THE NOVA SCOTIA MEDICAL BULLETIN

GUEST EDITORIAL

The Health of Nova Scotians

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The leading causes of morbidity and mortality in western industrialized countries are heart disease, cancer and accidents. The "big three" account for about two thirds of all mortality in Canada and in Nova Scotia. Each is amenable, at least in part, to preventive strategies.

The purpose of this brief paper is to depict the status of Nova Scotians in terms of the "big three", and to ask the key question from the standpoint of prevention: "Why is Nova Scotia lagging behind most of Canada?"

HEART DISEASE

Nova Scotia experiences higher mortality rates from circulatory diseases than does Canada as a whole. Much of the difference is due to ischemic heart disease (Figure 1). Because rates for Canada are a composite, reflecting those from all provinces (e.g., other Atlantic provinces have high rates), a comparison has been made with Saskatchewan which has the lowest rates in Canada. While the overall decline is encouraging, the sheer magnitude of mortality and the size of the interprovincial differences should evoke more than simple curiosity. Do people in Saskatchewan receive better health care, are they genetically superior in some way, or do they simply live healthier lives?

CANCER

Nova Scotians experience higher overall mortality rates for cancer (especially among males), than do Canadians generally, much of which is due to an excess in respiratory cancers (Table I). Are the lungs of Nova Scotians inherently "weaker" than their Canadian cousins, have other parts of the country discovered a "breakthrough" -- kept as a closely guarded secret to perpetuate the myth of Shangri-la to the west, is it the dreaded North Atlantic fog, or are Nova Scotians simply more compliant to cigarette promotion, and literally smoking themselves to death at a faster rate than other Canadians?

From 1966 to 1981, male lung cancer mortality in Nova Scotia increased faster than in Canada generally-from among the lowest ranked to now the second leading province in Canada for this disease. Nova Scotia will win the competition with Quebec before the end of this decade, despite René Lévesque -- or perhaps because of? The 1981 rate for female lung cancer is ten times higher than the rate recorded in 1951. Lung cancer in Nova Scotian women will take over from breast cancer as the leading cancer site by 1987, and will contribute a quarter of all female cancer mortality in that year.

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ISCHEMIC HEART DISEASE, AGE STANDARDIZED MORTALITY RATES (per 10^5), NOVA SCOITA, SASKATCHEWAN AND CANADA 1970-1978

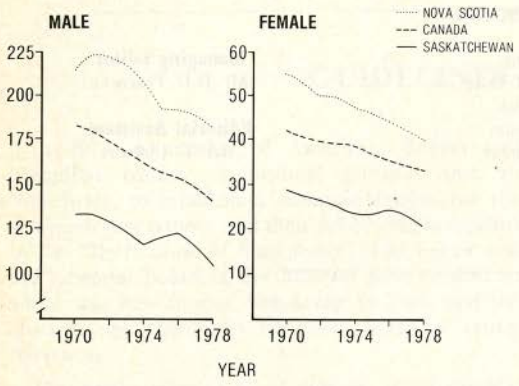

Figure 1

MOTOR VEHICLE ACCIDENTS

Thank Heaven, or at least the House of Assembly, that we finally have seat belt legislation. Better late than never. Among the last to implement such legislation, Nova Scotia has experienced higher age standardized mortality rates for motor vehicle accidents than the Canadain population for most years since at least the mid 1960s. Thousands have been killed, and tens of thousands maimed. Hopefully, with adequate promotion and enforcement of this new measure, along with the excellent effort on reducing "drunk driving", Nova Scotia will continue to show progress in these areas of accident prevention.

