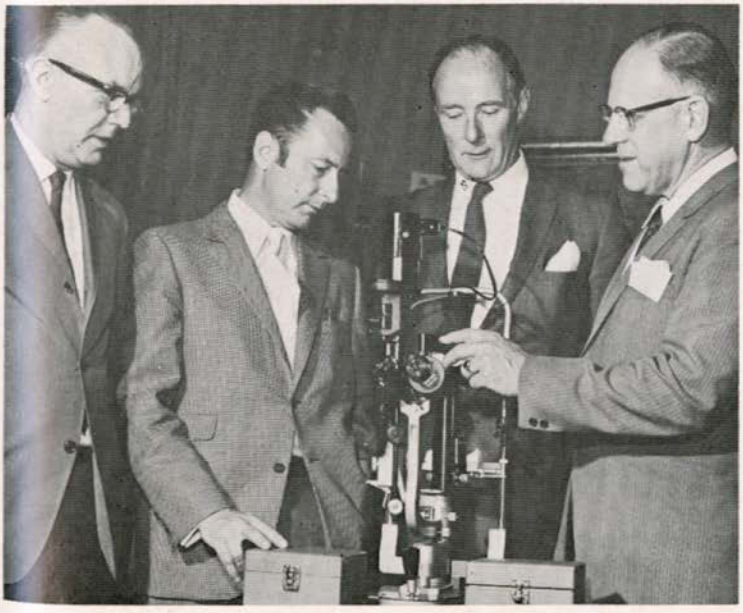
Personal Interest Notes

Portraits of former Deans of Medicine were presented by the Medical Alumni Association to the Faculty of Medicine on May 16th. **Dr. C. B. Stewart** received the portraits of Drs. A. P. Reid, A. W. H. Lindsay, John Stewart, W. H. Hattie, and H. G. Grant in a brief ceremony in the arcade of the Tupper Building. **Dr. R. O. Jones** presented the portraits.

Dr. J. McD. Corston, Halifax has been re-elected President of Maritime Medical Care at their Annual Meeting on May 28th.

The Canadian Arthritis and Rheumatism Society (CARS) has announced a grant of $100,000 over five years to support research, education and community service activities of Dalhousie University. Director of the medical program will be **Dr. J. Woodbury.**

Dr. C. M. Bethune was honoured at a banquet by the Active Medical Staff of the Victoria General Hospital on May 17th, on the occasion of his retirement as Administrator of the V.G.H., a post which he had held for almost thirty years.



Attending the recent Canadian Ophthalmological Society 32nd Annual Meeting in Halifax were, from left to right: Drs. J. Grimes, New Glasgow; W. Moreside, Charlottetown; D. K. Murray, Halifax, and W. H. Wright, Fredericton. Over 200 eye specialists registered for the meeting.

Dr. Charles A. Roberts, Executive Director of the Clarke Institute of Psychiatry in Toronto, has assumed the position of chairman of the Department of Psychiatry at the University of Ottawa, and psychiatrist-in-chief of the Royal Ottawa Hospital. Dr. Roberts graduated in 1943.

Dr. Alice Kitz, former part-time professor in Anatomy, died on May 20th.

Dr. R. B. Goldbloom and Dr. K. Scott, of the Children's Hospital, Halifax, were participants in the annual Clinical Day of the Prince Edward Island Medical Society. **Dr. L. C. Steeves** represented the Division of Continuing Medical Education of Dalhousie University.

Dr. W. A. Cochrane, former Professor of Paediatrics at Dalhousie University and Head of the Children's Hospital Halifax, and now Dean of Medicine at the University of Calgary, will visit Soviet Russia to study medical education programs and observe the system of health and medical care available to soviet citizens. During his three week tour, Dr. Cochrane will present lectures in Moscow, Leningrad and Kiev, and also at Zagreb and in the United Kingdom.

Dr. C. J. W. Beckwith, retired Executive Secretary of The Medical Society of Nova Scotia and presently Consultant to the Society was awarded an Honorary Membership in the Canadian Public Health Association at their Annual Meeting in Halifax on May 23, 1969.

Dr. Enid MacLeod, Instructor of Physiology at Dalhousie University, has been elected president of the Federation of Medical Women of Canada. Ceremonies took place at the C.M.A. Annual Meeting in Toronto on June 11th.

Medical Grants Top One Million Mark

Eighty-five research grants, fellowships and scholarships worth a total of $1,118,086 have been awarded to members of the Faculty of Medicine at Dalhousie University for 1968-69.

