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Good Nutrition: A natural necessity

"An army marches on its stomach". Napoleon

It is not just armies that deserve good food — everyone does. Whilst in Canada, unlike many of the developing countries, we have adequate food reserves, the very plethora of nutriment, the wide variety of choice and the commercial exploitation of processed foods, have all produced a population with a far from perfect healthy profile.

For several decades we encouraged mothers to bottlefeed their babies. Children were sent to school with sketchy midday meals, and pop and chips became the standard food supplements for every teenager. Succulent candies brought carious teeth and even Eskimo children suffered from devastating dental decay.

Obesity became commonplace. The automobile and television also contributed to the high incidence of arteriosclerosis, coronary artery disease and gallstones. Too late, many of our adult population desperately studied the cholesterol content of their food, or prepared to submit to delicate arterial surgery.

Fortunately, a more rational approach is possible. There is a great awakening of interest in nutrition, as was evident by the 600 in attendance at the Canadian Dietetic Society meeting, held recently at the Hotel Nova Scotian. Yet, the impact is not fully appreciated by all practising physicians, for this Society is largely a woman's world and very few physicians attended. Nevertheless, the subjects discussed were of wide significance and included breast feeding, new methods of preparing and distributing food, new containers, supplements for senior citizens, hospital and cafeteria food, food safety, and the whole realm of children's nutrition as well as parenteral supplements.

Advances in technology have been stupendous, and it is now possible to detect one part of a substance in a trillion. Some 50,000 potential carcinogens have been isolated in our environment. Wise standards for nutrition, ideal body weight and shape are now tabulated, and we can compare ourselves and others against these. A great effort is being made to educate the public but much ignorance remains. A recent survey of children, selecting foods from vending machines at lunchtime, showed no correlation between the items chosen and their nutritive value.

This *Journal* is pleased to present a selection of papers on nutrition from the Department of Medicine, from the Dietetic Department of the Victoria General Hospital, and from the Nutrition Section of The Medical Society of Nova Scotia. The subjects of cardiovascular disease, sodium and cholesterol metabolism are well covered. Cholesterol metabolism is now understood much better, and the control of hyperlipidaemia can be achieved by a combination of diet, vigorous exercise and special biochemical agents.

Physicians need to assume a more active role in advising their patients about good eating habits — teaching children to avoid junk food and sucking candies, and impressing on mothers the importance of breast feeding. Resource information is available. For example, there is an excellent tape on "Nutritional Challenges Throughout Life" available in the Kellogg Health Sciences Library.

Nova Scotia is rich in recipes for good food. MicMac, Acadian, English, New Englander, Scot and Negro have all contributed their traditional fare. It is only in the last decade that these nutritious blends have been replaced by homogenized junk food and pop as a widely acceptable diet. Our Scots ancestors knew better and their longevity is renowned. It is up to physicians and nutrition experts to get together and ensure that the nation's diet is more nutritive, active and scientifically sound. □

References on page 108.

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Cholesterol Metabolism

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Dietary cholesterol, which is insoluble in water, is absorbed in the upper small bowel only after it is incorporated into mixed bile acid and phospholipid micelles from bile. In healthy adults, median dietary intake varies from 325-600 mg a day but only 3-4 mg/Kg body weight is actually absorbed, i.e., up to only about 50%.

After absorption, cholesterol is transported in chylomicrons in lymph and eventually enters the blood stream. Electron microscopy shows that the lipoprotein is present mainly as discs. Under the influence of lecithin-cholesterol acyl transferase (LCAT), which controls the amount of esterified cholesterol in blood, these discs change shape at the tissue level to become spheres. The spheres represent chylomicron remnants which transport cholesterol esters from body tissue to the liver.

Hepatic bile contains non-esterified cholesterol solubilized by bile acid and phospholipid (mainly lecithin). Like dietary cholesterol, this endogenous cholesterol can only be absorbed in the duodenum and upper jejunum by micellar incorporation. Thus, cholesterol undergoes an enterohepatic circulation. Interruption of this cycle by any mechanism impairs cholesterol absorption, resulting in increased fecal loss.

Cholesterol is capable of being synthesized from acetate in virtually every cellular tissue (including arteries). However, the major synthesis occurs in the liver, with the small bowel mucosal cell (particularly in the distal ileum) being secondary. Hepatic synthesis of cholesterol is controlled by feedback inhibition of the rate-limiting enzyme, β -hydroxy- β -methyl glutaryl CoA (HMGCoA) reductase by cholesterol itself. Thus, dietary cholesterol can inhibit endogenous hepatic cholesterol production. This does not hold true for cholesterol synthesis of small bowel origin. Here, feedback control occurs from bile acids, as shown by diversion and specific inhibition studies.

The body can get rid of cholesterol directly in bile or via its major metabolite, bile acid, which is made in the liver. Two primary bile acids, cholic and chenodeoxycholic acid, are oxidative degradation products of cholesterol; they are conjugated with glycine or taurine prior to secretion in bile. In the gut lumen, bacterial degradation at the 7 α -OH position converts cholic acid to deoxycholic acid and chenodeoxycholic acid to lithocholic acid. Only deoxycholic acid is absorbed to any appreciable extent; it undergoes enterohepatic cycling via the portal vein, and hepatic conjugation with glycine or taurine before secretion into bile. No bile acid itself exerts any direct control on hepatic cholesterologenesis.

The exact contribution to blood levels of either dietary or endogenous cholesterol production must vary widely from time to time. Some evidence suggests that 60% of blood cholesterol is of endogenous origin. If this is so, dietary manipulation would not be expected to influence serum levels to any great extent, as is seen in clinical practice.

Effective control of hyperlipidemia requires a two (or more) pronged approach; reduction of dietary cholesterol should be combined with the products of pharmacologic wizardry aimed at different parts of the cholesterol metabolic cycle (for example, the use of cholestyramine which binds bile acid, interfering with micelle formation and resulting in reduced absorption). Other specific non-toxic agents are needed which inhibit HMGCoA reductase or increase cholesterol breakdown to bile acid by stimulating the rate-limiting enzyme 7 α -hydroxylase. □

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Diet and Cardiovascular Disease

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Cardiovascular disease is one of the major health problems affecting Canadians. Fifty percent of all deaths are related to arteriosclerosis, and up to one third occur before age 65 years. Risk factors associated with cardiovascular disease (hyperlipidemia, hypertension, cigarette smoking, and diabetes mellitus) and less well documented factors (obesity, lack of physical fitness) suggest that preventive measures may influence cardiovascular disease.

In 1974, an expert committee was appointed by the federal government to examine the relationship between diet and cardiovascular disease and make appropriate recommendations. In 1977, the *Report of the Committee on Diet and Cardiovascular Disease* was published by Health and Welfare, Canada¹. The background to this report was fully accepted, but several recommendations were amended for feasibility in implementation². These recommendations are preventive measures for well adult-populations and not therapeutic diets for established disease.

RECOMMENDATIONS

- (a) Follow Canada's food guide³ (see Table).
- (b) Avoid junk food, i.e., calories with no essential vitamins or minerals.
- (c) Reduce calories from fat to 35 percent; eat polyunsaturates, e.g., linoleic acid (found in sunflower, corn, soybean, and peanut oils).
- (d) Emphasize whole grain products, fruits, and vegetables and observe moderation in alcohol, salt and refined sugar intake.
- (e) Prevent and control obesity by reducing calories and increasing physical activity.

SUGGESTED GUIDELINES

Fifty percent of calories should come from carbohydrates, of which about 50% or less should be refined. Refined sugar should be replaced with starches. Thirty-five percent of calories should come from fat, including polyunsaturates. Fifteen percent of calories should be obtained from protein, using lean meats, fish, or poultry. High calorie snack foods and beverages, including alcohol, should be limited. Use cooking methods that reduce fat intake, e.g., baking, broiling, boiling. Limit salt at the table and in cooking.

These principles are endorsed by The Medical Society of Nova Scotia. The following points serve as rationale for the recommendations and/or guidelines.

1. Reduction of dietary cholesterol in patients with hyperlipidemia leads to reduced serum cholesterol levels and

eventual loss of tissue cholesterol as shown by arteriography in subjects with intermittent claudication (see the article by Dr. Tan). The Medical Society takes the position that it seems reasonable for healthy adults to reduce their dietary cholesterol intake from the generally quoted levels of 600 mg/day for North Americans to 400 mg/day or less, as the original report suggested¹. National Health and Welfare did not accept this recommendation, based on the studies quoted^{4,6}, but did stress, as does the Government of Nova Scotia, that reduction of fat *per se* will achieve the same result.

All parties agree that dietary cholesterol intake should be reduced in subjects with hyperlipidemia to reduce, in particular, the risk of coronary artery disease. Although eggs have a high cholesterol content, they are an excellent quality food and should be maintained. Thus, one egg per day can be eaten as long as other sources of cholesterol or saturated fats are reduced. This can readily be achieved by substituting fish or poultry for red meats (as discussed by Mrs. MacIntosh in this issue). Ideally, fat for deep frying should be limited to the polyunsaturated type, although cost may limit this.

2. Excessive use of salt may lead to hypertension. Consequently, reduced use at the table as well as during cooking is the easiest way to reduce overall intake since most manufactured foods have added salt (see the article by Dr. Hindmarsh in this issue).

3. Exercise and good eating habits go together. The ideal place to educate populations in nutrition is in the schools. The need for daily exercise both in promoting general well being and as an aid to preventing obesity is self-evident; this is discussed by Dr. Wheeler in a later article. □

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†Department of Health, Nova Scotia Government.

Eat a variety of foods from each group every day

Energy needs vary with age, sex and activity. Foods selected according to the guide can supply 1000-1400 calories. For additional energy, increase the number and size of servings from the various food groups or add other foods.

milk and milk products

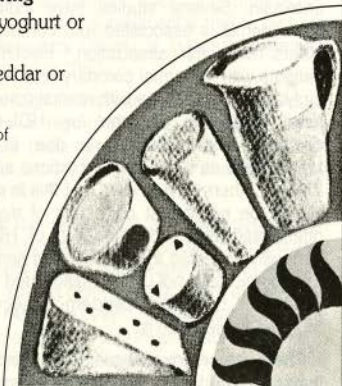
Children up to 11 years	2-3 servings
Adolescents	3-4 servings
Pregnant and nursing women	3-4 servings
Adults	2 servings

Skim, 2%, whole, buttermilk, reconstituted dry or evaporated milk may be used as a beverage or as the main ingredient in other foods. Cheese may also be chosen.

Examples of one serving

- 250 ml (1 cup) milk, yoghurt or cottage cheese
- 45 g (1½ ounces) cheddar or process cheese

In addition, a supplement of vitamin D is recommended when milk is consumed which does not contain added vitamin D.



meat and alternates

2 servings

Examples of one serving

- 60 to 90 g (2-3 ounces) cooked lean meat, poultry, liver or fish
- 60 ml (4 tablespoons) peanut butter
- 250 ml (1 cup) cooked dried peas, beans or lentils
- 80 to 250 ml (½-1 cup) nuts or seeds
- 60 g (2 ounces) cheddar, process or cottage cheese
- 2 eggs



bread and cereals

3-5 servings

whole grain or enriched. Whole grain products are recommended.

Examples of one serving

- 1 slice bread
- 125 to 250 ml (½-1 cup) cooked or ready-to-eat cereal
- 1 roll or muffin
- 125 to 200 ml (½-¾ cup) cooked rice, macaroni, spaghetti



fruits and vegetables

4-5 servings

Include at least two vegetables.

Choose a variety of both vegetables and fruits — cooked, raw or their juices. Include yellow or green or green leafy vegetables.

Examples of one serving

- 125 ml (½ cup) vegetables or fruits
- 125 ml (½ cup) juice
- 1 medium potato, carrot, tomato, peach, apple, orange or banana



Diet, Hyperlipidemia and Cardiovascular Disease

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The clinical importance of hyperlipidemia lies in its association with atherosclerosis, pancreatitis, and xanthomatosis. This discussion considers the association between hyperlipidemia and cardiovascular disease. (Hyperlipidemia means hypertriglyceridemia, hypercholesterolemia, or both.) It is important to realize that hyperlipidemia is only one of many risk factors associated with the development of atherosclerosis. The Seattle myocardial infarction study showed that only 31% of 500 survivors of myocardial infarction had hyperlipidemia.¹ In that study, 16% of patients had hypertriglyceridemia, 8% hypercholesterolemia, and 7% both.

Lipids are insoluble in water. They are complexed with proteins to form lipoproteins which are soluble. The three lipids (cholesterol, triglycerides, and phospholipids) are complexed with varying proportions of proteins to form four major classes of lipoproteins which can be separated either by ultracentrifugation according to their densities or by electrophoresis according to their mobilities (Table I). Each class of lipoproteins contains varying proportions of triglycerides, cholesterol, phospholipids, and apoproteins.

TABLE I
SERUM LIPOPROTEIN FRACTIONS

Ultracentrifugation fractions	Electrophoretic mobility
Chylomicrons	Stays at origin
Very Low Density Lipoproteins	pre- β
Low Density Lipoproteins	β
High Density Lipoproteins	α

In the fasting state, most of the serum cholesterol is carried in low density lipoprotein (LDL) and most serum triglycerides in very low density lipoproteins (VLDL). In the fed state, most serum triglycerides are present in the chylomicrons. An increase in total serum cholesterol can be due to LDL, high density lipoprotein (HDL) or VLDL cholesterol (LDL-C, HDL-C, and VLDL-C). An increase in total serum triglycerides can be due to VLDL or chylomicron triglycerides. Elevations of different fractions of lipoproteins have different clinical implications; for example, elevation of LDL-C is associated with premature coronary artery disease, whereas, elevation of HDL-C is associated with longevity and lack of cardiovascular disease.^{2,3} Reduction of HDL-C has been associated with increased prevalence as well as incidence of coronary artery disease.^{4,5}

Previously, the Framingham study established total serum cholesterol as a risk factor but more recently has questioned the usefulness of serum cholesterol as a predictor of coronary artery disease.⁴ This is because the use of cholesterol content in various lipoprotein fractions has shown new and better predictors for coronary artery disease. The Framingham study showed that the combined measurements of LDL-C, HDL-C, and total cholesterol formed the best predictor for coronary artery disease.⁴ It appears that lipoprotein fractionation can provide better information.