TABLE I

CANCER MORTALITY COMPARISON, NOVA SCOTIA AND CANADA, 1981

(based on Age Standardized Mortality Rates per 100,000)

	MALE		FEMALE	
	Nova Scotia	Canada	Nova Scotia	Canada
Total Cancer	260.3	237.3	171.7	169.4
Digestive Cancer	69.7	69.6	51.7	45.0
Respiratory Cancer	104.7	90.2	28.5	25.9
Breast Cancer	—	—	35.9	39.8
Genital Cancer	15.6	13.3	24.0	20.6
Urinary Cancer	11.1	11.9	3.9	4.7
Other Cancer	59.2	52.3	27.7	33.4

SOURCE: Health and Welfare Canada

RISK FACTORS

Heart Disease

Before discussing coronary risk factors, it is necessary to place them in epidemiologic perspective. "Primary risk factors" are those which are thought to contribute directly to the disease process and include both modifiable and non-modifiable factors. Age, sex, and family history are non-modifiable, the status of diabetes mellitus is debated, while hypercholesterolemia, hypertension and smoking are considered modifiable. "Secondary risk factors" are those which may have an indirect effect on the disease by influencing the primary risk factors, and include physical inactivity, obesity, alcohol and oral contraceptives.

Modifiable risk factors thought to influence the occurrence of ischemic heart disease (to varying degrees) are smoking, blood pressure, cholesterol, obesity and physical fitness. Taking Canada and Saskatchewan as yardsticks, how does Nova Scotia compare in relation to modifiable risk factors?

Smoking

Nova Scotia has a higher prevalence of smoking than Canada in every age group except those over 65 years. Saskatchewan has a lower prevalence in every age group (Table II). In addition, Maritimers generally smoke brands with a higher tar yield than our Prairie cousins. None of these new fangled cigarettes for us "byes"!

TABLE II

PERCENTAGE OF REGULAR CIGARETTE SMOKERS 15 YEARS OF AGE AND OVER BOTH SEXES COMBINED CANADA, NOVA SCOTIA AND SASKATCHEWAN, 1981

Age	Canada	Nova Scotia	Saskatchewan
15+	32.7	34.2	27.3
15-19	23.1	25.2	16.4
20-24	40.4	42.9	31.6
25-44	38.0	40.7	35.9
45-64	33.7	35.4	28.0
65+	15.3	15.1	12.6

SOURCE: Health and Welfare Canada

Blood Pressure

The comparison here, based on the Canada Health Survey, is conflicting. Mean diastolic blood pressure is generally lower in Nova Scotia than Saskatchewan while the reverse is generally true for systolic blood pressure. However, the percentage of Nova Scotians with a diastolic blood pressure higher than 94 mm Hg is greater than in Saskatchewan in the age range 25 to 64 years.

While I am less than fully confortable with the quality of available data on interprovincial blood pressure differentials, this does reflect what is currently available. Furthermore, it highlights the need for better information in the future. We take great pains to establish individual diagnosis -- but why do we not allocate equivalent priority to obtaining an equally adequate data base for community diagnosis? Where are the provincial blood pressure surveys?

Cholesterol

Average serum cholesterol, also determined by the Canada Health Survey, is higher in every age group in Nova Scotia than in both Canada and Saskatchewan (Table III). A comparison with data from the earlier Nutrition Canada Survey suggests that the interprovincial difference in cholesterol levels is widening in favour of Saskatchewan.

TABLE III

AVERAGE SERUM CHOLESTEROL LEVEL (mg/dl) FOR POPULATION 15 YEARS BY AGE GROUP: CANADA, NOVA SCOTIA AND SASKATCHEWAN 1978-79.

Age	Canada	Nova Scotia	Saskatchewan
All Ages	203	212	207
15-24	176	179	176
25-44	200	217	209
45-64	225	234	229
65+	225	233	223

SOURCE: Canada Health Survey 1978-79

Obesity

The Nutrition Canada Survey indicated a higher prevalence of obesity in Nova Scotia than in either Canada of Saskatchewan in most age-sex groups. Interprovincial comparisons based on more recent surveys have not yet been published.

Physical Fitness

Physical fitness has been evaluated in both the 1978-79 Canada Health Survey and the 1981 Canada Fitness Survey.

In 1978-79 a lower percentage of the Nova Scotia population 15 years and older was classified as active than in either Canada or Saskatchewan in every one of 6 age-sex categories analysed. Only 32% of Nova Scotians surveyed were found to exceed the recommended fitness level for age, sex and weight, compared with 40% for Canada and 34% for Saskatchewan. These comparisons are based on aerobic capacity, calculated from actual exercise testing.