The total exceeds that awarded in 1967-68 by $273,530. This is the first year that the research grants from national agencies have reached a million dollars, putting Dalhousie in a similar position to other Canadian medical schools of similar size.

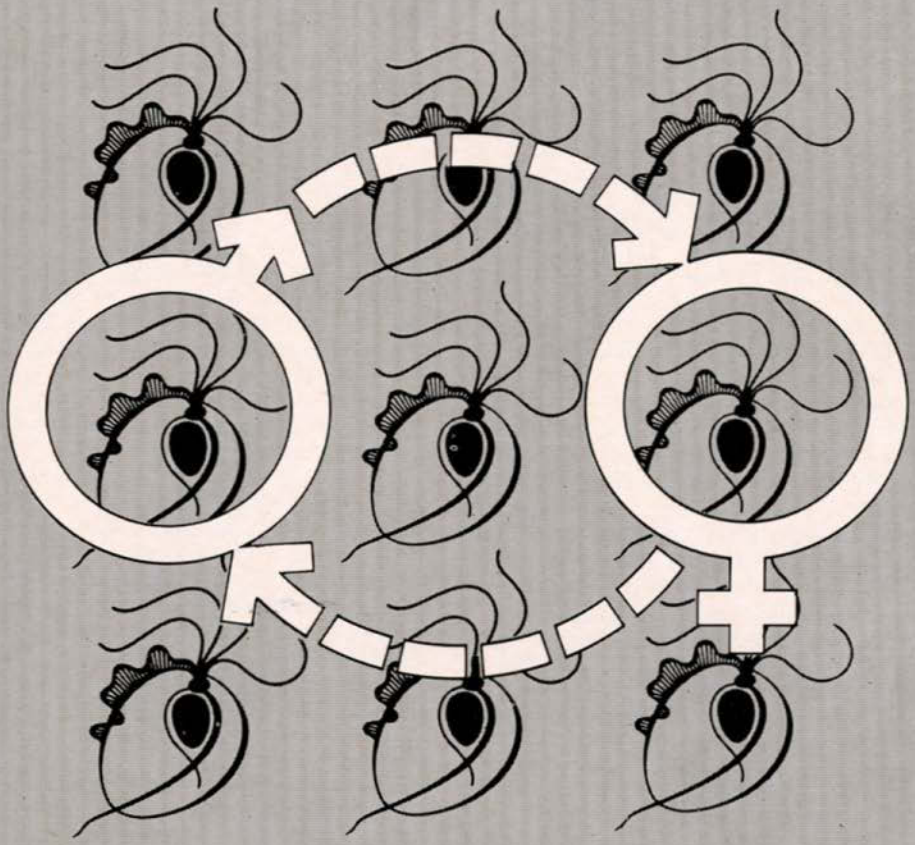
Sixty-three M.D. Degrees were awarded at the Graduation exercises of Dalhousie University this Spring. Approximately half of the graduating class are Nova Scotians.

History was made in Halifax in June with the laying of the cornerstone for the **Izaak Walton Killam Hospital for Children** by Madame Georges Vanier. Also this month the first sod was turned on the site of the **Lane Memorial Hospital** due to replace the existing City Mental Hospital.

OBITUARIES

Dr. Sushil Chandra Sarkar, of Halifax, died June 8, 1969 at the age of 39. He was lecturer in the department of Paediatrics, Dalhousie University, a member of the staff at Children's Hospital, and consultant pediatrician to the Nova Scotia Hospital. He had maintained an active pediatric practice in Dartmouth since 1966. Dr. Sarkar was born in Purnia, Bihar, India. He moved to the United States in 1959 and Canada in 1963. To his wife and son we extend our deepest sympathy.

Dr. Fritz H. Bachman died in Halifax June 4, 1969. Born in Germany April 5, 1900, Dr. Bachman graduated with his M.D. summa cum Laude in 1927. He practiced in Germany until 1933 when he went to South Africa. In 1961 he came to Canada becoming a Canadian citizen in 1966. At the time of his death he was Medical Officer on Research ships of the Department of Energy Mines and Resources. Our sympathy is extended to his wife and son.



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MEDITATIONS ON MORBIDITY

Physician, one day you will become sick. That day will cause you to make many decisions never made before, and your sickness will educate you as never before in the species of human being we know as the patient.