Whether hypertriglyceridemia is a risk factor remains uncertain. Several studies have indicated that hypertriglyceridemia is associated with coronary artery disease;^{1,6} others refute this association.⁴ Recently we showed that patients with abnormal coronary arteries have higher serum triglycerides than those with normal coronary arteries.⁷ It has been suggested that the low HDL-C in patients with coronary artery disease is due to their high serum triglycerides as the two lipid fractions are inversely related.⁸ We and others have shown that this is not so.^{4,7} However, it should be noted that correction of hypertriglyceridemia is often associated with elevation of HDL-C.⁹ Finally, it is generally accepted that elevation of serum triglycerides due to chylomicron is not usually associated with atherosclerosis, whereas, hypertriglyceridemia secondary to VLDL-triglyceride elevation is.

There are many causes for hypercholesterolemia and hypertriglyceridemia. Whether the hypercholesterolemia of obstructive liver disease and hypothyroidism have the same prognosis is not well established. I feel that familial hypercholesterolemia (\uparrow LDL-C) carries a worse prognosis than any of the secondary hypercholesterolemia. In addition, I feel that the duration of hypercholesterolemia is important. Life-long exposure is particularly detrimental. Whether hypercholesterolemia in the familial cases is the critical variable in predisposing the patient to developing premature atherosclerosis is not proven. It is possible that another factor (Factor X) associated with cholesterol is the important variable. More research needs to be done.

In managing hyperlipidemia, it is important to determine the cause of the metabolic disorder. If there is a secondary cause, treatment of that problem often corrects the hyperlipidemia. Diet manipulation is common to all management of hyperlipidemia; one must consider weight control, alcohol restriction, cholesterol restriction, and alteration in the composition of fat in the diet. If diet alone is unsuccessful in lowering the serum lipids, a hypolipemic agent is used. Regular patient follow-up is an essential part of management.

Elsewhere in this issue of the *Bulletin*, the recommendations of the Committee on Diet and Cardiovascular Disease are listed. Included are restriction of daily cholesterol intake and decrease in saturated fat intake. How does a diet high in cholesterol and saturated fat affect the lipoprotein fractions? In rats, such a diet decreased the HDL-C and increased

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LDL-C.¹⁰ In man, several studies have shown that such a diet increased the total cholesterol and LDL-C. The effect on HDL-C is less well established. Others have shown little or no increase in HDL-C,^{11,12} whereas, we found a definite increase in HDL-C in six subjects after three weeks of a high cholesterol and saturated fat diet.¹³ Because the LDL-C and VLDL-C were also significantly increased, we do not recommend this as a means of increasing the HDL-C. Others have shown that a diet high in polyunsaturated fat will lower total serum cholesterol by decreasing the cholesterol in HDL, LDL, and VLDL.¹⁴

Does dietary-induced hypercholesterolemia cause atherosclerosis? In various animal species, the answer is yes^{15,16} but the connection has not been proven in man. Does lowering serum cholesterol lead to a regression of atherosclerosis? Again the answer in animals is positive^{17,18} but there are few studies in man. Two recent reports suggest that reducing serum lipids with modification of other risk factors in hyperlipoproteinemic man leads to regression of atherosclerosis in femoral arteries¹⁹ and lack of progression of coronary atherosclerosis.²⁰ A multicentre trial to test this hypothesis (Lipid Research Clinics) is currently underway and results will be known in 1983.

It is important to recognize that each of us is an individual. Our responses to low or high cholesterol diets differ. Some people can eat all the cholesterol they want without raising their total cholesterol; others develop hypercholesterolemia on a high cholesterol diet.²¹

Recommending a low cholesterol and high polyunsaturated fat diet for an entire nation would unquestionably create controversy. In a recent review, Mann stated that we have no right to recommend such a diet²² but his views are strongly challenged.^{23,26} A recent survey²⁷ shows that while the controversy persists, physicians and investigators involved in treating patients with lipid disorders tend to practice the theory they find most acceptable. □

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Dietary Fat and Cholesterol

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In December, 1976, a National Committee on Diet and Cardiovascular Disease submitted an extensive report (which became known as the Mustard Report¹), accompanied by recommendations, to the Minister of Health and Welfare. The recommendations were amended and adopted in June, 1977. They were then forwarded to provincial health departments for consideration.

The recommendations included guidelines for public education programs to change the food habits of Canadians and thereby reduce the incidence of coronary artery disease (CAD). Recommendations for altering fat consumption were based on the "lipid hypothesis", that saturated fats and cholesterol (from food sources) elevate serum cholesterol and this, in turn, leads to an increased incidence of CAD.² It is generally agreed that dietary fatty acids and cholesterol influence serum cholesterol,^{2,3} but there remains a question in medical circles, as to whether the incidence of CAD can be affected by altering the lipoprotein profiles in a general population.²

Immediately, controversy erupted as to whether the moderate approach advocated in the recommendations was appropriate for treating the general population. Despite clinical evidence correlating diets high in saturated fat with increased incidence of CAD,^{2,4} many scientists argue that a more sophisticated understanding of the pathogenesis of hyperlipoproteinemia is necessary before a public health program is implemented to effect major changes in fat consumption by the general public. They argue that elevated blood cholesterol is only one of the risk factors associated with CAD and that altering the nation's diet en masse is unwarranted. Furthermore, they point out that the moderate recommended restrictions are unlikely to be effective in correcting a lipid abnormality in an individual within the population and hence should be abandoned. They advocate screening procedures to identify individuals who have hyperlipoproteinemia and treatment as indicated.⁴

Other scientists feel that present knowledge is sufficient to justify altering the public's diet. Based on intervention studies in Finland showing that cholesterol-lowering diets reduced the mortality from CAD in males,² they suggest that it is difficult to avoid the conclusion that dietary intervention affects the incidence of CAD.⁴

The issue has been further complicated by evidence that binding of cholesterol to a particular lipoprotein, the high density lipoprotein (HDL), may be beneficial in reducing the risk of CAD in some individuals.³ Cholesterol and other lipids in the blood are transported by lipid and protein complexes. The lipoproteins most relevant to a discussion of hyperlipoproteinemia are HDL and low density lipoproteins (LDL). There is now evidence that the level of serum HDL cholesterol is inversely proportional to the prevalence of CAD; i.e., the lower the level of HDL cholesterol, the higher the risk of developing CAD. The exact function of HDL is unknown, but present evidence suggests that it exerts its

protective effect by transporting cholesterol from the tissues to the liver for catabolism.⁵ HDL may compete with LDL for binding sites, and thus, higher levels of HDL may have the net effect of decreasing the amount of cholesterol available for deposition as atherosclerotic lesions.

Since both these lipoprotein fractions (HDL and LDL) contain cholesterol, research emphasis is divided between finding dietary factors that decrease LDL and discovering factors which have been associated with higher HDL. The question to be answered then is how HDL cholesterol levels can be increased so that they may bring about a lower incidence of CAD in the general population.

Current evidence suggests that HDL is increased in those who exercise vigorously and reduce their body weight to ideal.^{3,5} Dietary intervention restricting food cholesterol seems to have no effect on HDL; moderate alcohol intake appears to increase HDL.^{3,5} Thus, factors known to increase HDL are consistent with already accepted methods of decreasing the incidence of CAD.

The following discussion considers the recommendation pertaining to fat, the type and quantity recommended for the diet of Canadians. It must be noted that cholesterol restriction *per se* is not stated in the recommendation which suggests "a reduction in calories from fat to 35 percent of total calories" and the inclusion of "a source of polyunsaturated fatty acid (linoleic acid) in the diet."² However, the original Mustard Report did state: "Limit the total amount of fat, cholesterol, . . ." and "reduce the total amount of saturated fat in the diet and replace some of this with polyunsaturated fats."⁶

A probable reason for the change is that published recommendations for dietary changes in total populations have far-reaching economic and agricultural implications. Food policies cannot be made in isolation from the political realities of the age. A government health agency proposing such dietary changes must consider the availability, both in terms of supply and cost, of the recommended foods to the total population, as well as the economic viability for food producers. Thus, a government is not likely, in spite of clinical evidence to support the theory, to state unequivocally that polyunsaturated fat must replace the saturated fat, butter, as the staple fat for its population, or that the intake of the high cholesterol egg be reduced dramatically.

Implementation of such recommendations is, therefore, difficult, and recommendations become compromises between what is ideal and what is acceptable in practice. An excellent example is the egg. Egg yolk contains a significant amount of cholesterol; however, the egg is an excellent source of vitamins and minerals as well as protein of high biological value at reasonable cost. Eggs are generally well-tolerated and are acceptable to the majority of people; they can be quickly and easily prepared for eating with a minimum of equipment. Hence, eggs become a highly recommended food, particularly for those who are elderly, ill or on a very restricted budget. Undoubtedly, Canada's egg

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producers could state a number of additional reasons to encourage the consumption of their product!

Major sources of dietary cholesterol are egg yolk, organ meats such as liver, and dairy products. Since both eggs and liver are nutritious foods, their consumption should be only moderately restricted. Dairy foods are readily available in fat-modified forms which reduce total fat and hence cholesterol intake.

Instead of: whole milk (4% butter fat)	Use: 2%, 1% or skim milk
cheddar or processed cheeses	cottage or skim milk cheeses
ice cream	ice milk, yoghurt, or sherbet

Saturated fats are consumed primarily through meats — beef, lamb, pork — with particularly high levels being present in processed meats — bologna, weiners, sausage, and processed meat slices packaged especially for sandwich making. It is advisable that consumption of these meats be decreased and that fish (other than shrimp) and poultry, with their lower total fat content, be used with greater frequency.

In recommending to the public that the percentage of polyunsaturated fatty acids (PUFA) be increased in their diets, it is essential that a sufficient explanation be given as to how to accomplish this. Polyunsaturated fats are generally of vegetable origin, although not all vegetable fats are unsaturated; notable examples of highly saturated vegetable oils are coconut and palm oils. Due to their availability and

low cost, these two oils are frequently utilized as the fat source in commercial food products or in simulated dairy products such as coffee whiteners or margarine.

To say "use margarine instead of butter" is misleading, as is "use a 100% vegetable margarine"; it must be stressed that package labels should be read, and a choice made from margarines which clearly state on the label that contents include a minimum of 27% PUFA. The ideal product is likely to be of corn oil composition, since for Canadians it is the most widely available unsaturated fat, and of soft consistency, i.e., packaged in a plastic tub rather than a paper-wrapped block. As the percentage of PUFA increases, the margarine will be more oil-like. Through hydrogenation, margarine becomes more solid, but this chemical action reduces the degree of unsaturation. Therefore, the key words on the label are: polyunsaturated, non-hydrogenated. Corn, safflower, or sunflower seed oils are in themselves excellent sources of PUFA and are easily incorporated into food preparation.

The most practical approach to these recommendations, and the compromise most agreeable to producer, consumer, physician, and nutritionist, is one that emphasizes control of the total fat intake through (a) moderate use of eggs and liver, (b) use of fat-modified dairy products, and (c) increased consumption of fish and poultry with concomitant decrease in consumption of processed meats. The net overall effect may be a reduction in total calories, and a consequent reversal of the overweight status of 50% of our population, as well as reduced mortality from coronary artery disease. □

References on page 106.

RECOMMENDED MARGARINES* AND OILS†

Brand	Content	% Polyunsaturated and Saturated as Listed on the Label
Margarines:		
Blue Bonnet Soft	100% soya oil 51% liquid soya oil	35% polyunsaturated 20% saturated
Dominion Corn Oil	52% liquid corn oil 28% hydrogenated	35% polyunsaturated 18% saturated
Dominion Soft	48% liquid vegetable oil 32% hydrogenated	35% polyunsaturated 18% saturated
Fleischman (hard)	100% corn oil 51% liquid corn oil	27% polyunsaturated 18% saturated
Fleischman (soft)	100% corn oil 55% liquid corn oil	40% polyunsaturated 18% saturated
Good Lick Soft	80% soybean oil	35% polyunsaturated 18% saturated
Imperial Soft	100% vegetable oil 60% liquid oil	35% polyunsaturated 18% saturated
Oils:		
Mazola Corn oil	100% corn oil	54% polyunsaturated 14% saturated
Safflo Sunflower Oil	100% sunflower seed oil	63% polyunsaturated 12% saturated

*Margarines surveyed, April, 1978.

†Other brands of margarine and oil may be available.

ALWAYS READ THE LABELS CAREFULLY.

(Prepared by: Nutrition Services, Nova Scotia Department of Health, 1978)

Exercise and Cardiovascular Disease

B. R. Wheeler*, M.B., B.S., M.R.C.S., L.R.C.P., D.Obst. R.C.O.G.,

Truro, N.S.

INTRODUCTION

The value of exercise in preventing ischemic heart disease is one of the most controversial issues in medicine. Experiments with animals, especially young animals, have shown changes in the heart related to exercise, but these have been difficult to duplicate in man, and epidemiologic studies suffer from the drawback that they may involve some selection.

EPIDEMIOLOGIC EVIDENCE IN MAN

In an English study of the effects of activity on ischemic heart disease, Morris *et al.*¹ found that conductors of London buses who climbed up and down stairs many times a day had only half as many heart attacks as sedentary drivers. The difference in exercise alone may not account for this finding since drivers have to face the stress of driving in heavy traffic. However, the conductors also experienced obvious stress in looking after difficult passengers and waiting for the inspector to board the bus.¹

Morris and Crawford conducted a further study in which deaths from heart attack were compared in three groups of civil servants — postmen, male telephonists and civil service clerks. Here, stress may have been present for the telephonists but did not seem to be a factor for the clerks who were doing purely routine work; the exercise factor was strongly in favour of the postmen. Results were similar to those in the previous study; the postmen were much less likely to have heart attacks than the other two groups.²

Since mechanization has eliminated much of the physical work involved in many occupations, Morris *et al.* also examined the effects of leisure time activity. They chose senior executives in the civil service and their group consisted of many thousands. Without warning the members of the selected group were given a form to complete on a Monday morning showing a minute by minute account of their activities on the previous Friday and Saturday. Their scores were determined and they were placed in groups accordingly. They were then followed to see what would happen over succeeding years. There was virtually no difference except in the highest activity group which consisted of people who recorded such activities as chopping wood with an axe, building in stone, heavy digging and some sporting activities; the incidence of heart attack among these people was markedly lower than that in the other groups.³

In the United States, Brand and his associates showed that activity gave a marked protective effect among longshoremen and when combined with the absence of other risk factors such as smoking and hypertension the protection was very great.⁴

In Sweden, Wilhelmsen and his co-workers conducted a

prospective study on men born in 1913 and still alive at the age of fifty. They showed that physical inactivity during leisure time but not at work was a weak risk factor. They felt that activity did modify other more important risk factors such as diet and smoking.⁵

EXERCISE AND SERUM LIPIDS IN MAN

It has not been possible to show that exercise affects serum cholesterol levels to any great extent, but there is a marked association between high density lipoproteins (HDL) and exercise. The amount of HDL is greater in people with less risk of coronary disease⁶ and less in people who have heart attacks or later onset diabetes, or those who take various medications such as oral contraceptives or some anti-diabetic agents.⁷ The combination of propranolol and hydrodiuril seems to lower HDL. In contrast, HDL is raised in runners and cross-country skiers, provided the distance they cover is sufficient.^{8,9}

HDL can be divided into several fractions and it appears the HDL II is the factor that exerts the main protective effect and the one that is increased by exercise. HDL is normally higher in women than in men and it is also increased by consumption of alcohol.