However, the 1981 survey indicated a major improvement in all three jurisdictions, particularly in Nova Scotia whose population is now more physically active than the national population in all age groups except those 10-19 years where the rate is equal to the overall Canadian level although lower than the level achieved in Saskatchewan. Greatest relative gains were observed in the age group over 65 years. These trends are very encouraging because they suggest that, in Nova Scotia as elsewhere, substantial behavious changes can be accomplished over a relatively short time frame!

Cancer Risk Factors

That the common fatal cancers occur in large part as a result of lifestyle and other environmental factors and are in principle preventable was recognized by an expert committee of the World Health Organization in 1964. The evidence that much cancer is avoidable, as summarized by Doll and Peto in 1981, falls within four categories: "differences in the incidence of cancer among different settled communities, differences between migrants from a community and those who remain behind, variations with time in the incidence of cancer within particular communities, and the actual identification of many specific causes or preventive factors".

These authors estimate that, "in terms of age-sex specific rates, more than 90% of current cancers may be preventable" -- although this does not exclude the possibility fatal conditions at a later point in an extended life span. Recently a similar exercise to allocate by proportions the avoidable risks of cancer has been carried out for Canada (1981) and the following percentages emerged: diet 35%; tobacco 30%; infection 10%; reproduction 7%; occupation 4%; alcohol 3% and unknown 11%.

I would be quick to concede that these figures, based on the premise of shifting age-sex rates, are necessarily quite abstract. Nonetheless, they help to focus attention on the largely untapped potential for cancer prevention. Surely the medical profession should play a more active advocacy role in favour of prevention. Respiratory cancers alon in 1981 accounted for 24.9% of all cancer mortality in Nova Scotia (34.5% in males and 12.3% in females). As previously noted, Nova Scotia exhibits a higher prevalence of smoking than Canada in every age group except those over 65 years. Were it not for smoking, cancer would be decreasing as a cause of death — not increasing.

WHY ARE WE STILL IN THE JUNIOR LEAGUE?

Prevention appears to have received a low priority in Nova Scotia. Not only is this illustrated by the generally discouraging major mortality and risk factor status of Nova Scotians, but this deficiency has been vocalized by a very wide ranging spectrum of opinion in this province.

According to the recently released Report of the Select Committee on Health of the Nova Scotia Legislature: "prevention — defined as programs and services which avert the occurrence of disease, illness or dysfunction and which promote the optimal health of the population -- has long been recognized as a major theme in health care. However, emphasis in the health care system has been placed on curative medicine and treatment". In fact, "more groups and individuals referred to 'prevention' in their submissions to the Select Committee on Health than any other single issue", and "overall it was the view of the Select Committee than the balance between preventive health programs and the curative care system must be tipped more in the direction of prevention".

Interestingly these conclusions are consistent with the findings of the Canadian Medical Association sponsored Task Force on the Allocation of Health Care Resources. I would like to quote from the chapter on "Wellness":

"There are many examples of health education programs that could be cited. In Saskatchewan the "Feeling Good" program encourages good nutrition and exercise as a means of promoting good health, and the "Aware" program emphasizes the dangers of alcohol abuse. Both are funded by the Ministry of Health. A classic example of a health education program is the North Karelia Project in Finland. The toll of heart disease has been greater in Finland than in any other country. Curious regional differences were observed within the country, with the highest rates found in the province of North Karelia. The local population signed a petition asking for help. A program was planned and funded by the government. In order to reduce heart attacks, particularly among middle-aged men, the government decided to promote healthy life-style changes including non-smoking and low-fat diets to reduce the known heart disease risk factors: smoking, serum cholesterol, high blood pressure. The plan also included early detection, treatment and rehabilitation of patients. Information was provided through newspapers, radio, leaflets, posters, health education meetings, and programs at school and work. Banning cigarette advertising was part of the campaign.