Patients come in many sizes, shapes, and colours, but in their complaints they all fit into the following categories:

1. "There is nothing wrong, I ignore it, it will go away";
2. "There is something wrong, I ignore it, it will go away";
3. "There is something wrong, I will take a pill, it will go away";
4. "There is something wrong, I will take pills, double doses, it will go away"; and finally,
5. "There is something wrong, I must see a Doctor".

Let us consider just Category 5, a hard test for the active Physician who knows he must not trouble another doctor as "Doctors are so Busy"; he also knows that apparently trivial complaints are sometimes suspect and any problem he has must be worthy of complaint. His own objective view of the complaints of others therefore tends to make him a Difficult Patient before he has even made an appointment.

He must also decide in whom he will confide his problem; every doctor is not everyone's cup of tea and he knows a bit too much about some of his colleagues! However the decision is made, the end result of carefully balancing opposing or complementary talents, relating skill to human warmth and sympathy. The appointment is made (don't catch him in the corridor), the diagnosis established the treatment planned; my own result: Surgery. Perhaps elective surgery is hard on the nerves, but urgent matters are welcomed as much as a woman looks forward to the caseroom at term; after eight months it seems unpleasant while after nine it is eagerly anticipated.

The transition from the self-reliant state to one of utter dependence is interesting. I walked, or at least limped, into the Admitting office, but when I left I was cut down in size being in a wheelchair; along the corridors I normally strode with great confidence, now I was gently pushed, a shadow of my old competent self. The transition was completed on arrival in my room where I was required to go to bed - in broad daylight!

Various blood samples, thermometer, sphygmomanometer and an empty bottle to be filled: all at my leisure although I was told "the lab would be closed soon". A coy nurse made the point very clearly that I would certainly not be told the levels of either my temperature or my blood-pressure and having no such curiosity I pondered on her need to protect her professional status by such remarks. My final and weak admission day offering was to write for posterity my own History and Physical.

O.R. day was a day of fasting. Myelography was an interesting experience as the tip of the needle tickled the cauda equina - right foot, left foot and back again. Seeing the dye move up and down on the closed circuit TV was a great diversion as I clutched wildly at the handles on the table while being swung up and down. Finally a preoperative injection and then time stood still: a curious disjointed stretcher journey to the OR, a blur of familiar faces and an instantaneous blackout of pentothal.

The recovery room was a swaying hangover; thoughts and impressions jostled and zoomed as past and recent events gently rearranged themselves. Words formed but speech failed; and after an injection I was scarcely aware of the return trip home to my own little room.

From here on nursing was the biggest factor in my life, a great mixture specially designed to banish boredom, and if I remember the trivia, this is not to discredit its importance. But, bottles of backrub lotion do seem to be handed out to the girls as parking tickets are given to the police for distribution. One day I was offered three within twenty minutes: very diverting. Another afternoon when rather sleepy I had my blood-pressure taken and was surprised to have a return visit from a different nurse for the same purpose only twenty minutes later. The interesting explanation offered was that the first nurse had taken the 2 p.m. blood-pressure, while the second nurse had, on the other hand, been checking the 6 p.m. reading. But, as a glance at the clock showed, it was then precisely 4.15 p.m.! One more nursing memory: since analgesics were difficult to stabilise, nothing working quite as expected, it was one day decided to try a combination of Codeine Capsules and sleeping pills with extras to be left by my bed for a repeat during the night. Here Nursing stepped in for in spite of Doctor's orders I was not allowed to have the reserves by my bedside, rather, I would have to ring, for sleeping pills must *not* be left about. I was therefore given no extra

sleeping pill but Codeine enough to kill myself - about 12 grains.

In an effort to maintain some background of normality I shaved standing at the basin and did not miss a single morning. I am also pleased to report that carrying an I/V bottle on journeys elsewhere is quite practical; perhaps a shoulder harness could be designed to make it even easier.

Eventually I was able to go home on a bright sunny day and as I arrived I knew the whole venture was a success, for my boys' greeting was just this: "Look at Daddy's sideburns"!

A.N.L. □

PANTRY POLITICS

I have a deep and abiding respect for doctors. And this is probably not the proper attitude to have in a Just Society. I might be accused of discriminating. There are all these other professionals including lawyers, businessmen, engineers, scientists, and politicians. And the thing is I probably never had an opportunity to see all these other professionals performing at their best because I've never actually needed any one of these as urgently as I've needed a doctor.