ANIMAL EXPERIMENTS

An increase in both the ratio of capillaries to cardiac muscle fibre and the size of the coronary arteries was shown in rats that were trained (exercised).¹⁰ This effect was greater when the experimental period was continued through puberty. In a similar experiment, myocardial contractility was found to be greater in conditioned rats.¹¹ In preparations of cardiac sarcoplasmic reticulum, calcium was transported to a greater extent in trained rats than in sedentary rats.¹² It has also been found that a stronger electrical current is required to produce fibrillation in trained rats than is needed in more sedentary rats.

Another study showed that complete regression of the cardiovascular adaptation occurred when exercise was stopped for ten weeks. Maintenance of cardiac adaptation required five one-hour sessions every week, whereas, the increase in myocardial capillaries was maintained by two thirty minute sessions per week. An hour of exercise only once a week was enough to maintain the increase in collateral circulation.¹³

In a study of beagle dogs, Cohen *et al.* found that the coronary collateral response to acute ischemia was not affected by exercise.¹⁴ However, other workers, who also used dogs,¹⁵ obtained results that were similar to those in trained rats, as outlined above.

EXERCISE IN MAN

Saltin *et al.* showed that following a three-week period of bed rest there was a marked increase in maximal oxygen uptake, and that this was accompanied by an increase in stroke volume and heart mass.¹⁶ After two months of training,

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the uptake levels rose to the pre bed rest levels or above, depending on the degree of previous fitness.

It has been shown that training exerts a central effect on the heart and a peripheral effect on muscles in which there is an increase in the number of capillaries relative to the number of muscle fibres.¹⁷ There is also an increase in the number of mitochondria and an increase in oxidative enzymes. With exercise, the muscle is better able to take up oxygen from blood and this results in arteriovenous oxygen differences. There is also some increase in blood volume.¹⁶

People who exercise regularly tend to reduce various risk factors, including obesity, hypertension and cigarette smoking. However, there is an increased risk of cardiac death during exercise and this may occur in well trained people as well as in the untrained. The role of exercise in cardiac death is difficult to determine. Deaths that occur during jogging, for example, receive attention from the press, whereas, deaths while watching television do not. Furthermore it may well be that those who exercised succeeded in delaying their deaths by their activity.

CONCLUSION

The work on animals suggests a beneficial effect of exercise on coronary circulation and myocardial behaviour. As yet, the same finding has not been achieved in man, mainly because of experimental difficulties and the relatively crude methods that are needed when subjects cannot be sacrificed. It appears, however, that there is benefit and that this is greatest when exercise is started at an early age and continued throughout life. Encouraging life-long exercise presents a major challenge, however, since parents and education authorities pay only lip service to the value of physical education in schools; when spending is restricted, physical education is the first service to be cut from the curriculum.

It is apparent from the recent epidemic of jogging injuries that the public needs education about the exercise it undertakes; government funded programs could be most helpful in providing the needed advice. People must accept the obvious need for exercise to be year-round, despite the problem of our climate. Outdoor exercise is possible even in winter as long as one avoids ice, freezing rain and high wind-chill factors. Only after many years of regular exercise that began in early youth can the full benefits of training be determined. □

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The Role of Excessive Salt Intake in the Genesis of Essential Hypertension

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Halifax, N.S.

A popular aphorism states that all things pleasurable are immoral, illegal or fattening; one might add that whatever pleasures escape the above guidelines are probably bad for one's health. The latest contender to be added to the increasing list of health hazards is table salt; arguments for and against its inclusion have waged hot and long for more than a decade and there is little doubt that we eat much more salt than we require.

Several features of man's metabolism are more attuned to the diet of our ancestors which had a high vegetable, fruit and cereal content. These include the renal handling of sodium and potassium. The largely vegetable diet of primitive man contained large amounts of potassium but little sodium; consequently, the kidney is very efficient in retaining the latter but not the former. This is evident from the experiments of Benedict¹ who studied a human subject fasting with free access to distilled water for a month. After 7 days, urine sodium losses had fallen to about 10 mmol per day and remained at that level or often much less for the duration of the study, whereas, urine potassium losses remained around 30 mmol/day throughout the study.

Daily salt requirements, in the absence of abnormal losses, amount to about 8 mmol, but the daily intake of North Americans ranges from 100 to 250 mmol. Salt is added in great abundance to our foods (Table I)². This appetite for salt is apparently acquired as people who are not used to adding salt to their food dislike it at first.³ This is in contrast, of course, to the salt craving experienced by patients with true salt deficiency, for example, those with Addison's Disease, and that which drives herbivorous animals to seek out salt licks; the latter is probably a deficiency induced response as these animals, once sated, will moderate their salt usage (W. J. Longley, personal communication).

TABLE I
SODIUM CONTENT OF COMMON FOODS

Food	mmol/serving (except where stated)
Jello pudding	7
Dill pickle	19
Bacon	5
Potato chips	3
Dehydrated soup	14
Cottage cheese	7
"Big Mac"	26
Cow's milk	10 mmol/L
Human milk	2 mmol/L

(From Editorial . . . Consumer Report, March:147-149, 1979)

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The evidence for a relationship between chronic over ingestion of salt and essential hypertension is largely epidemiological. Dahl³ demonstrated a positive correlation between salt intake and essential hypertension in a variety of population groups (Fig. 1) but it is obvious that there are differences other than salt intake between these groups.⁴ Other studies have shown similar relationships between salt and hypertension in more homogeneous population groups, however.⁵ Of particular interest is a report⁶ that blood pressure in Massachusetts school children is positively related to the sodium content of drinking water. Dahl³ found a relationship between salt usage and hypertension in Long Islanders but Miall⁷ and Dawber *et al*⁸ were unable to demonstrate such a relationship in Wales and Framingham respectively.

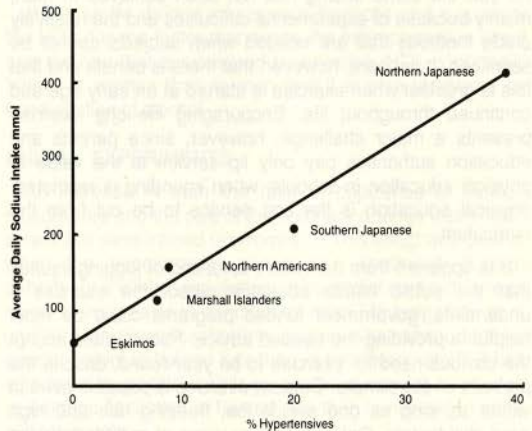


FIGURE 1

Relationship Between Daily Salt Intake and the Prevalence of Hypertension (> 140/90) in Different Populations.
(From Dahl . . . Am. J. Clin. Nutr. 25:231-244, 1972)

The "Salt" theory would be more convincing if essential hypertension were associated with an expanded extracellular fluid volume rather than the result of increased peripheral resistance to blood flow,⁹ although some¹⁰ have argued that the "normal" E.C.F. volume in essential hypertension is maintained at the expense of the increased blood pressure. Pietinen *et al*¹¹ have demonstrated a correlation between both 24 hour and overnight sodium excretion and a history of first degree relatives with hypertension in normotensive individuals; this suggests a difference in sodium intake and/or metabolism in this group. However, his study also showed a correlation between salt output and obesity, a factor known to be related to hypertension in the absence of differences in salt ingestion.¹²

The pond is muddy and I am unable to clarify it. The facts are:

1. Thirty per cent of patients with essential hypertension respond well to severe dietary sodium restriction (less than 10 mmol/day).
2. Most studies agree that moderate dietary sodium restriction does not affect established essential hypertension.
3. Epidemiological studies have shown a clear relationship between salt intake and the prevalence of essential hypertension.

Until the dispute is resolved and because "salt appetite" is apparently a conditioned phenomenon, it would seem prudent to be less heavy handed with the salt shaker in the hope that we will be rewarded with a normotensive old age, but I cannot offer a guarantee of success. □

Acknowledgement

I am grateful to Dr. E. Carl Abbott for reviewing this manuscript.

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NEW MEMBERS

The Physicians listed below have joined The Medical Society of Nova Scotia between May 1, 1979 and July 31, 1979. A most cordial welcome is extended by the Society.

Dr. Duncan S. Armstrong	Yarmouth, N.S.
Dr. Gilles E. Bisson	Halifax, N.S.
Dr. C. Benjamin Boucher	Halifax, N.S.
Dr. Marcus J. Burnstein*	Toronto, Ont.
Dr. Kenneth MacL. Cameron	Tatamagouche, N.S.
Dr. Stewart M. Cameron*	Vancouver, B.C.
Dr. Jacqueline F. Cloney	Amherst, N.S.
Dr. Alban L. Comeau	Meteghan, N.S.
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Dr. Thomas R. Park	New Glasgow, N.S.
Dr. Michael G. Quigley*	Halifax, N.S.
Dr. Felicity C. Simms	Halifax, N.S.
Dr. John H. Sullivan	Bridgewater, N.S.
Dr. Terry A. Rohland	Parrsboro, N.S.

*Recent Dalhousie Graduates

— NOTICE —

Dr. M. A. Smith, Chairman of the Executive Committee, will present at the 1979 Annual Meeting of The Medical Society of Nova Scotia, on behalf of the Executive Committee the following proposed amendment to the Society By-Laws. The purposes of the amendment are to bring general practitioner representation on Council in line with Branch representation on Council, and to ensure that this occurs automatically as Branch representation on Council changes because of Branch growth in numbers.

Section 9.2.1. (n) be amended to read:

"To increase General Practitioner representation on Council representatives as follows: from each Branch having 50 members or less in good standing in the Society, one member, and for each 50 over the first or fraction thereof one additional member provided that no Branch shall have the right to nominate more than three General Practitioner representatives."

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Contraindications

1. In status asthmaticus or in patients with moderate to severe bronchitis.

2. Active or quiescent untreated pulmonary tuberculosis.

3. Untreated fungal, bacterial or viral infections of the respiratory system.

4. In children under the age of 6 years.

Warnings

1. Glucocorticoids may mask some signs of infection and new infections may appear during its use.

2. "THE DEVELOPMENT OF PHARYNGEAL AND LARYNGEAL CANDIDIASIS IS CAUSE FOR CONCERN BECAUSE THE EXTENT OF ITS PENETRATION OF THE RESPIRATORY TRACT IS UNKNOWN. IF CANDIDIASIS DEVELOPS VANCERIL SHOULD BE DISCONTINUED AND APPROPRIATE THERAPY INSTITUTED".

3. In patients previously on high doses of systemic steroids, transfer to Vanceril Inhaler may cause withdrawal symptoms: tiredness, aches and pains, and depression. In severe cases, acute adrenal insufficiency may occur necessitating the temporary resumption of systemic steroids.

4. The safety of Vanceril in pregnancy has not been established. If used, the expected benefits should be weighed against the potential hazards to the fetus, particularly during the first trimester of pregnancy.

Precautions

1. The transfer of a patient from systemic steroid to Vanceril Inhaler has to be very gradual and carefully supervised by the physician. The guidelines under Dosage and Administration should be followed.

2. A decreased resistance to localized infection has been observed during corticosteroid therapy.

3. During long-term therapy, pituitary-adrenal function and haematological status should be periodically assessed.

4. Fluorocarbon propellants may be hazardous if they are deliberately abused. Inhalation of high concentrations of aerosol sprays has brought about cardiovascular toxic effects and even death, especially under conditions of hypoxia. However, evidence attests to the relative safety of aerosols when used properly and with adequate ventilation.

5. It is essential that the patients be instructed that Vanceril Inhaler is a preventative agent which must be taken at regular intervals, and is not to be used during an asthmatic attack.

6. There is an enhanced effect of corticosteroids on patients with hypothyroidism and in those with cirrhosis.

7. Acetylsalicylic acid should be used cautiously in conjunction with corticosteroids in hypoglycaemia.

8. Patients should be advised to inform subsequent physicians of the prior use of corticosteroids.

Adverse Reactions

No major side effects attributable to the use of recommended doses of Vanceril Inhaler have been reported. No significant systemic effects have been observed when the daily dose was below 1 mg (twenty inhalations).

Above this dose, reduction of plasma cortisol, indicating adrenal cortical suppression, may occur. Therapeutic doses may cause the appearance of Candida albicans in the mouth and throat. In some patients the appearance of hoarseness or pharyngeal irritation has been observed, occasionally necessitating withdrawal of treatment. The replacement of systemic steroids with Vanceril Inhaler may unmask symptoms of allergies which were previously suppressed by the systemic drug. Conditions such as allergic rhinitis and eczema may thus become apparent during Vanceril therapy after the withdrawal of systemic corticosteroids and should be treated appropriately.

Symptoms and treatment of overdose

Overdose may cause systemic steroid effects resulting in symptoms of hypercorticism and/or adrenal suppression. Decreasing the dose will abolish some of these side effects, when due to excessive dosage. Adrenal suppression should be treated symptomatically.

Dosage and Administration

Optimum doses vary, but the total daily dose should not exceed 1 mg of beclomethasone dipropionate (20 inhalations), and should not be initiated until the severe attack has been controlled with systemic corticosteroids.

Adults: Two inhalations twice to four times daily as the usual maintenance dose. In severe cases it is advisable to control the symptoms with systemic corticosteroids before starting treatment with beclomethasone dipropionate inhaler.

Children: Insufficient information is available to warrant the safe use in children under age 6. For children over 6 years of age one inhalation (50 µg) up to four times daily.