In five years, they reduced the incidence of severe heart attacks by 20 per cent. The proportion of male smokers decreased by 17 per cent and that of female smokers by 22 per cent. This experience shows that a well-conceived community-based program can have a meaningful impact on the lifestyles, risk factors, cardiovascular disease rates and general health status of the population and that through this kind of systematic action, the modern epidemic — heart disease — can be effectively fought".

Perhaps North Karelia should become the role model for Nova Scotia, and Atlantic Canada generally.

Further on the Task Force observed that "fee schedules could be amended to encourage doctors to spend more time on counselling and consultation". Is this the final obstacle in achieving change within the medical sector of the health care system? Lack of knowledge and payment for prevention services mean doctors don't do as much preventive work as they should— resulting in other health professionals and lay people doing more of it, thus it seems putting them at odds with the medical profession.

ENVOI

I shall give the last word to Sir William Osler (1849-1919): "To wrest from nature the secrets which have perplexed philosophers in all ages, to track to their sources the causes of diseases, to correlate the vast stores of knowledge that they may be quickly available for the prevention and cure of disease — these are our ambitions."

In the area of prevention, it would appear that Nova Scotians have a lot of catching up to do. The need is demonstrable, the knowledge is available, and the political will finally might have surfaced. What we need now is "Prevention in Action" not just in words. □

ACKNOWLEDGEMENTS

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Hooked on Tobacco

The political conflict between wealth and health is as old as tobacco itself. Governments have long condemned it, whilst rubbing their hands at the money it brings into their Treasuries. When King James I came to the English throne in 1603, he described smoking in *A Counter-Blaste to Tobacco* as 'a custom loathsome to the eye, hateful to the nose, harmful to the brain, dangerous to the lungs'. To discourage smoking, he increased the tax on tobacco by 4,000 per cent from two pence a pound to six shillings and eight pence a pound. He then found that tobacco was a source of much-needed revenue which was not being fully tapped because people could not afford to smoke. He slashed the tax to two shillings a pound, and money poured into the royal coffers. Governments have been hooked on tobacco ever since.

Smoke Ring: The Politics of Tobacco,
by Peter Taylor.
London: The Bodley Head, 1984.

A Community Hospital Smoking Survey

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Nova Scotia has the country's worst provincial smoking record. In 1982, 48% of its inhabitants over age fifteen smoked. One hundred thousand Nova Scotians may be expected to die prematurely as a result of their habit. A survey conducted at a Nova Scotia community hospital amongst staff and patients (between ages 11 to 70 years) showed that 58% of the total survey group smoked. Doctors smoked the least (25%), and outpatients smoked the most (74%). Over one half of the nursing staff smoked (52%). The proportion of smokers (in the 11 to 70 year age group) in the community may approach 70%, leaving it with a serious public health problem. Recommendations include a firm hospital nosmoking policy and health education programs, as part of a concerted effort to promote community health.

INTRODUCTION

Cigarette smoking was first linked to lung cancer in the late 1940s, and since then we have also become aware of its causative role in ischemic heart disease, chronic obstructive pulmonary disease (COPD), fetal morbidity and mortality, and several forms of non-pulmonary cancer. Recent evidence suggests that non-smokers exposed to "second-hand" smoke may as a result experience a higher incidence of respiratory infections, lung cancer and acute exacerbations of angina and COPD[1]. Cigarette smoking has been cited as the largest cause of preventable death, the largest cause of premature death, and the greatest single cause of disease in developed countries.[2,3]

However, despite our knowledge and public health education programs, Canada still has one of the highest per capita consumption rates of cigarettes in the world.[3] In a 1982 survey, approximately 35% of Canadians over the age of 15 smoked regularly (at least once daily).[4] Nova Scotia held the country's worst provincial record: 47% of its 855,000 inhabitants were regular smokers. If one in four smokers can expect to die prematurely from their habit[5], then over 100,000 Nova Scotians are smoking themselves to an early death.