Any crisis, such as a war for instance, brings out the best and also the worst in people, depending on what role they play and in what situation they find themselves. Vast forces are unleashed. All the trimmings are blown away – and what's left is just frightened, partly primitive, people.

Doctors live and work every day in a world of crisis. This leads them to regard such things as racial problems, the unrest of students, the power contest of nations, and the pomposities of politicians, as afflictions of a man-made world brought on by man's shortcomings. It may well be a march to destruction. But the doctor has no time to orate on possibly panaceas. He's too busy picking up the pieces.

The pieces are put together inside a hospital, where priority must be given to the marshalling of forces to fight disease and repair injuries. This particular war has been going on for centuries and if the doctors hadn't been continually gaining ground, a great many of us wouldn't be alive and a great many more would be less than half alive. Nor do the doctors win these battles and come out unscathed, and without casualties in their own ranks. Everyone enlisted here must be highly trained and dedicated. This battlefield is no place for green troops or for armchair admirals.

Two People in One

The last time I was operated on I realized how amazingingly different this world is, I saw one man — an eminent surgeon, change into two different people right before my eyes. When he first showed up he looked quite similar to any business executive — middle aged, medium height, with a wise and kindly mien. So I thought I knew him.

Then came the time to wheel me off to the unknown. When the operation was over I began to emerge from a billowing fog and I heard a voice say "Are you all right?" I opened my eyes and there he was — the handsomest man I ever saw, and I was never so glad to see anyone in all my life — a miracle worker!

A few days later when I could navigate a little bit I woke up in the night and decided I'd make a solo flight to the washroom across the hall. I wasn't sleeping so well and thought it would be a little adventure to relieve the boredom. Nurses don't always approve of little adventures, so I opened the door carefully and sneaked out of my room. Half way across the corridor I glanced toward the nurse's desk, beyond which was a small special care room. The nurse wasn't there at the moment. But an elderly patient was in the bed under a dim halo of light. And standing very still observing her was the same surgeon. He had looked in on me about 10 p.m., and I'd heard his voice in the corridor at midnight. I thought "when does this man rest?"

Nobody noticed me. I stood still, almost afraid to move for fear I'd blunder or make a noise, and break a spell. And as I observed this scene, all of a sudden that man looked at least 10 feet tall. Ever since that my eyes occasionally play tricks on me. One evening recently I saw Dr. Dunsworth, president of our Medical Association, a guest on TV, discussing Medicare, — and he looked 10 feet tall.

Doctors live in a world of crisis. And on this battleground the "good guys" don't always win. And the hero doesn't go galloping into the sunset on a white horse. Some of us are aware that in this kind of outdated melodrama, the real hero is the horse. And chances are he'd go farther and faster if he got to pick his own rider. □

By Helen Hogan-Dartmouth Free Press

News Flashes

THE MEDICAL SOCIETY OF NOVA SCOTIA

116th Annual Meeting
5th Meeting of Council

The 116th Annual Meeting of The Medical Society of Nova Scotia and the 5th Meeting of Council will take place at the Hotel Nova Scotian. The Annual Executive Meeting will be held on Sunday, November 16th with the first Meeting of Council to commence on Monday morning, November 17th.

The activities of the Medical Society and the Dalhousie Refresher Course are being more closely co-ordinated than in previous years with the hope of increasing attendance at both. Plans call for termination of Medical Society business at 12:30 p.m. on Tuesday, November 18th, with a mixed buffet luncheon to follow in conjunction with doctors arriving to attend the Refresher Course. The Refresher Course Program is being especially tailored to interest all members of the Profession.

Tuesday evening will see a full-scale combined social program with the Dalhousie Medical Alumni and the Medical Society joining together for the Reception, Banquet and Ball at the Hotel Nova Scotian.

Perhaps it is not too soon to lay your plans and make your reservations for the Annual Meeting this coming November.

BIOMEDICAL ENGINEERING TECHNICIANS

The Algonquin College of Applied Arts and Technology announces the graduation of its first class of Biomedical Engineering Technicians. These students, starting with a Grade 12 education, receive a four-semester course of electronic theory and practice and basic medicine, combined with field work in affiliated hospitals during the summer recess.

A Biomedical Engineering Technician is employed by a hospital to operate, test and repair modern medical electronic equipment. Electronic equipment used for diagnosis, patient monitoring and operating room procedures is being added to every day, and hospitals require technicians with a comprehensive theoretical knowledge and a good mechanical aptitude in order that they may thoroughly utilize this equipment. They will work under the direction of doctors and Bio-medical engineers.