MAXIMUM DAILY DOSE SHOULD NOT EXCEED 20 INHALATIONS FOR ADULTS AND 10 INHALATIONS FOR CHILDREN UNDER 12 YEARS OF AGE.

Since the effect of Vanceril Inhaler depends on its regular use and on the proper inhalation technique, patients must be instructed to take inhalations at regular intervals. They should also be instructed in the correct method: to exhale completely, lips to be placed lightly around the mouth-piece and activate the aerosol in the next respiratory period. In the presence of excessive mucous secretion, severe attacks of asthma, and/or infection or high atmospheric concentrations of appropriate antigens, the drug may fail to reach the bronchioles. Therefore, if an obvious response is not obtained after 7 days, appropriate therapy including a short course of systemic corticosteroids should be instituted before returning to the use of inhaler, as well as the concomitant use of a broncho-dilator aerosol.

Careful attention must be given to patients previously treated for prolonged periods with systemic corticosteroids when transferring them to beclomethasone. Initially, Vanceril and the systemic steroid must be given concomitantly for 10-14 days, followed by a gradual withdrawal of the systemic steroids. Dose reductions should be the equivalent of 1.0 mg every 10-14 days if close continuous medical supervision is not feasible. It may be possible to withdraw systemic corticosteroids more rapidly if the initial dosage was 7.5 mg daily of prednisone (or equivalent) or less, or if the patient is under close continuous medical supervision. Some patients may not be able to completely discontinue the use of systemic steroids. In such cases a minimum maintenance dose should be continued in addition to Vanceril Inhaler.

Dosage Form

Vanceril Inhaler is a metered-dose aerosol, delivering 50 µg per inhalation. Each canister provides 200 metered sprays.

Full information is published in the Compendium of Pharmaceuticals and Specialties and available on request from Schering Canada Inc., Pointe Claire, Quebec H9R 1B4.

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M. W. Spence,* M.D., Ph.D. and J. T. R. Clarke,** M.D., Ph.D., F.R.C.P.(C),
Halifax, N.S.

INTRODUCTION

Over 25 Nova Scotian men and at least an equal number of women in the province have an hereditary metabolic disease characterized in many by recurrent attacks of excruciatingly painful tingling in the hands and feet — called by some, "the bloody prickers". The bizarre and protean features of the condition commonly result in months of hospitalization and futile laboratory investigation and ineffective treatment with anti-inflammatory drugs or vitamins. In medical books, the condition is called Fabry's disease, Anderson-Fabry disease or angiokeratoma corporis diffusum.

WHAT IS IT?

Fabry's disease is an hereditary, systemic disorder of glycosphingolipid metabolism transmitted as an X-linked recessive condition and characterized by accumulation of glycolipid in the walls of small arteries, kidney, muscle, nerve, and other tissues. Female carriers of the condition may be asymptomatic, but they often exhibit attenuated features of the disease.

HOW COMMON IS IT?

Once considered very rare, Fabry's disease is now recognized to be relatively common among the hereditary metabolic diseases. Indeed, it is the commonest of the various inborn errors of glycosphingolipid metabolism, which include conditions like Tay-Sachs disease, Gaucher's disease, and GM1 gangliosidosis. During the past 15 years, literally hundreds of cases have been reported in North America alone. In Nova Scotia, where the condition was recognized almost 20 years ago, the prevalence of Fabry's disease is relatively high — about 1 in every 15,000 Nova Scotia males may be affected. It is particularly prevalent in Lunenburg County. In families affected with the condition, the signs and symptoms are well known, though their significance has only recently become fully appreciated.

WHAT CAUSES IT?

Fabry's disease is caused by an hereditary deficiency of an enzyme, alpha-galactosidase, involved in the breakdown of two glycosphingolipids which normally occur in only trace amounts in most tissues of the body. The enzyme defect results in accumulation of glycolipid (principally ceramide trihexoside, but also digalactosyl ceramide) in various tissues.

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Accumulation of glycolipid in the small blood vessels in the skin produces the characteristic pathognomonic skin rash called angiokeratoma corporis diffusum. The vascular changes are quite distinctive and different from atherosclerotic vascular disease. The endothelial cells of small arteries in affected individuals are swollen by the accumulation of lipid which stains like carbohydrate. Accumulation of the lipids in the kidney causes progressive swelling and destruction of glomerular and renal tubular epithelial cells. Lipid deposition also occurs in peripheral nerves and nerve roots, but the deposits are very small; accumulation in brain is apparently negligible.

HOW WOULD I RECOGNIZE IT?

The symptoms of Fabry's disease are often very bizarre. Affected individuals are to all appearances normal and usually asymptomatic until late in childhood. The presence of the disease is usually heralded by the occurrence of recurrent attacks of fever and/or exquisitely uncomfortable paraesthesias in the hands and feet. Some affected boys have attacks of migratory arthritis with fever which superficially resemble rheumatic fever or rheumatoid arthritis. Attacks may be precipitated by unusual fatigue or extremes in temperature; more often, however, no obvious antecedent can be identified. They usually last for a few hours, but they may go on for days. Although a number of folk remedies have been employed in efforts to interrupt the attacks, none has been particularly effective.

In addition to the painful crises, patients with this disease may complain of an inability to sweat. This may be associated with some heat intolerance, but it does not produce major disability.

In most instances, the painful crises tend to moderate in early adulthood. Affected men may then be asymptomatic



FIGURE 1A

Typical skin lesions of Fabry's disease on the umbilicus.

again until they develop clinical evidence of progressive renal failure. By their late 30's and early 40's patients with the condition usually demonstrate significant dependent edema and later increasing lassitude and other signs of renal impairment.

Physical findings early in the course of the disease are frustratingly few. By the time they reach teenage, however, many affected boys are showing a few of the pathognomonic skin lesions of the disease. The lesions are tiny, usually less than 1 mm. in diameter, punctuate, raised, dark red to black, discrete lesions distributed symmetrically on the trunk and in the genital regions of the body (Figure 1). We have found that the most fruitful area to search for them is in the umbilicus and on the skin of the scrotum. They are not itchy or otherwise irritating, and there is no associated inflammatory reaction. Similar telangiectatic lesions occur on the conjunctiva and also on the buccal mucosa, especially in the gingivo-labial groove.

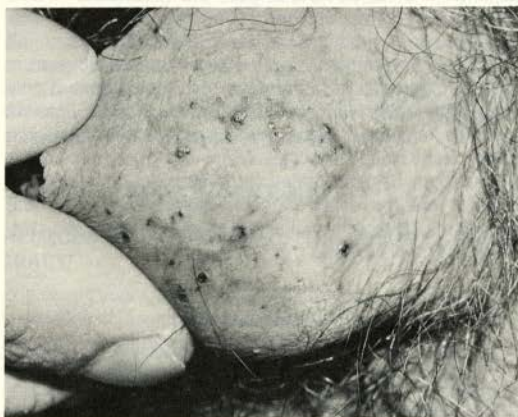


FIGURE 1B

Typical skin lesions of Fabry's disease on the penis.

Most affected men, and many carrier women, have subtle, feathery corneal opacities. These are usually visible only with the aid of a slit-lamp, and they do not interfere with vision.

A list of some of the more common signs and symptoms is shown in Table I.

TABLE I

SIGNS AND SYMPTOMS OF FABRY'S DISEASE

- Recurrent painful paraesthesias in the hands and feet
- Characteristic skin rash: tiny, discrete, punctate, raised, painless, red-black lesions distributed symmetrically on lower trunk and genital area
- Feathery corneal opacities: only visible with slit-lamp
- Fever of unknown origin
- Absence of sweating
- Migratory arthralgia or arthritis
- Swelling of the ankles
- Proteinuria
- Arterial hypertension

HOW DO I MAKE THE DIAGNOSIS?

The clinical diagnosis of Fabry's disease is often difficult, particularly in children. We have seen some young men in their early 20's with no physical manifestations of the disease whatsoever. On the other hand, the presence of the pathognomonic skin lesions virtually clinches the diagnosis. The corneal lesions are also typical and are also seen in many carrier females who do not have any other manifestations of the disease.

Routine measurements of blood lipids are of no value in the diagnosis; serum cholesterol and triglyceride levels are normal. By special analytical techniques, elevated levels of glycolipids can be demonstrated in plasma and also in urinary sediment, but the procedure is very cumbersome and expensive.

By far the easiest way to establish the diagnosis, the method used routinely in the research laboratories of the Atlantic Research Centre for Mental Retardation, is to demonstrate deficiency of alpha-galactosidase in plasma, white blood cells, or tissue from the patient. Males with Fabry's disease have almost total deficiency of the enzyme in all of these. Most carrier females have levels of enzyme activity intermediate between that of men affected with the disease and healthy controls. However, it is sometimes very difficult to establish whether a particular woman is in fact a carrier of Fabry's disease or not.

CAN IT BE TREATED?

So far, there is no practical, specific treatment for Fabry's disease. Efforts have been made at specific enzyme replacement with purified alpha-galactosidase, but the value of this approach is still unconfirmed.

By far the most bothersome aspect of the disease early in its course is the recurrent attacks of pain and paraesthesias in the extremities. In our experience, most attacks can be prevented by continued prophylactic treatment with dilantin, maintaining blood levels of the drug similar to those effective in the treatment of idiopathic epilepsy. Tegretol has also been reported to be of benefit to patients with Fabry's disease.

The management of the renal insufficiency is the same as that due to any other renal disease. Ultimately, most patients become uremic and require chronic hemodialysis and renal transplantation. The indications for dialysis and transplantation are the same as for any cause of renal failure.

WHAT IS THE OUTLOOK FOR PATIENTS WITH THIS DISEASE?

Until recently, most patients with Fabry's disease could expect to suffer recurrent attacks of excruciating pain in the extremities and to die of the condition by a late middle life. Death usually occurred as a result of progressive renal failure or premature cerebrovascular or cardiovascular disease. Today, the disability attributable to the recurrent painful crises can be completely averted by prophylactic treatment with dilantin or tegretol. As a result, men with the condition may be kept completely asymptomatic until their late 30's or early 40's; some are free of symptoms until even later in life. Ultimately, signs of progressive renal insufficiency develop: proteinuria, pedal edema, arterial hypertension, azotemia.

Nevertheless, with continuing improvements in the management of chronic renal insufficiency and the success of

renal transplantation, life expectancy may be extended by a decade or more. Female carriers of the disease generally have a normal life expectancy, though many experience mild episodes of distal paraesthesias and possibly other ill-defined symptoms referable to the condition.

WHAT ABOUT OTHER MEMBERS OF THE FAMILY?

Fabry's disease is transmitted as a X-linked recessive condition (Figure 2). Thus, all the daughters of affected males would be expected to be carriers of the mutant gene. On the other hand, father to son transmission of the disease is genetically impossible. Among the offspring of carrier women, half the boys would be expected to be affected with the condition, and half the girls would be expected to be carriers.

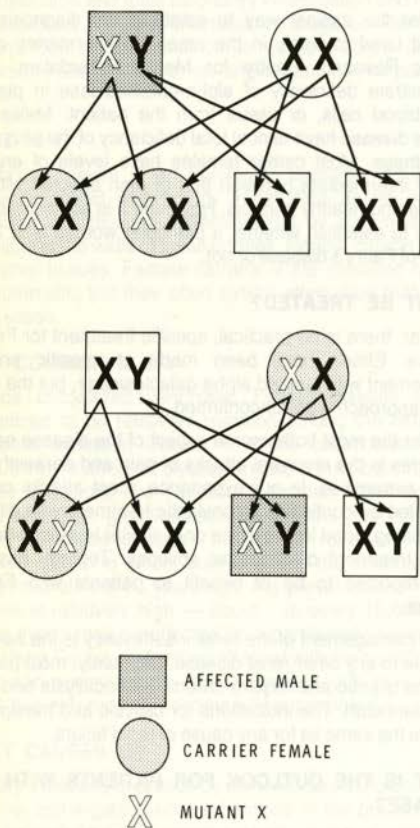


FIGURE 2

Inheritance of X-linked diseases. The upper panel shows the risk to progeny of an affected male; the lower panel shows the risk to progeny of carrier females.

WHEN SHOULD I CONSIDER THE DIAGNOSIS OF FABRY'S DISEASE

The painful paraesthesias in the extremities occurring in youngsters affected with the disease may resemble a peripheral neuropathy. Any boy with a history of recurrent attacks of tingling, numbness and lightning pain in the extremities, who does not have any evidence of nutritional or toxic neuropathy, should be investigated for Fabry's disease.

Similarly, the disease may mimic rheumatic fever or acute juvenile rheumatoid arthritis. It should be included in the differential diagnosis of migratory arthritis with fever in young boys.

Later in life, Fabry's disease should be considered a possibility in any adult male presenting with signs of chronic renal insufficiency. Care should be taken in the interpretation of renal biopsies; the findings have been confused with chronic glomerulonephritis.

The rash, when it is present, is virtually pathognomonic of the disease. There is only one condition, occurring in elderly men, that produces similar angiokeratomas. This condition, called Fordyce's angiokeratoma of the scrotum, is easily differential from Fabry's disease by the absence of any other evidence of systemic disease.

SUMMARY

Fabry's disease, or "the bloody prickles", is a relatively common inherited disorder of glycolipid metabolism in Nova Scotia. Many of the symptoms are bizarre, and their severity is often out of proportion to the physical findings of the condition. The skin rash, angiokeratoma corporis diffusum, is virtually diagnostic of the disease, but it is present in only about half of affected males. Definitive diagnosis is possible by measurements of alpha-galactosidase activity in plasma or peripheral blood leukocytes. Many of the symptoms of the disease can be ameliorated by treatment with dilantin or tegretol, but the drugs probably have no effect on the progressive glycolipid accumulation in blood vessels, kidneys, and other tissues.

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"In order to be a realist you must believe in miracles."

— David Ben-Gurion.

Urinary Incontinence in Women

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FEMALE URINARY INCONTINENCE

Female urinary incontinence is one of the commonest problems encountered by the primary care physician and is a complex, poorly understood subject. Publications are confusing and often contradictory because different terminology is used, various diagnostic tests are recommended, and conflicting treatment schedules are advised. The physician must make a diagnosis and then initiate treatment, which may be either medical or surgical.