Despite a decline in smoking amongst Canadian males and certain professional groups, the overall national proportion of regular smokers changed little from 1975 to 1982 (it dropped only from 37.3% to 35%).[6] This piece of discouraging news has been variously attributed to several factors. Many feel our educational programs are inadequate to meet the public health problem. (A recent U.S. government poll indicated 50% of cigarette smokers were unaware that smoking was a major risk factor for heart attacks).[6] Others feel too little research and teaching has been done in smoking cessation techniques. Perhaps most importantly, the tobacco industry has not remained idle through all of our warnings. Cigarettes are the most heavily advertised product in America, where in 1980 expenditure for promotion exceeded one billion dollars.[6]

While serving as a G.P. locum in a Nova Scotia community hospital, I was struck by the apparently high proportion of patients and nurses who smoked. Patients and their visitors frequently smoked in patients' rooms and in the outpatient's waiting room (and even in the treatment cubicles!). Despite hospital policy, this activity seemed to go unchallenged by the hospital staff.

Was the problem of smoking in hospital by patients partly a reflection of the smoking behaviour and attitudes of staff? A survey was conducted to determine the prevalence of cigarette smoking in both patient and staff groups.

MATERIALS AND METHODS

The community hospital surveyed has 88 beds and serves a population of 9,000, many of whom are coal miners and their families. In 1984, approximately 2500 patients were admitted, and another 40,000 were seen in its O.P.D. — emergency department. The hospital has four wards (medical, surgical, pediatric and obstetrics), an active general surgical O.R., and a staff of 190 people.

With the help of two volunteers, the author surveyed all staff and patients (aged 11 to 70 years inclusive), who were present in the hospital at any time over the 24 hour period of Wednesday, September 12, 1984. (There was nothing unique about the survey date chosen. It consisted of the same type and quantity of workload and staffing as any other weekday).

By direct interrogation, 240 people were surveyed: 123 staff, 86 outpatients and 31 inpatients. The following information was gathered from each person: age, sex, hospital status (staff position, inpatient or outpatient) and cigarette smoking status (nonsmoker, smokes one pack or less a day, or smokes more than one pack per day).

The staff were divided into six subgroups: doctors, administration, registered nurses, nursing assistants (C.N.A.), para-medical staff and non-medical staff. Note that the para-medical staff consisted of 7 laboratory tech-

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nicians, 4 E.C.G./X-ray technicians, 2 pharmacists, 2 respiratory technicians and 1 physiotherapist. The non-medical staff consisted of 11 dietary workers, 11 clerical staff, 10 house-keepers, 8 laundary staff, 3 security guards and 2 maintenance workers.

RESULTS

Figure 1 shows that 58% of the total survey group smoked, and 9% of the entire group smoked more than one pack of cigarettes a day (hereafter defined as "heavy smokers"). Although males smoked more than females (68% vs 55%), a larger proportion of females were heavier smokers (10% vs 6%).

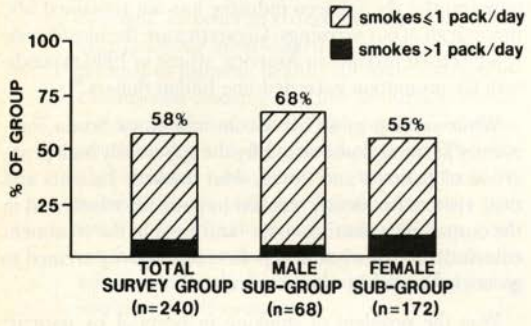

Figure 1. Percentage of smokers in total hospital survey group, and male:female sub-groups.

Figure 2 illustrates smoking behaviour according to age groups, which are arbitrarily divided into six decades. Although the 11-20 year olds had the highest proportion of smokers (67%), caution must be exercised when interpreting this result as only nine people were surveyed in this group. From age 21 years onwards, the proportion of smokers rises in each progressive age group, peaking in the 41-50 year olds, and falling off considerably thereafter.