The Algonquin Technical Centre, 1385 Woodroffe Ave., Ottawa, Ont.

THE 43rd. DALHOUSIE REFRESHER COURSE

Dalhousie's Annual Refresher Course gets under way at the Sir Charles Tupper Medical Building on Tuesday November 18th, 1969. The Four days will include thirteen hours devoted to **Small Group Clinics,** three **Medical Grand Rounds,** two simultaneous **Family Physician Case Conferences, Pediatric Grand Rounds** and three **Socratic Luncheons.**

These sessions are designated as the Scientific Program of the Annual Meeting of The Medical Society of Nova Scotia, who will sponsor a Banquet and Ball jointly with the Dalhousie Medical Alumni on Tuesday evening at the Hotel Nova Scotian.

Associated meetings of other groups such as the Atlantic Provinces Orthopaedic Society, and the Nova Scotia Society of Ophthalmologists and Otolaryngologists will be held.

The John Stewart Memorial Lecture will be delivered by Dr. R. A. Good, Professor of Paediatries, and Microbiology, University of Minnesota, and the Visiting Lecturer in Orthopaedics will be Dr. F. R. Tucker, Professor of Surgery (Orthopaedics) University of Manitoba.

Plan now to attend the Dalhousie Refresher Course, November 18th to 21st inclusive. Further details from Dr. J. F. Filbee, Chairman, Dalhousie Refresher Course Committee, Division of Continuing Medical Education, Dalhousie University Halifax, N. S.

CHILD ACCIDENT FACTS

Accidents are not inevitable. They can be prevented, but, too often, we make mistakes, are ignorant of the hazards involved in certain situations or activities, or permit obvious hazards to go uncorrected. In short, when an accident happens, somebody has failed. Every 15 minutes in this nation, a child is accidentally poisoned. In 1967, accidental poisoning took the lives of 32 children under fifteen years of age. 25 were under five, 8 were due to the misuse of aspirin or salicylates. The cause in nearly every instance was carelessness. Too often adults do not take the few seconds of time required to put pills away in the medicine cabinet, or to place household chemicals, garden sprays and other toxic substances out of reach.

Poisoning is one of the easiest home accidents to avoid, but parents must fight a continual battle against carelessness. Never assume a bottle of pills or tablets on top of a bedroom bureau or kitchen sink cannot be reached. Never leave medicine anywhere but in the medicine cabinet.

Council on Family Health in Canada.

News Flashes

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

ADAPTING THE MARCH OF DIMES

The Ontario 'March of Dimes' campaign organised by that province's Rehabilitation Foundation for the Disabled will switch over this year to a new name and a new symbol. This change will emphasize the role of rehabilitation for all types of disabilities, not only those associated with polio, the original disability dealt with by the March of Dimes Campaign. Called 'The Ability Fund', the campaign will use the symbol of an evergreen tree with one branch missing, accompanied by the slogan 'Though much is taken, much remains'.

The new symbol has been offered to, and accepted by, the Canadian Rehabilitation Council as a national symbol of help for disabled adults. The name of 'The Ability Fund' has also been offered to the Council for national use.

FORTHCOMING MEETINGS

Dalhousie University is offering the following short courses:

Symposium on Nuclear Medicine – Sept. 5 & 6, 1969. Experts in the field of Nuclear Medicine from Canada and the U.S.A. will be in attendance.

The American College of Physicians announces the following postgraduate courses:

Clinical Gerontology	Aug. 21-23, 1969.
Medical Oncology	Sept. 1969.
Vascular Disease	Oct. 1-4, 1969.
Office Psychiatry for Internists	Oct. 20-24, 1969.

Symposiums will be held in New York:

The Fifth Annual Symposium on Air Pollution and Respiratory Disease. October 23, 1969.

The Pharmacology of Selected Drugs used in Dermatology: Oct. 29-31, 1969. Principles of Action and Uses.

17th Annual Meeting of the Canadian Association of Physical Medicine and Rehabilitation to be held in Halifax, N.S. August 21-22-23, 1969.

Annual Assembly of The College of Family Physicians of Canada, Toronto, Ont., Sept. 29 to Oct. 2, 1969.

The Medical Society of Nova Scotia	
116th Annual Meeting	
5th Meeting of Council	Nov. 17 & 18, 1969
43rd Dalhousie Refresher	
Course	Nov. 18 to 21, 1969

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.