Neither gynecologists or urologists have found the final answer concerning the diagnosis or treatment of urinary incontinence in the female. However, in the last ten years, diagnostic advances and new concepts have increased the chances of success in treating this distressing condition. The subject matter of this paper refers to incontinence in women with an intact lower urinary tract and not based on fistulae, congenital anomalies, or damage due to radiation.

PART I: Basic Physiology

NEUROLOGICAL CONTROL OF THE LOWER URINARY TRACT¹

In recent years, some of the most exciting advances in our knowledge of incontinence in the female have been made in the area of neurological control of the lower urinary tract. These advances have resulted in the relatively new neuropharmacological treatment for disorders of the bladder and urethra. The following material on the neurological control and pharmacology of the lower urinary tract is reproduced from the work of Dr. Donald R. Ostergard of the University of California, Irvine Women's Hospital.

(a) Central Nervous System Loops and Circuits

The current concept of central nervous control of the lower urinary tract identifies four interrelated loops and circuits, and the successful functioning of one loop requires the integrity of the remaining loops. Therefore, successful integration of the micturition reflex requires a balanced contribution by all four loops.

LOOP 1 originates in the frontal lobe of the cerebral cortex and terminates in the brain stem. It coordinates volitional control of the micturition reflex and may be interrupted by Parkinson's disease, brain tumors, trauma, cerebral vascular disease, multiple sclerosis, and local lower urinary tract disease. Clinically, any interruption of this circuit removes the micturition reflex from volitional control. Cystometry tests its integrity.

LOOP II originates in the brain stem and terminates in the sacral micturition centre. It provides detrusor muscle contraction of sufficient duration to allow total evacuation of intravesical contents. This loop can be interrupted by any

lesion of the spinal cord and a cystometrogram will test its integrity.

LOOP III constitutes the vesical-sacral-external sphincter loop and provides coordination of detrusor and urethral muscular activity during voiding. It may be interrupted by multiple sclerosis, spinal cord trauma, spinal cord tumors, peripheral neuropathy of diabetes, and local urinary tract disease. Clinical malfunction is indicated by absence of urethral sphincter relaxation during micturition and its integrity can be tested by electromyography.

LOOP IV constitutes the cerebral-sacral loop and provides for volitional control of the striated external urethral sphincter. Electromyography tests will demonstrate the integrity of this loop.

An alternative and complementary explanation of the neurology of micturition involves an understanding of twelve integral reflexes, which function during coordinated micturition. They provide the function of continence, permit storage, and allow the initiation, continuation, and cessation of micturition. They are stimulated by an increase in the detrusor mural tension as a result of bladder filling, and by increasing tension in the perineal and pelvic floor muscles. Depending on circumstances, some of these reflexes favour continence and retention while others assist in the initiation and maintenance of micturition. The seemingly contradictory nature of these reflexes emphasizes the delicate balance between stimulatory and inhibitory influences which exist in the micturition process.

(b) Autonomic Control of the Lower Urinary Tract²

Recent published studies indicate clearly the dual sympathetic and parasympathetic innervation of the bladder and urethra with the existence of adrenergic or sympathetic, and cholinergic or parasympathetic neuroreceptors in all layers of the bladder. The discovery and localization of these autonomic neuroreceptors have shed considerable light on the mechanism whereby pharmacologic agents alter urethrovessical function, by their effects on these autonomic neuroreceptors. The predominance of cholinergic receptors in the body of the bladder is responsible for detrusor contraction on urination — a parasympathetic response. Beta-sympathetic receptors also predominate in the body of the bladder, and stimulation of these receptors results in relaxation — explaining detrusor relaxation which facilitates bladder filling. Alpha-sympathetic receptors predominate in the base of the bladder, the outlet, and the urethra, and are responsible for maintaining continence.

This new concept can be summarized as follows: filling of the bladder is a sympathetic phase, beta-adrenergic stimulation allows bladder filling, and alpha-adrenergic stimulation of the base of the bladder and urethra maintains continence. The parasympathetic system is inhibited during the filling phase. Emptying of the bladder is primarily a parasympathetic function. The act of voiding involves cholinergic induced

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detrusor contractions and the opening of the outlet and urethra, due to sympathetic inhibition caused by the parasympathetic component of the autonomic nervous system. The external sphincter responds to both autonomic (involuntary) and somatic (voluntary) controls.

NEUROPHARMACOLOGY³

Recent understanding of the neurological control of the lower urinary tract has resulted in the neuropharmacological treatment of disorders of the bladder and urethra. There are a multiplicity of drugs which influence the physiological activity of the bladder and urethra. In many instances, drugs given for unrelated disease produce symptoms of urinary incontinence which brings the patient to the primary care physician. An example of this phenomenon would be female incontinence secondary to antihypertensive drugs which are adrenergic. Awareness of drug reactions allows the physician to effectively manage patients with lower urinary tract problems.

I. Parasympathetic Drugs

(a) **Cholinergic Medications** — their primary effect is stimulation of the detrusor muscles of the bladder with little effect on the urethra. Examples of these drugs are Urecholine and Prostigmin.

(b) **Anticholinergic Medications** — their primary effect is relaxation of the detrusor muscle of the bladder with little effect on the urethra. Examples of these drugs are Probanthine, Darbid, and Tofranil.

II. Sympathetic Drugs

(a) **Alpha-Adrenergic Blockers** — produce urethral relaxation. Phenoxy-benzamine (Dibenzylene) is a prototype drug.

(b) **Beta-Adrenergic Blockers** — produce contraction of the urethral sphincter. Propranolol or Inderal has been reported as an effective blocker.

(c) **Alpha-Adrenergic Stimulators** — increase the smooth muscle activity of the urethra, and a general adrenergic stimulator is Ephedrine. Specific alpha-adrenergic stimulation is accomplished by drugs such as Imipramine (Tofranil). It should be noted that Tofranil has both anticholinergic and alpha-adrenergic stimulating effects, and therefore can be very useful in the treatment of incontinence.

(d) **Beta-Adrenergic Stimulators** — cause urethral relaxation but at present, these drugs are not clinically applicable in uro-gynecology. Appropriate manipulation of adrenergic drugs allows treatment of urethral smooth muscle spasm and urethral relaxation occurring in stress incontinence.

Finally, there is a miscellaneous group of drugs with effects on the autonomic nervous system of the lower urinary tract. Phenothiazine derivatives constitute one group which appear to possess alpha-adrenergic blocking activity and may produce urinary stress incontinence; however, these drugs may also cause urinary retention. Similarly, certain antihistamines may produce incontinence. Dilantin and progestational agents cause urinary incontinence. Estrogenic medications in excessive amounts may cause urinary stress incontinence; however, the estrogen-dependent urethral sphincter mechanism usually responds to estrogen therapy

with increased muscular tone. Estrogen therapy, therefore, has definite clinical usefulness in the treatment of hypoes-trogenic patients with mild stress incontinence.

A final comment on urinary tract pharmacology: the multiplicity of drugs which affect the lower urinary tract has an aura of extreme complexity. However, it lends itself to simple generalizations which are useful in the management of the uro-gynecological patient. There are four basic areas which involve bladder or urethral contraction and relaxation, and the physician has the means to stimulate or inhibit them.

(1) **Inhibit Bladder Muscular Contraction** — the pharmacologic inhibition of spontaneous or uninhibited bladder contractions with anticholinergic medications.

(2) **Stimulate Bladder Muscular Contraction** — if the bladder is unable to contract efficiently when challenged with a volume load, or when the patient desires to micturate, then judicious use of cholinergic agents improves its functional activity.

(3) **Excessive Intraurethral Pressure** — a urethra may either generate excessive or low intraluminal pressures, or demonstrates discordant functional activity during vesical contractions. Alpha-adrenergic blockade or beta-stimulation produces a decrease in urethral tone in these patients.

(4) **Low Intraurethral Pressure** — alpha-adrenergic stimulation provides a means for increasing urethral pressure.

In summary, various disorders of the lower urinary tract are effectively treated by cholinergic and adrenergic drugs. Since drugs possess similar pharmacological properties which may produce lower urinary tract symptoms, the physician must seek a thorough and accurate history in evaluating the incontinent patient.

THE MECHANISM OF NORMAL URINARY CONTROL⁴ AND PHYSIOLOGICAL FEATURES OF INCONTINENCE

Hodgkinson has shown that the normal female fills her bladder without any increase in intravesical pressure. Simply stated, urinary continence results from suppression of the innate tendency of the smooth muscle of the bladder to contract with filling, and the ability of the vesical neck to remain closed except during voluntary voiding. The exact mechanism whereby a woman is able to suppress this autonomic function of the bladder has yet to be explained, for anatomic studies have failed to show a circular sphincter at the neck of the bladder comparable to the sphincter ani. However, this area functions unquestionably as a physiological sphincter, and it remains closed to the passage of urine except during voluntary voiding.

It has been shown that the proximal two-thirds of the urethra is an intra-abdominal organ and during stress the increased intra-abdominal pressure is equally transmitted to both the bladder and urethra. Regardless of how elevated the intra-abdominal pressure becomes, urine will not be lost from the urethra as long as the pressure differential between bladder and urethra remains in favour of the urethra. The highest intra-urethral pressures normally occur about mid-point.

Maintenance of the urethral pressure is a factor of the Law of LaPlace, which states that the resistance offered by the wall of an elastic tube to its fluid content varies directly as the

inherent tension in the wall of the tube and the length of the tube, and inversely as the diameter of the tube. The inherent tension of the wall is caused chiefly by the musculature of the urogenital diaphragm and the vesical neck, and is dependent on proper urethral support, freedom from scar tissue, and a normal neuromuscular mechanism. The patient with a lax, poorly supported urethra and an open vesical neck, cannot maintain pressure within the urethra adequate to prevent urine loss during stress.

The sequence of events in normal voiding in the female would appear to be as follows:⁵

Filling of the bladder is accomplished by very little increase in intravesical pressure. The sensation of fullness and the desire to void are the result of stimulation of the stretch receptors in the bladder wall and not a manifestation of increased intravesical pressure. This stimulation sets in action a reflex via the spinal cord (Loop II) which results in a detrusor contraction and emptying of the bladder. The normal adult can voluntarily control this reflex (Loop I) and inhibit detrusor activity until circumstances are convenient: When they are, voiding is initiated at will and the steps are:

- (1) Removal of cortical inhibition.
- (2) Contraction of the abdominal muscles — the importance of which is doubtful.
- (3) Simultaneous relaxation of the levator ani muscles, which results in descent of the urethrovesical junction and backward rotation of the axis of the upper urethra.
- (4) Contraction of the involuntary detrusor muscle of the bladder which is perhaps triggered by the downward movement of the urethrovesical junction, and may be further augmented by a rise in intra-abdominal pressure as a result of bearing down.
- (5) The detrusor muscle, which is continuous with the smooth muscle of the urethra, pulls open the internal urethral meatus, bringing the trigone in line with the posterior wall of the urethra (resulting in loss of the posterior angle and funneling of the urethrovesical junction).
- (6) The urethra then opens from above downward to close in the reverse order when the bladder is empty.

ANATOMICAL FEATURES OF STRESS INCONTINENCE⁴

The key to urinary control is a competent vesical neck backed-up by a secondary control mechanism, consisting of the voluntary muscles surrounding the distal urethra. If the urethra is well supported, if the bulk of its length is intra-abdominal in position, and if the physiological sphincter is able to remain closed during sudden increased intravesical pressure resultant to stress, continence will be maintained.

Beck has described the normal anatomy in a nullipara with a well-supported vesical neck as giving a "lollipop" appearance to the relationship of the urethra and the bladder. The well supported vesical neck is about one centimetre above and behind the inferior edge of the pubic symphysis. The angle of inclination of the urethra is less than 45° at rest, the posterior vesical angle is normally less than 120° at rest. These relationships change during the Valsalva maneuver (increasing the intra-abdominal pressure) and, as stated above, during micturition even in normal continent women

the vesical neck descends, the vesical neck and the upper urethral funnel, and the posterior urethral vesical angle flattens out and is lost.

Any, all, or none of these altered relationships can be demonstrated in patients with stress incontinence. The important anatomic feature is that the bladder is in a pre-voiding position at rest. This means that the urethrovesical junction is located at the most dependent part of the bladder, and therefore, subject to the maximum effect of any increase in intra-abdominal pressure. When the latter occurs, the urethrovesical junction descends even further, funneling of the proximal urethra increases, there is a failure of equal transmission of pressure, the pressure differential shifts in favour of the bladder, and urine is lost.

For years a point of contention has been the significance of the various anatomic changes and the importance of the anterior and posterior urethrovesical angles as measured by chain cystography. No particular angle or derangement invariably means that a woman will be incontinent. Angle loss indicates poor periurethral and bladder neck support — nothing more, and nothing less. Normal angles are frequent findings in patients with recurring incontinence following corrective surgery, an unstable bladder or both.

Physicians generally fail to appreciate that the incidence or severity of stress incontinence cannot be correlated with the degree of pelvic floor relaxation. A cystocele or uterine prolapse does not cause stress incontinence, although incontinence may be present if the urethrovesical junction is inadequately supported. Associated stress incontinence is present in only 20 to 25 percent of women with cystoceles. In fact, it is a common observation that women with the most severe degrees of pelvic floor relaxation are not incontinent. Procidencia tends to promote continence rather than incontinence because with descent of the uterus the neck of the bladder is elevated whereas with loss of periurethral support and resultant incontinence the vesical neck descends to become the most dependent part of the bladder. In many instances, women with the most severe form of procidencia have to replace the uterus within the vagina in order to void.

At the other end of the spectrum, the patient with normal pelvic support may have stress incontinence. In the absence of any demonstrable anatomic defect a large number of patients evaluated by cystometric tests will be found to have incontinence based on bladder muscle dysfunction. (See Unstable Bladder)

PART II: Clinical Assessment of Urinary Incontinence in Women

Once we have ruled out urine loss due to fistula, overt neurological disease urethral diverticula, and radiation damage, the aim during the assessment of women with lower urinary tract symptoms is to separate them into one of the following three groups: (1) infection, (2) bladder instability, and (3) anatomic stress incontinence or incontinence due to sphincter weakness.

HISTORY

The importance of an adequate history is stressed in every report on the diagnosis and treatment of incontinence in women. It is imperative to use a questionnaire, so that

important questions are not overlooked, and our questionnaire is designed to direct attention to the three previously designated major categories causing incontinence.

Patients complaining only of frequency of micturition, dysuria, or pain on voiding with suprapubic discomfort, without confirmation of urine infection, most likely fall into the so-called chronic urethral syndrome group. Patients with an unstable bladder have an urgent desire to micturate, and know they will leak urine if they do not reach the toilet in time. Once they start to void, they are usually unable to stop the stream until the bladder is empty. The sound of running water will often produce a feeling of urgency, and a history of enuresis in childhood is very suggestive of bladder instability.

The patient with sphincter incompetence classically leaks urine only on coughing or sneezing, or other physical stress, which produces a sustained rise in abdominal pressure. Urine is never lost as a result of a detrusor contraction and the imminence of loss is usually not heralded by the sensation of urinary urgency. These patients experience loss of urine in spurts, synchronous with, and ending abruptly after the spike of the increased intra-abdominal pressure — they cough and they lose urine. Patients with an unstable bladder, on the other hand, tend to dribble or leak urine during and after stress, and often the delay is 10 to 20 seconds. The cough increases the intra-abdominal pressure which triggers off a detrusor contraction with resultant loss of urine.

Diagnostic and treatment difficulties are created by the fact that all three groups — infection, bladder instability, and sphincter weakness — can exist together or in any combination of the three. The chronic urethral syndrome can reflexly produce uninhibited bladder contractions, the unstable bladder may be stimulated to contract by stress (running, coughing, standing-up suddenly) as may the bladder with a weakened sphincter mechanism. In an attempt to keep her bladder empty the patient with a sphincter weakness may develop frequency and urgency as a defence mechanism. She knows that when she coughs, urine in the bladder will escape and wet her panties. Thus from a practical point of view there is a considerable overlap among the symptoms associated with the common causes of urinary incontinence.

THE GENERAL HISTORY

A good general history must be taken, and this should include gynecologic, neurologic, and psychiatric questions as well as a general health profile of the patient including medical disorders such as diabetes. We have already mentioned the importance of obtaining a current drug history, since so many drugs effect the function of the lower urinary tract.

EXAMINATION OF THE PATIENT

A careful pelvic examination is crucial to exclude pelvic masses or any other evidence of gross pelvic disease. If the history or examination in any way suggests evidence of disease of the upper or lower urinary tract, a consultation with a competent urologist is mandatory. The initial visit should include the history and general assessment, and on the second visit we attempt to have the patients present to our unit with a full or nearly full bladder. In the lithotomy position, the vaginal walls are observed during resting or straining to determine the degree of relaxation.

If stress incontinence is present, a Marshall test is performed with elevation of the urethrovesical junction. This simple test may indicate whether or not the patient will be relieved by a surgical procedure, although its reliability is questioned by many. A sterile lubricated Q-tip is placed in the urethra up to the urethrovesical junction. Deviation of the urethral axis is measured during resting and straining states. Rotation of the end of the Q-tip towards the pubic symphysis indicates loss of urethral support with downward and backward rotation of the vesico-urethral junction, thus eliminating the posterior urethrovesical angle. Sensation of the perineum is tested by needle prick and the bulbocavernosus and anal reflexes are tested to indicate neurological changes or defects.

The patient voids and any residual urine is measured by sterile catheterization. This specimen is sent for bacteriological study. Although stricture and urethral stenosis are relatively uncommon in the female, occasionally it may be necessary to calibrate the urethra with Hegar dilators.

URODYNAMICS INVESTIGATION

The CO₂ urethroscope is invaluable in the diagnosis of abnormal bladder function. When slowly inserted into the urethra with carbon dioxide flowing at a rate of approximately 100 ml per minute, a urethral pressure profile is created and the urethral resistance can be measured. Direct visualization of the urethral mucosa detects inflammatory conditions, diverticula, or undue rigidity of the urethra. The bladder and urethra function as a unit — as the normal bladder fills the vesical neck gradually closes. Water cystography gives little information about the function of the urethrovesical junction, and visualization of the vesical neck is obscured by the churning effect of the water as the vesical neck closes. With the CO₂ urethroscope the response of the vesical neck to filling as well as its response to straining, coughing, and holding maneuvers can be observed directly.

With anatomic stress incontinence, the vesical neck closes sluggishly and at a low pressure, and with coughing or straining it opens and descends because of lack of support. With the unstable bladder a different pattern is noted as the urethral pressure profile tends to be normal, the bladder filling pressure is higher than normal and the vesical neck closes and opens like the shutter of a camera but finally closes normally. Patients complain of bladder fullness before the vesical neck is observed to completely close, and coughing will usually illicit uninhibited bladder contractions.

CO₂ cystometry is "an explosive bladder detrusor technique", and many investigators feel that it is impossible to discriminate between abnormal detrusor behavior and normal detrusor contractions with this procedure. The sudden stretching of the bladder muscle or the irritating effect of carbonic acid which can form from the combination of CO₂ and water, may produce abnormal cystometry in the normal patient. For these reasons, we rely on simple water cystometry to measure vesical pressures and capacity, and to detect uninhibited detrusor activity. The chain cystogram is not used as a routine, although it is valuable in detecting abnormal descent of the vesical neck in the resting and straining positions and may be useful in assessing women who have had previous incontinence surgery.

TREATMENT

(1) The Unstable Bladder

The unstable bladder is described in the literature under various names such as detrusor micturition, hyperreflexic bladder, neurogenic bladder, dysnergia detrusor syndrome, etc. Patients are unable to voluntarily inhibit detrusor contraction of the bladder, and the intra-vesical pressure may rise to the point where it exceeds the intra-urethral pressure with resultant incontinence. The etiology, although somewhat clouded, is believed to be a malfunction of neurologic pathways which normally inhibit detrusor activity. The pathway interruption producing the unstable bladder is probably Loop 1, between the frontal cortex and the brain. It is sometimes referred to as a "neurogenic bladder", but most workers narrow the definition to those patients with uninhabitable contractions without overt neurological disease. Surveys suggest that as high as 40 percent of all patients attending an incontinence clinic have in fact an unstable bladder.

The incontinence of older patients, such as the nursing home population, is nearly always due to this condition, presumably from arteriosclerotic changes in the cerebral cortex rather than from primary anatomic defect or sphincter weakness. Urinary tract infections may be associated with an unstable bladder. Is the patient with an unstable bladder syndrome more susceptible to infection, or is the infection the cause of the irritability and instability of the bladder? The etiology is uncertain. The unstable bladder syndrome may mimic anatomic stress incontinence, or may coexist with it, and the clinical picture is usually one of the small bladder capacity with urgency, frequency, and nocturia. The majority of patients are best treated with anticholinergic medications such as Tofranil, Probanthine, Diazepam, etc. The performance of stress incontinence operations in the presence of an unstable bladder invites high surgical failure rates.

(2) The Chronic Urethral Syndrome

The chronic urethral syndrome is a diagnosis made whenever a patient complains of any combination of the following: frequency, urgency, dysuria, urge incontinence, pelvic pressure, failure of relief with bladder emptying suprapubic pain, and dyspareunia — in the absence of urinary tract infection. Reflexly, the chronic urethral syndrome may produce uninhibited bladder contractions associated with the unstable bladder.

Physical findings as detected by urethroscopy are myriad and any combination of stenosis, edema, hyperemia, mucosal pallor, mucosal atrophy, vaginal type of squamous epithelium in the trigone, granularity, urethral polyps, exudate, and mucosal cysts may be seen.

The etiology is unknown, but suggested factors are anxiety neurosis, infection of the periurethral glands with stasis of gland secretion, periurethral soft tissue infection, estrogen deprivation, trigonal vaginal metaplasia, allergy, meatal stenosis, etc. There is no consensus in treatment protocols and each proposed regimen seems to have a similar proportion of cures. Urethral dilatation, urethral estrogens, systemic therapy, and periurethral steroid injections have been advocated, all pointing to the fact that a plan of consistently effective therapy has not yet been formulated. We prefer to treat this condition by urethral dilatations under a nerve block to 36 or 38 French, repeated two to three times

at two weekly intervals. It is theorized that the procedure exerts its effect by expression of infected material from the periurethral glands or by alteration of the urine flow pattern through the urethra. It is an on-going type of treatment and it has been stated, "we cure a few, we help some, but we comfort all."

(3) Anatomic Stress Incontinence

Surgical treatment of true anatomic stress incontinence is permanently correctable in over 90 percent of women with the proper surgical approach. Operative failure commonly results from diagnostic failure rather than operative technique. Proper patient selection is the keyword to surgical success, which the experts feel requires urethroscopy and cystometric testing procedures. The importance of selecting the primary operation for stress urinary incontinence has not been generally appreciated, for the first operation is the easiest to perform, and the most likely to succeed. Successive operations are not only more difficult, but they have a decreased chance for success because of scar tissue formation and fixation of the urethra.

In the past, the most common operation for anatomic stress incontinence has been an anterior colporrhaphy, and many techniques have been described. Most authorities feel that elevation and support of the bladder neck from the vaginal approach is not as effective as with the retropubic approach, and indeed, it is felt by some that an anterior vaginal repair has no place in the treatment of stress incontinence. The latter would argue that any attack on the anterior vaginal wall will, of necessity, result in damage to the intrinsic sphincter mechanism of the urethra with its partial or complete denervation and conversion to little more than a fibrous tube. It is then unable to develop a proper intraurethral pressure, and hence, the low cure rate for stress incontinence with this procedure. The latter has been reported as only slightly better than 50 percent.

A cystocele does not cause incontinence or residual urine *per se*, and indeed, is responsible for few symptoms except pressure and discomfort which vary according to the degree of herniation. If surgery is necessary, and in most cases it is not, the urethra and urethrovaginal junction should be avoided and our efforts confined to the area of the posterior cystocele. The overly enthusiastic repair of the cystocele can result in incontinence in women who were previously continent. This probably results from disturbing a good posterior urethrovaginal angle, and straightening it in the course of the surgery. A good working rule in repairing cystoceles that do not have stress incontinence is to leave some cystocele, and avoid the periurethral area.

There is little doubt that the retropubic urethropexy is the most successful operation for stress urinary incontinence. Certainly in all patients with recurrent stress incontinence, very obese patients, patients with a history of asthma or chronic bronchitis, patients involved in athletic or other physically strenuous occupations, the surgical approach should be retropubic; and, as indicated above, there is a strong argument for handling all cases in this manner. The primary consideration in patients with socially incapacitating stress urinary incontinence should be correction of the urinary incontinence, rather than repair of associated incidental anatomic defects. According to Hodgkinson, the practice of performing an anterior repair first, to be followed in

failures by a retropubic procedure, is an approach which should be relegated to antiquity.

Of the multiple retropubic procedures, including the Marshall-Marchetti, it is felt that the urethrovaginal fixation to Cooper's ligament is the procedure of choice (Burch repair). There are several advantages:

(1) Cooper's ligament is a strong, thick band of fascia which offers a firm, secure fixation.

(2) In true anatomic stress incontinence, the intrinsic sphincter mechanism is intact but weakness of support results in abnormal position and motility and thus impairing its efficiency. The objectives of the surgical repair are to preserve the intact sphincter mechanism, to restore its proper position, and to provide it with adequate support. It is imperative to avoid any urethral compression or obstruction, such as may occur with a Marchetti type of repair if the urethra is compressed against the back of the symphysis. With the Burch type of procedure, we place the sutures as far laterally as possible through the entire thickness of the vaginal wall at the level of the urethrovesical junction. When tied to Cooper's ligament, the sutures elevate the bladder neck restoring the urethra to its normal intra-abdominal position and leaving it free in a wide retropubic space, thus eliminating the possibility of compression and obstruction with creation of false continence.

(3) Elevation of the anterior vaginal wall by the Burch procedure provides a satisfactory restoration of the normal anatomy of the bladder neck. In addition, it produces a surprising correction of most of the anterior cystoceles involving the neck of the bladder, and also posterior cystoceles involving the base of the bladder.

If the patients have completed their family, an abdominal hysterectomy along with the Burch procedure may be preferable, particularly if there is an accompanying prolapse, an enlarged uterus, or an acutely anteverted uterus impinging on the bladder. On the other hand, if the uterus is small, involuted, and in the posterior pelvis, a hysterectomy will probably not improve the cure rate of the Burch procedure. The high elevation of the vaginal vault with this procedure results in an increased incidence of enteroceles, and therefore, special attention must be paid to the cul-de-sac. Obliteration of the cul-de-sac is usually accomplished by approximately the uterosacral ligaments in the midline and closing the pararectal gutters with interrupted sutures. The operative procedure is completed by a posterior colpoperineorrhaphy when indicated.

SUMMARY

The casual method of deciding on incontinent surgery, with no investigation other than a history with a few questions such as, "do you leak your urine?" with testing which consists of a pelvic examination and possibly stress and vesical neck elevation tests, is archaic. Authorities feel that urethroscopy and cystometric testing are essential to clarify the cause of urinary incontinence. We have learned that bladder instability, infection, or anatomic loss of support may coexist in the same patient. Surgery may not be required, and many patients will be markedly improved on medical management. Incontinence due to bladder instability responds poorly to surgery whereas relief from anatomic stress incontinence requires surgery and has a surgical cure rate of over 90 percent. Most failures are due to faulty diagnosis; the key to successful treatment is proper patient selection. □

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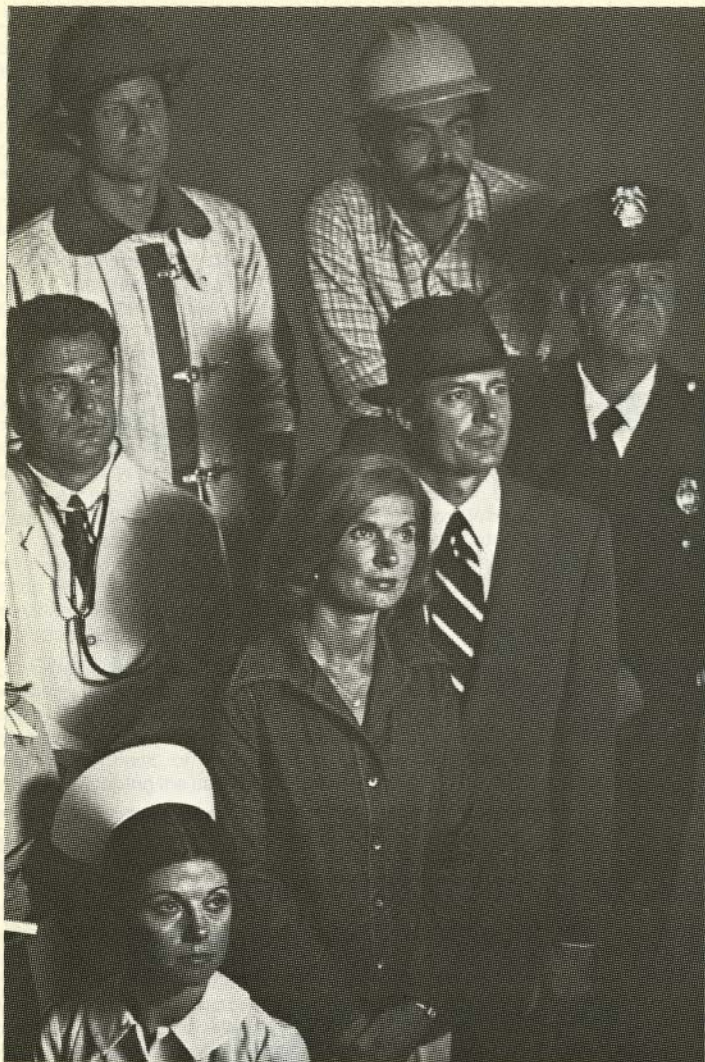
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The Case of the Poisoned Umbrella

Wanted: Mr. Sherlock Holmes, 1979

B. J. S. Grogono*, M.B.,
Halifax, N.S.

A large gathering of Sir Arthur Conan Doyle's devotees recently gathered at the Charing Cross Hotel in London, to celebrate the 75th anniversary of the death of the famous detective, Mr. Sherlock Holmes. In a spate of nostalgia concerning things English and Victorian, there has been a tremendous surge of interest in every conceivable angle of the celebrated detective, whose office and comfortable home was in Baker Street, just a short bus ride away. People travelled from all over the world just for the dinner, dedicated to the art of detection.