GROUP LIFE INSURANCE PLAN MEDICAL SOCIETY OF NOVA SCOTIA

A recent amendment to the Contract with the North American Life now permits insured members to retain their protection under the Plan even though they have retired from active practice. Membership in good standing in the Society is, of course, still a requirement as well as being a resident of Canada.

News Flashes

THE SUDDEN DEATH SYNDROME

The very strong feelings of guilt and anxiety associated with the tragedy of an apparently healthy infant being found dead in its crib prompted Drs. Abraham B. Bergman, Joyce D. Miller and J. Bruce Beckwith to develop an information sheet to give the facts about this syndrome to grieving parents. This fact sheet was published by the American Academy of Paediatrics, and has been drawn to the attention of the Editorial Board by Dr. Bruce Morton, who has obtained permission from the authors to reproduce this information for doctors in this Province. Copies of the fact sheet may be obtained by writing the offices of The Medical Society of Nova Scotia.

DRUGS AND THE TEENAGER

During the past three months, Dr. Mark Segal of the Department of Pharmacology, Dalhousie University, has spoken at many meetings of parents and students in the Halifax Dartmouth area, to students at Antigonish High School, and at St. Francis Xavier and Acadia University, concerning the use and abuse of drugs. Dr. J. G. Aldous and Mr. R. K. Siegel, a psychologist, participated in a discussion of this subject at the Allied Youth Provincial Conference in Wolfville on April 10th, and Dr. Bruce Morton spoke to the Harbor-View Home and School Association on the effects of drugs on school age children. This reflects the widespread interest in, and reaction to, the use of drugs among teenagers in the Province.

ADVERTISERS' INDEX

Clinical Staff Conferences Effective Aug. 1969

PATHOLOGY INSTITUTE

Seminars in Pathology and Bacteriology	Monday	4:00 p.m.	Lecture Rm. 207	Weekly
Surgical Pathology	Tuesday	4:00 p.m.	Lecture Rm. 207	Weekly
Gynecological Pathology	Tuesday (1st of mo.)	5:00 p.m.	Lecture Rm. 207	Monthly
Neurosurgical Pathology Clinical	Wednesday (1st of mo.)	8:00 a.m.	Autopsy Room	Fort-Monthly
Neuropathology Slide Cont.	Thursday	8:00 a.m.	V.G. Hosp.	Weekly
Completed Case Conference	Friday	2:30 p.m.	Lecture Rm. 207	Weekly
Autopsy Cases (Gross)	Friday	3:30 p.m.	Surgical Reading Rm.	Weekly
Autopsy Cases Quiz Cont.	Friday	4:00 p.m.	Lecture Rm. 207	Weekly

THE CHILDREN'S HOSPITAL

Admission Rounds	Monday		9:00 a.m.
Hematology Rounds*	Monday		12:00 noon
Genetic Rounds*	Monday		12:00 noon
Cardiology Rounds	Tuesday		10:30 a.m.
Case Presentation and Discussion	Tuesday		4:00 p.m.
Medical Grand Rounds	Wednesday		9:00 a.m.
Infectious Disease Rounds*	Wednesday		12:00 noon
Respiratory Rounds*	Wednesday		12:00 noon
Physician-in-Chief's Rounds	Monday	– Gyro I	10:30 a.m.
	Tuesday	– Gyro II	9:30 a.m.
	Wednesday	– B ward	10:30 a.m.
	Thursday	– I.D.U.	10:30 a.m.
	Friday	– 1st South	10:30 a.m.
Admission Rounds	Thursday		9:00 a.m.
Metabolic Rounds*	Thursday		12:00 noon
Neurology Rounds*	Thursday		12:00 noon
Pathology Rounds*	Thursday		3:30 p.m.
Radiology Rounds*	Thursday		3:30 p.m.
Surgical Grand Rounds	Friday		11:00 a.m.
Basic Science Seminar	Friday		12:30 p.m.
Orthopaedic Rounds	Friday		2:00 p.m.

*Alternates Weekly

GRACE MATERNITY HOSPITAL

Nursery Ward Rounds	Daily	8:00 a.m.
Ward Rounds	Daily	10:00 a.m.
Staff Meeting	Monday (Last)	12:00 noon
Endocrine & Infertility Clinic	Monday	10:00 a.m.
Nursery Chart Rounds	Monday (First)	2:00 p.m.
Perinatal Mortality Review	Monday (Third)	5:00 p.m.
Obstetrical Conference	Tuesday (Third)	5:00 p.m.
Journal Club Luncheon	Thursday	12:15 p.m.
Prenatal Clinic	Tuesday, Thursday, Friday	2:00 p.m.
Well Baby Clinic	Tuesday, Thursday, Friday	2:00 p.m.
Postnatal Clinic	Tuesday, Thursday, Friday	2:00 p.m.
Nursery Radiology Conference	Friday (Second)	1:00 p.m.
Family Planning Clinic	Wednesday (by appointment)	2:00 p.m.