Despite many scientific and social improvements, crime has not been eliminated. In fact, London — the peaceful and Cockney idyll of my dreams — has been invaded by visitors from around the globe, and among them are crooks. Ambassadors and agents abound, making it a milieu in which both Holmes and Watson would thrive.

Terrorist activities are countered by an antiterrorist squad of Scotland Yard, headed by Commander James Nevill but, despite all precautions, experts have not solved the case of Georgi Markov, who died mysteriously on September 8 of last year. He was a Bulgarian scientist who defected to Britain some seven years ago, and who had broadcast regularly on the overseas service of the British Broadcasting Corporation.

Were Sherlock Holmes and his colleague alive today, you can imagine them solving the conundrum. Sherlock sits back in his comfortable chair, ruminating over the precise details, puffing on his curved pipe, and giving a perfunctory remark to his assistant. (Only a complex computer belies that this is 1979).

"You say that some fellow brushed against Markov while he was at a bus stop?"

"Yes, that's right. He was standing at the Waterloo bus stop and noticed a sharp jab in his thigh. Then he looked up and saw a man drop his umbrella. The man apologized and hailed a taxi. He must have been a foreigner, as he had difficulty in making the driver understand him". Dr. Watson explained the details carefully as usual.

"Did they ever see the umbrella man again or examine the umbrella?", enquired Holmes.

"No, but a Bulgarian friend, who is a linguistic typist at the BBC, met Georgi on the same evening, September 7, and was asked by him to look at his leg as 'I think someone bumped it with an umbrella'".

"Mrs. Markov noticed blood on the back of her husband's jeans that night, and a small mark on the back of his thigh, such as a ballpoint pen might have made". Holmes looked puzzled. "You're not going to tell me Watson that in these

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days of scientific knowledge, antibiotics and intensive care units, a small puncture wound produced by some pointed object piercing his clothing, was sufficient to kill him?"

"Yes precisely! This fellow was dead within 48 hours!"

"What was his illness like?", Sherlock enquired.

"He was admitted to St. James Hospital, Ballam, next day and his leg was examined by the medical officer, Dr. Bernard Riley, at 11:45 pm. The victim had a high fever, and looked toxic. Examination of the right thigh revealed a circular area of inflammation and a central puncture wound, which was only 2 mm in diameter".

"By 5 am the next morning, Mr. Markov became violent, pulled out his intravenous drip and became confused. His white count rose to 33,000 and he died at 9:15 a.m."

"What did the post mortem show?"

"The pathologist reported that death was due to acute toxæmia of unspecified type. Examination of the thigh showed a dusky area, and a puncture mark the size of a pin head. Beneath this was a metallic pellet 1.52 mm in diameter".

"Was a chemical examination of this material carried out?"

"Yes, Dr. David Gaull, pathologist at the Chemical Defence Establishment in Devon, analysed the material and found it to be composed of platinum and iridium, two rare metals". Dr. Watson had carefully studied the reports in *The Scotsman* of January 3.

"Presumably then, this metal sphere was blown into the victim's thigh from the umbrella and the minute fragment contained a very potent poison", said Holmes.

"There are very few poisons of such a lethal effect — cyanide, the toxin of botulism, cobra venom", suggested Watson.

"No, NO! my dear Watson, they don't cause death in that way. It must be a vegetable poison. Something readily available, but little used — something not easily detectable such as ricin or abin!"

"That's extraordinary", the doctor's face lit up with admiration, "that's exactly what the pathologist suggested. He said that he had given some ricin to a pig in the equivalent dose and the pig died in the same dastardly way. In fact, the judge's verdict was that there was no doubt ricin was the substance introduced into Mr. Markov's body".

"Yes", explained Professor Holmes, "it is an extraordinary poison. You cannot absorb it by swallowing castor oil, but you can extract the castor oil seed and it is lethal. Furthermore, it is broken down in the body and cannot be easily detected".

He took a long puff at his pipe.

"Now it is getting late! What do Scotland Yard want us to do about it?"

"They've contacted almost every country, trying to detect the assassin, but so far they have no clues. They suspect he has escaped across the Atlantic and need your help".

"Hurry up Watson, pack your bag. Get the two air tickets and a dictionary, we're going to Nova Scotia".

"Why Nova Scotia? What dictionary?". Once again, Watson was outwitted.

"Gaelic, Watson Gaelic! They speak that language in Cape Breton and it's the only language a London taxi driver doesn't understand". □


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Future Developments in C.M.E. at Dalhousie

R. Wayne Putnam,* M.D., and Lynn Curry,** Ph.D.,
Halifax, N.S.

Until recent times, continuing medical education was a completely voluntary activity participated in by the majority of physicians for the personal satisfaction derived from knowing that they were maintaining competence to practise. The Division of Continuing Medical Education at Dalhousie has a long history of involvement with practising physicians of the Maritimes, co-operatively responding to requests for further educational experiences. Our aim has always been to assist physicians in the assurance of quality health care in the Maritimes by means of education. In the past, we have attempted to meet this objective through community hospitals programmes, university-based short courses, refresher courses, and clinical traineeships.

Currently however, recent trends in the United States threatened to change the voluntary basis of our participation. Our American colleagues in approximately twenty states have been caught up in the tide of public demand for accountability in other sectors, and carried into a situation in which they must document C.M.E. credit hours for relicensure. We at Dalhousie, as well as most of our colleagues at other universities in Canada, are skeptical of the value of such mandatory C.M.E. and feel that we must carefully analyze and evaluate all effective methods of continuing medical education before endorsing anything of this nature. Up to this point, only Manitoba has moved to give its licensing authority the power to request certification of C.M.E. hours before relicensure.

The present thrust of the Division is in the direction of gaining the capacity to do the research and development

necessary for creating and testing new and better methods of C.M.E. for Maritime physicians. We recognize that one of the most effective forms of C.M.E. has always been reading of journals. Therefore, improving the access of physicians to the most recent literature through the Kellogg Library system will continue to be one of our aims.

The first step in this new direction was taken in the Fall of 1978 when the Division filled two of its three vacancies with new staff members. Dr. David Gass, who had been in family practice in Nashwaaksis, New Brunswick for five years, joined the Division to take primary responsibility for the community hospital programme and to participate in the development of new research projects. Dr. Lynn Curry, who obtained her Ph.D. in educational psychology at Stanford in California, brings to the Division the needed expertise in research methodology and design. She has played a key role in planning a number of projects that are now in the development stages. In the spring of 1979, Dr. Mo lype, a faculty member in the Department of Surgery in Saint John, accepted a cross-appointment to the Division and will become increasingly involved in course planning and research projects.

Over the next year, we will periodically be writing in these pages about each of our new programmes in some further depth. We will also, from time to time, be requesting volunteers to help us out by participating as subjects in a specific project, and we hope that you will look favourably on our request. We are in the process of developing closer relationships with C.M.E. committees in all three Maritime Provinces and will attempt to use these ties to ensure that our efforts continue to be practical and applicable to "real life practice" in the Maritimes. □

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Early Hospitals

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The word hospital was derived from the Latin "hospitium", a place where guests were received. Related forms are hotel, hostel, spital.

Medical treatment has always been associated with religion. It is, however, a mistake to believe that early hospitals were Christian institutions. I was somewhat surprised to read that the Indian Emperor, Asoka, founded a hospital at Aurat 260 B.C. In the Pre-Christian era Ireland apparently had an organization of hospitals. The Brehon Laws made provision for a hospital in every military camp and women were trained to look after the sick and wounded. These laws required that hospitals shall be free of debt. They also demanded that whosoever inflicted bodily injury on another must pay for his maintenance either in a hospital or a private home.

In olden days it was common practice for the people to make yearly pilgrimages to religious centres (temples), with the hope that their god or gods would relieve them of their ills. It is known that as early as 4000 B.C. Egyptians visited the temple of Saturn. Here with improved environment, better diet, patient association and hope (psychotherapy), many were helped.

Early priests were also physicians or medicine men ministering to mind and body. Through vast experience they did acquire a considerable knowledge of disease. There is archeological evidences to show that in Egypt extensive dentistry, also trephining was practiced.

The custom of having hospitals associated with the temple passed from Egypt to Greece. Temples were erected to Saturn,** Aesculapius** and Hygieia,** and the Greeks with their love of beauty soon erected magnificent health resorts.

A striking example of this medical development was at Epidaurus in northern Greece. A description of the temple hospital indicates that the Greeks used practically every form of physical therapy. There were shady walks for patients during the day and lofty airy sleeping quarters with a southern exposure and an open colonnade. Tunnels connected one building with the other. Entertainment was provided. In the Greek theatre were 10,000 seats where the great classic plays were shown. There was also a stadium seating 12,000 for athletic games. In the hippodrome other types of physical activity were to be seen, including those in which animals took part.

Under the Greeks the healing art was much improved. Patients were frequently visited by cultured priests and in the course of time some became exclusively concerned with medical ministrations. Now we learn that herbs, drugs, poultices, fomentations and baths were combined with physiotherapy and some surgery. Classes were organized

for the teaching of students — many were recruited from slaves.

As early as the 6th century B.C. the Greeks had instituted a form of Health Insurance — a tax was collected to support a communal physician, who operated from a medical centre, which with the growth of cities, became the community hospital and dispensary.

In Greece there were several types of physicians: Itinerant, city, court, military and many others.

Roman Medicine

The first physicians in Rome were slaves. Later Julius Caesar granted freedom to all Greek physicians practising in Roman territory. However the most important contributions by the Romans were in the field of public health, and government. They did maintain health resorts for treatment and convalescence of sick or wounded soldiers in tents behind the lines and at home. In Rome large land owners established a form of hospital on their estates for the care of their sick slaves and it is strange to find that members of the landlord's families would frequently use these resorts.

By the fourth century, the Christian churches had built hospitals for lepers, cripples, the blind and sick poor. Religious communities emphasized care of the sick and Sisterhoods and Brotherhoods devoted much of their time to nursing, with hospitals constructed adjacent to monasteries. It was during these centuries that various hostels developed through the mountain passes of the Pyrenees and the Alps.

Several of the Roman emperors and Roman matrons became Christians — Julius, Constantine, also Helena, the mother of emperor Constantine, Marcella, Paula, Fabiola. These dedicated people gave large sums of money for the building and maintenance of such asylums.

Galen (130-200 A.D.) was the greatest physician of his day, born a Greek, he practised most of his life in Rome. Early in his career he attended gladiators, later Roman emperors which included Marcus Aurelius.

The heritage of Greek medicine was perpetuated and brought back to Europe by the Arabs, who established hospital and medical schools in several of their large cities — Bagdad, Damascus, Alexandria, etc., about the 12th century. They became especially eminent for diseases of the eye. They carried on a form of hospital social service by providing needful care for discharged patients who were not quite able to work, and had systems of free medical attention for the poor of the cities.

Responsibility for the administration of hospitals, which were heavily endowed by Emperor Charlemagne (768-814) in various parts of his domain, became a matter of considerable importance to the church and in time was entrusted to the monastic orders. The care of the sick then became the exclusive prerogative of the monks and nuns, and lay physicians almost disappeared.

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**Saturn — God of sowing and seed corn.

**Hygieia — Greek goddess of health.

**Aesculapius — Greek god of medicine (son of Appolo and Coronis)

The Crusades led to the establishment of military monastic fraternities, the most important of them was The Knights of St. John of Jerusalem.* Many of the crusading knights who became sick or injured in battle or in journeying to or from the Holy Land were cared for in these various establishments. The most famous English crusader, Richard Coeur de Lion, was in all probability attended in some of these institutions as he travelled to and from Palestine, for he was seriously wounded during one of these excursions. He eventually recovered but was killed by a bolt from a cross-bow while besieging a castle in France, at the age of 42.

Hospitals in the middle ages were very beautiful places. This was the time of the Gothic architecture and not only the churches, but town halls, guild halls and other public buildings were very beautiful.

Viollet le Duc in his dictionary of architecture describes a hospital built by Marguerite of Bourgogne, sister of King Louis IX (1214-1270), which shows how well these hospitals were built.

"The hospital ward was fifty-five feet wide and two hundred and seventy feet long, had a high arch ceiling, large windows in the walls, tiled floors, a gallery for observation purposes, probably used also for convalescent patients. The patients were separated by moveable partitions and more space was given to each patient than we can now afford. The gallery shielded the patients from the dazzling light and drafts from the windows. The vaulted ceilings were very beautiful; the wood work was richly carved, and the great window over the altar at the end of the ward was filled with stained glass".

In the thirteenth century Pope Innocent III (1198-1216) summoned to Rome the best hospital authority of the day, Guy of Montpellier, who was instructed to build a modern hospital in Rome to be named Santo Spirito. This continued to be one of the sights in that city, until it was destroyed by fire about the year 1955.