This listing is as complete as possible, and all excerpts listed are open to any interested Physician.
Information regarding other areas of the Province is welcomed and will be published when available.

HALIFAX INFIRMARY

Department of Anaesthesia			
Department Meetings	1st & 3rd Monday	2:00 p.m.	O.R.Suite
Department of General Practice			
Grand Rounds	Tuesday	8:30 a.m.	4A Clinic Room
Department of Medicine			
Grand Rounds	Thursday	11:30 a.m.	4C Clinic Room
Intern-Resident Conference	Friday	12:00 Noon	4C Clinic Room
Monthly Meeting	1st Tuesday	5:30 p.m.	3C Clinic Room
Department of Obstetrics and Gynecology			
Daily Conference	Mon.-Fri.	9:00 a.m.	3C Clinic Room
Weekly Rounds	Friday	12:00 Noon	3C Clinic Room
Monthly Meeting	4th Tuesday	5:00 p.m.	3C Clinic Room
Department of Ophthalmology			
Weekly Conference	Tuesday	6:30 p.m.	Outpatient Dept.
Monthly Conference	3rd Tuesday	4:30 p.m.	3C Clinic Room
Department of Otolaryngology			
University Dept. Joint Meeting	4th Tuesday	7:00 p.m.	Alternate Hospitals
Department of Pathology			
Staff Education Conference	4th Wednesday	12:00 Noon	Auditorium
Department of Psychiatry			
Daily Ward Rounds		8:00 a.m.	Psychiatric Dept.
Case Presentation	Wednesday	9:00 a.m.	2A Clinic Room
Monthly Conference	3rd Wednesday	9:00 a.m.	2A Clinic Room
Department of Radiology			
House Staff Conference	Tuesday	1:00 p.m.	Radiology Dept.
Weekly Conference	Thursday	3:30 p.m.	Radiology Dept.
Department of Therapeutic Radiology			
Grand Rounds	1st & 3rd Mondays	12:30 p.m. 12:30 p.m.	3C Clinic Room 3C Clinic Room
Department of Surgery			
Weekly Grand Rounds	Wednesday	8:00 a.m.	
Monthly Departmental Meeting	2nd Wednesday	12:30 p.m.	3C Clinic Room
Department of Urology			
Weekly Conference	Thursday	12:00 Noon	Urology Dept.
Monthly Meeting	Last Thursday	4:30 p.m.	Urology-Dept.

Department of Medicine

Cardiac Working Conference	Monday	1:00- 2:00 p.m.	Room 4-017
Metabolism & Endocrinology	Monday (1st & 3rd)	1:00- 2:00 p.m.	Room 3-077
Renology	Monday (2nd & 4th)	1:00- 2:00 p.m.	Room 3-077
Medical Grand Rounds	Tuesday	8:30-10:00 a.m.	Room 3-077
Cardiology	Tuesday (1st & 3rd)	1:00- 2:00 p.m.	Room 3-077
Pulmonary	Tuesday (2nd & 4th)	1:00- 2:00 p.m.	Room 3-077
Haematology (Out Patients)	Tuesday	2:00- 4:30 p.m.	OPD 3rd Floor
Neurology-Neurosurgery	Wednesday	9:00-10:00 a.m.	Room 11-018
Haematology	Wednesday (1st & 3rd)	1:00- 2:00 p.m.	Room 3-077
Gastroenterology	Wednesday (2nd & 4th)	1:00- 2:00 p.m.	Room 3-077
Rheumatology (Out Patients)	Wednesday	11:00-12:00 a.m.	OPD 3rd Floor
Resident Orals	Thursday	1:00- 2:00 p.m.	Room 4-017
Neurology	Friday (1st & 3rd)	1:00- 2:00 p.m.	Room 3-077
Rheumatology	Friday (2nd & 4th)	1:00- 2:00 p.m.	OPD Conference Rm.
Cardiology	Friday	2:00- 4:00 p.m.	OPD 3rd Floor

Department of Surgery

Weekly Clinical Conf.	Saturday	10:30 a.m.	Room 11-018
Surgical Pathology Conference	Tuesday	4:00 p.m.	Path. Bldg.
Ward Rounds			
Surgery A	Friday	9:00 a.m.	7B
Surgery B	Thursday	8:00 a.m.	
Surgery C	Wednesday	8:30 a.m.	6A
Surgery D	Saturday	9:00 a.m.	7A
Orthopaedics	Tuesday	11:00 a.m.	7A & B
Out Patients Clinics			
Surgery A	Friday	9:30 a.m.	Outpatient Dept.
Surgery B	Thursday	9:30 a.m.	Outpatient Dept.
Surgery C	Wednesday	9:30 a.m.	Outpatient Dept.
Surgery D	Tuesday	9:30 a.m.	Outpatient Dept.
Orthopaedic	Tuesday	9:00 a.m.	Outpatient Dept.

Department of Psychiatry

Ward Rounds	Monday & Friday	10:30 a.m.	Room 9-064
Seminar	Friday	4:00 p.m.	Room 9-023
Child Guidance Clinic	Thursday	9:00 a.m.	Child Guidance Clinic
Case Presentations	Monday, Tuesday, Friday & Saturday	9:00 a.m.	Room 9-023

Department of Urology

Conference	Monday Wednesday & Friday	4:30 p.m.	5B
Seminar	Tuesday	4:30 p.m.	X-ray Conf. Room
Opd. Clinic	Thursday	10:00 a.m.	Out Patient Dept.

Department of Gynaecology

Ward Rounds	Daily	9:00 a.m.	5W & 5N
Grand Rounds	Monday	8:00 a.m.	5th Floor Clinic Rm.
Pathology Conference	Tuesday	5:00 p.m.	5th Floor Clinic Rm.
Tumour Clinic	Tuesday & Friday	11:30 a.m.	Tumour Clinic
Gyn. Outpatient Clinic	Monday	2:00 p.m.	Outpatient Dept.
Gyn. Post-op Clinic	Wednesday	2:00 p.m.	Outpatient Dept.

Department of Anaesthesia

Conference	Last Monday	3:30 p.m.	10-021
Residents' Seminar	Thursday	7:00 p.m.	10-021
Journal Club	2nd Tuesday	7:00 p.m.	10-021
Intensive Care Rounds	Daily	2:30 p.m.	10-021

Department of Radiology

Nuclear Medicine Conference	Tuesday, Wednesday, Thursday & Friday	2:00 p.m.	Radiology Dept.
Therapeutic Radiology Ward Rounds	Thursday	8:30 a.m.	11th Floor, Victoria
Diagnostic Radiology Conference	Daily	3:00 p.m.	Radiology Dept.
Proven Case Conference	Thursday	1:00 p.m.	Radiology Dept.
Clinical Conference	Thursday (1st)	5:15 p.m.	Radiology Dept.
Departmental Conference	Friday	3:00 p.m.	Radiology Dept.
Radiological & Pathological Conference	Thursday	4:30 p.m.	Radiology Dept.

Nova Scotia Tumour Clinic

Clinics			
Rectal	Wednesday	10:00 a.m.	Tumour Clinic
Breast	Monday	2:00 p.m.	Tumour Clinic
Gynaecology	Tuesday	10:00 a.m.	Tumour Clinic
Skin, Soft Tissue	Tuesday	11:00 a.m.	Tumour Clinic
Colon	Tuesday	11:00 a.m.	Tumour Clinic
Leukemia & Lymphoma	Tuesday	2:00 p.m.	Tumour Clinic
Paediatric	Tuesday (Fourth)	2:00 p.m.	Tumour Clinic
Ophthalmology	Tuesday	2:30 p.m.	Outpatient Dept.
Head & Neck	Monday	10:30 a.m.	Tumour Clinic
Otolaryngology	Wednesday	11:00 a.m.	Outpatient Dept.
Neurosurgery	Wednesday	11:00 a.m.	Outpatient Dept.
Urology	Wednesday	10:00 a.m.	Tumour Clinic
Breast	Thursday	11:00 a.m.	Tumour Clinic
Pulmonary	Friday	12:00 noon	Tumour Clinic
Gastric & Esophageal	Friday	12:00 noon	Tumour Clinic
Orthopaedic	2nd Friday	10:00 a.m.	Tumour Clinic
Gynaecology	Friday	11:00 a.m.	Tumour Clinic