Bishops were required to visit Rome every 3-5 years and each was encouraged to establish a hospital in his diocese. A chain of hospitals followed, best illustrated by those in Germany. It was in these institutions that the great surgery of the middle ages became possible.

In the 16th century the hospitals of England, which had fallen into a state of decadence, were closed by Henry VIII. During his reign, however, St. Bartholomew's and St. Thomas' hospitals received new charters.

Between the middle ages and the eighteenth century hospitals fell into a stage of ill repute due to overcrowding, inadequate staff, poor ventilation and infections. Towards the end of the 18th century, as the result of John Howard's exposure of offensive conditions prevailing in hospitals and prisons, a movement was initiated to reform conditions in these institutions. Nursing standards improved through the work of Pastor Gliedner and his wife in Germany and Florence Nightingale in England.

The discovery of bacteriology by Louis Pasteur, in the nineteenth century, led to a revolution in hospital care. A few years later Lord Lister of Glasgow, applied this theory in antiseptic surgical technique. Hospitals then became very popular places for the treatment of the sick.

*A famous hospital founded in 1882, for the treatment of diseases of the eye, is still active in Jerusalem, now supported by the Saint John Ambulance's Association.

Miss Nutting and Miss Dack in the magazine "Hospital of Nursing," note that the first hospital in the new world was established by Cortez in the city of Mexico in 1524, a very beautiful building yet standing, and the superintendent of which is still appointed by descendants of Cortez.

In 1639 — The Hotel Dieu was established In Quebec and the Montreal Hotel Dieu five years later. The first hospital in what is now the United States, was built on Manhattan Island in 1663.

An excellent record of hospitals in Halifax, dating back to the founding of that city, in 1749, has been written by my classmate Dr. H. L. Scammell, Here he describes the several institutions that preceded the construction of the Victoria General Hospital in 1887. All, who are interested in the history of early medicine in this Province, will find it instructive and interesting reading. □

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DIETARY FAT AND CHOLESTEROL

Continued from page 85.

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Dr. Harris Miller — The New Deputy

In the Department of Health for nearly a year.

It's a long way in space and time from the Second World War killing ground of northwest Europe to the key position in Nova Scotia's most heavily budgeted government department. Still, a postwar medical degree for a young artillery officer and a quiet determination to get things done now see Dr. Harris Miller well beyond the first six months of duty as Deputy Minister of the Provincial Department of Health.

For several years — and certainly since the inception of MSI — it was a loosely guarded secret in government circles that perhaps more down-to-earth medical experience might be helpful in executing and administering the policies which now chart the direction for an almost totally insured government service.

In November, 1978, Dr. Miller's appointment to the senior civil service post was confirmed.

Even to a layman, Dr. Miller's credentials appear awesome, mixing as they do the practical and administrative ends of the medical spectrum. He did, after all, practise family medicine in his native Halifax for 15 years as well as holding the post of chief of the Department of General Practice at the Halifax Infirmary for about five years . . . and, of course, during the same general period he also served on the faculties of Dalhousie and Mount St. Vincent University, posts which he maintained until his appointment announcement in the fall of last year.

Incidentally, his move to the latest position was made public while he was also serving as STMO (Halifax) and Regional Medical Officer, Atlantic, for the Department of Veterans Affairs.

Combine all that with appointments and honours in at least 11 other medically related fields, and retrospect indicates the government's choice of Dr. Miller must have been obvious.

But experience and prestige also carry with them heavy responsibilities. A top level administrative post dealing with just about every aspect of the province's health delivery system, particularly during critical financial times, is no exception.

There's the question of ageing facilities, for instance: a question which asks, in nautical parlance, is it better to make or to mend?

Dr. Miller has few doubts on that score.

"Nova Scotia has more than enough facilities right now. Some are in good shape, and some aren't. The problem we have to face is whether or not to spend the available money on renovations or to put it into new facilities and tear the old ones down.

"The fact is, in some instances it will definitely be cheaper to upgrade with new hospital facilities than to attempt renovations on existing buildings, and that seems to me to be the route we have to go."

But, Dr. Miller points out, geographical decisions also have to be made.

He feels that in general terms that eastern half of the province probably has about twice as many acute care beds



as it really needs, while the western half is perhaps under-bedded. And, he adds, some areas are in dire need of long-term disability care facilities.

The answer?

In terms of overall hospital care, Dr. Miller feels the trend should be to downgrading smaller facilities — possibly to ambulatory care status — while concentrating on regional hospital improvements with the money saved.

He says this should improve specific and general patient care.

"Right now, a patient in a small hospital of, say twenty beds or so, which does major surgery and obstetrics is a little divorced from the sophisticated facilities of a major regional hospital which may be only 15 or 20 miles away. That's not to say that the standards of nursing care or of the care afforded by the patient's doctor are any less than they would be in the regional hospital, but the scope is limited."

Dr. Miller says he's convinced that, in terms of both patient care and a better return on the dollar, more emphasis must be placed on capital investments at the regional level.

"But," as he points out, "this will be tied to the availability of dollars. Obviously we can't build everywhere at the same time. Priorities must be set."

In a sense, outside forces have already set one of those priorities. Major institutional changes based on the province's

assumption of responsibilities for Halifax's Camp Hill Hospital are looming for the capital city. Federal money, unalterably designated for veterans' care, combined with capital grants for other health care services is making the Halifax urban and regional area number one on the priorities list.

But there are other areas, too, which demand attention.

Dr. Miller says his interest is also focused on Antigonish and industrial Cape Breton, although the assessment period for those two areas will take some time.

He does note, however, that bed, utilization and length of stay ratios in the Sydney area are considerably higher than on mainland Nova Scotia. This naturally leads to greater per capita costs.

Rather than encourage an increase in those per capita costs he feels a major new regional hospital in Sydney could substantially diminish the rate of cost escalation while providing the area with new facilities which could be justified in budget decision-making.

One of those facilities should have burn treatment capabilities somewhat below a full burn unit which would be unacceptably expensive and statistically not needed.

He says that at present, with only about 7-8 patients a year from industrial Cape Breton are medically "eligible" for the sophisticated burn unit treatment available in Halifax, a four to five bed unit costing about a quarter-of-a-million dollars to operate each year would be hard to justify in an existing Sydney facility.

Dr. Miller feels that a new burn facility can be built in a new regional hospital which could well meet the criteria of both economics and regional need.

Hospitals, of course, aren't just money and equipment. They're staffed by people. One concern expressed in recent months by the Society is that the province is losing some of those key people — the doctors — because of a variety of discouraging economic factors.

Dr. Miller, however, has some reservations about the situation.

"A lot of considerations come into play on that question," he says. "New lifestyles, new opportunities and even competition are probably part of the picture. The last figure I heard was 12 doctors leaving the province. That's only one percent of our total doctor population; a population which has increased by at least 50 percent in recent years."

If competition is a factor, does that mean we have too many doctors?

"Yes, I think we're generally over-producing right now. That may not be a very popular opinion but it's the way I see it. Perhaps the way to correct the problem — if it is a problem — is to start increasing standards and to weed out those with the lower averages . . . but that's just a personal opinion, of course."

At the same time, though, he'd like to see greater encouragement of specialist services outside the Halifax metropolitan area. "Most services should be based on two doctors in each required specialty working from a regional hospital base. This would provide for mutual support and, of course, would allow one to cover off the other."

Hospitals are also more than the medical staff they provide. Lay boards are part of the picture, too, and Dr. Miller

feels that on the whole they're doing a good job — "Particularly when you consider that so many of them are volunteers and are contributing their time and energy because they're sincerely interested in their communities."

Still, he admits that problems can arise from time to time. Inter-community competition for improved facilities plays its part in creating the occasional period of unrest, as do the often persuasive arguments of a zealous administrator who can set his particular board on a near-collision course with the rules of the game.

It is essential that we measure needs against wants in the health field if we are to contain costs. On the whole, though, I think we're pretty lucky and the job is being well done."

Outside of the Halifax area what the future may hold for capital expenditures in health care is still fairly vague, but one thing is certain, Dr. Miller is a firm advocate of local consultation before major changes occur in any community.

"Perhaps this is not so important in the smaller communities because so many of the local government representatives are on the local hospital board as well and they can carry the message. But certainly, in cities like Halifax and Sydney, I would never think of initiating major changes without first making sure that the mayor and council are fully briefed and have had a chance to make their opinions known."

Most physicians would agree that Dr. Mill has a tough row to hoe. The same physicians would probably agree that he's the man for the job. After all, they'll argue, it's the kind of work that demands the efficient impeturbability of a man whose hobbies include philately, fishing, fly tying, woodworking and gardening, as well as the precision of a marksman — which, of course, is another of Dr. Miller's leisure time activities. □

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GOOD NUTRITION: A natural necessity.

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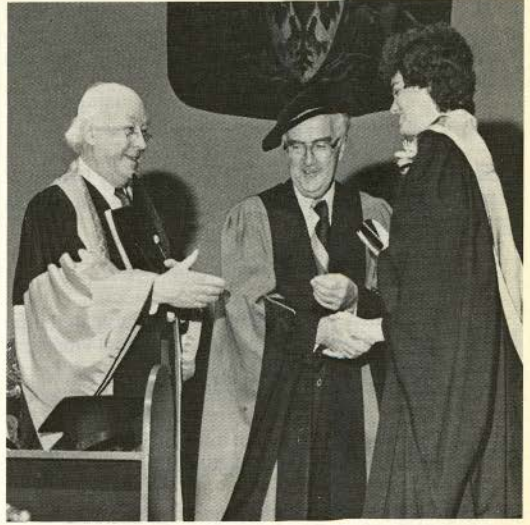
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Personal Interest Notes

1979 Dalhousie University Medical School Convocation

The 1979 Convocation of Dalhousie University Medical School was held in the Rebecca Cohn Auditorium on May 25. The graduating class numbered 86 of which 28 were women. Ten graduated with distinction.

Dr. Anne Marie Gillis of Sydney River received the Dr. C. B. Stewart Gold Medal. She was also awarded the Dr. Clara Olding Prize and tied for the prize in Medicine and the Dr. J. W. Merritt Prize in Surgery.



Dr. Anne Marie Gillis shown receiving her awards.



Dr. Edmund Henry Botterell



Dr. Marie Jean Whittier



Dr. Chester B. Stewart



Dr. William Wrixon being presented with his prize by Dr. Geoffrey Bland, president of the graduating class.

Three distinguished Canadians were honoured at this most recent Convocation: **Dr. Edmund Henry "Harry" Botterell**, distinguished neurosurgeon and teacher; **Dr. Chester Bryant Stewart**, former Dean of Medicine and Vice-President Health Sciences; and **Dr. Marie Jean Whittier**, formerly of Upper Rawdon, Hants County — a distinguished medical resource to her mission in India over a 30 year period.

Dr. William Wrixon was nominated "Professor of the Year". This award is the decision of the 4th year class and is based on their judgement of the teacher who was most effective during their time in medical school.

Dr. Jean Whittier, who was honored by Dalhousie last month, has returned to the house in which she was born to spend the summer months. Her autobiography, "My Life Tapestry", will be published this summer.

Dr. Helen Holden, Director of the Respiratory Service of Kentville Hospital, received an honorary degree from Acadia University in May.

Dr. Joyce Curtis was recently elected President of the Nova Scotia Branch of the Federation of Medical Women of Canada. Dr. Anne Hammerling was nominated for an Honorary Membership. Dr. Hammerling, past President of the Federation, was Chairman of the Scholarship Loan Committee and was President of the local Branch. She has been a faithful member for over thirty years.

Dr. Peter Gordon, Head of the Department of Preventive Medicine, Dalhousie University, has been named Chairman of the Duke of Edinburgh awards in Canada. Over 1000 people, ranging in age from 14 to 27, participate in this project dedicated to improving personal achievements and good health.

Dr. J. D. Carson, former preceptor in Dalhousie University and now practising in Bathurst, New Brunswick, has been named "family physician of the year" at the College of Family Physicians meeting in Ottawa.

Dr. J. Harris Miller, Nova Scotia Deputy Health Minister, was awarded a Citation by the Red Cross for outstanding work as chairman of the National Blood Donor Recruitment.

Dr. Arthur Parsons was recently elected President of Canadian Association of Medical Clinics and Chairman of the Ethics Committee. He presented a paper to the International Congress on Group Medical in West Berlin in June.

Dr. Julia Terzis participated in a 16 hour operation to repair a sciatic nerve in Montreal. She will be remembered as a member of the Plastic Surgical Department of the Victoria General Hospital.

Dr. Wayne Putnam, Director of Continuing Medical Education, Dalhousie University, recently attended a conference on the Evaluation of the Efficacy of Medical Action, at the University of Bordeaux, in France. He presented a paper entitled: "Patient care appraisal: Computer technology potential".

Dr. Basil K. Coady, 66, Halifax, died on June 15, 1979 in the Victoria General Hospital. Born in Cobalt, Ontario, he attended St. Mary's University and then graduated in medicine from Dalhousie University in 1938. He undertook three years of postgraduate training in General Surgery at the Cleveland Clinic.

Following service with the Royal Canadian Navy, he was on the staff of the Victoria General Hospital and the Halifax Infirmary. In 1958, he was appointed Chief of Surgery at the latter hospital and he served in that capacity until his retirement in 1978.

Sincere sympathy is extended to his wife and two sons.

Dr. Bogdan Erjavec, (57) of Weymouth, N.S. died July 17, 1979 following an automobile accident. Born in Yugoslavia, he graduated from the Sorbonne in Paris in 1950 and came to Canada in 1957. After practising in several locations in Eastern Canada he settled in Weymouth where he practised until his death. He is survived by his wife Barbara, one daughter and three sons to whom we extend our sincere sympathy.

Dr. John C. Crosbie, (47) of Kentville, N.S. died July 29, 1979. Born in Newfoundland, he received his medical degree from Dalhousie University in 1957. He practised in Grand Falls, Nfld., for ten years and in Kentville for six years. Our sympathy is offered to his wife and family.

Dr. Charles G. Harries (72) of New Glasgow, N.S. died July 30, 1979. He was born in England and educated at Sydney Academy and Dalhousie University, graduating in medicine in 1936. He was a contract doctor at the Malagash Salt Mine. Following this, he established a general practice in New Glasgow. He retired in 1976 because of ill health. The Bulletin extends deepest sympathy to his wife and family. □



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