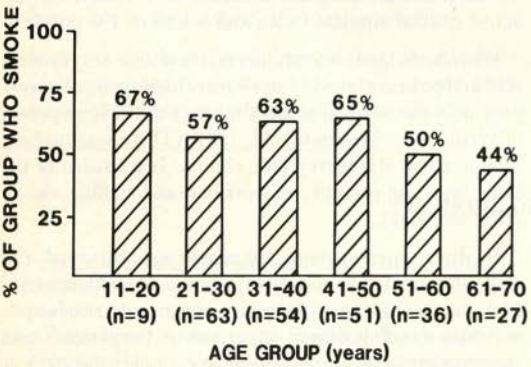

Figure 2. Percentage of smokers according to age group.

Table I lists the eight patient and staff subgroups and various characteristics of each.

TABLE I

CHARACTERISTICS OF HOSPITAL SURVEY GROUPS

Group	Number	% Smokers	Mean Age ±1 s.d. (years)	Female: Male Ratio
M.D.	8	25	43±13	1.7
Para-Medical Staff	16	37	37±11	15:1
Administration	8	37.5	51± 9	8:0
Inpatients	31	39	52±17	1.5:1
R.N.	31	52	37±12	31:0
Nursing Assistants (CNA)	15	53	34± 9	14:1
Non-Medical Staff	45	65	40±11	8:1
Outpatients	86	74	40±15	1:1
	240			

Figure 3 illustrates the smoking behaviour of the Table I groups. Outpatients had the highest proportion of smokers (74%), and the M.D.s had the lowest proportion (25%). The highest proportion of "heavy smokers" were found in the two patient groups (each with 13%). Again, the low numbers surveyed in the M.D. and Administration groups dictates caution in interpreting their results. The R.N.s and C.N.A.s had a roughly equal proportion of smokers (52% vs 53% respectively). However, the C.N.A.s had a higher proportion of heavy smokers (6% vs 3%). Outpatients smoked considerably more than inpatients (74% vs 39%), but outpatients were also considerably younger on average (mean ages: 40 yrs vs 52 yrs old).

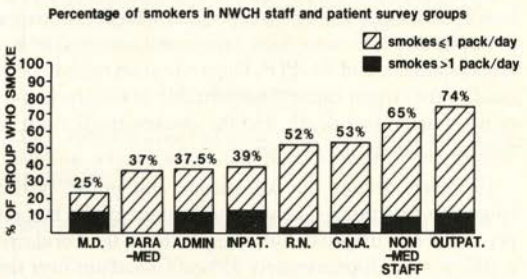

Figure 3. Percentage of smokers in hospital staff and patient survey groups

CONCLUSIONS AND RECOMMENDATIONS

Some of the results of this small survey confirm several previoulsy recognized patterns of smoking behaviour. Namely, men smoke more than women; higher socioeconomic groups smoke less than lower ones; and smoking correlates roughly to age groups, with an increasing percentage of smokers seen up until middle age (30-50 years of age), and declining thereafter.

However, contrary to previous evidence[7], this survey demonstrated more heavy smokers amongst women than men (10% vs 6%). It should be noted that heavier smokers

carry a higher risk of illness and premature death than light smokers. Furthmore, as a result of their smoking habit, 1985 marks the year when lung cancer will surpass breast cancer as the number one cancer killer in women.[8] Another interesting feature of this survey showed that nurses smoked condsiderably more than the para-medical staff (52% vs 37% respectively), and in the same proportion as the C.N.A.s. Note that all three groups were very similar with respect to average age and sex ratios (see Table I).

One might have thought that nurses, with their more intensive and specific health education, would have smoked *less* than the other two groups. Does this finding reflect a deficiency in the nursing education program or, does it indicate that education and awareness of the hazards of smoking are not the only factors determining whether one smokes or not? In this as in any small community, where nurses are highly respected, surely their attitudes and behaviour towards smoking influence that of their patients. In a situation where over 50% of the nurses and C.N.A.s smoke, how likely are patients to be warned or counselled if found smoking in hospital? Conversely, when patients see nurses and C.N.A.s smoking, what of their perceptions of the dangers of smoking? They may well rationalize that if nurses smoke, smoking cannot be that bad for you.

This survey also demonstrates that almost 75% of all outpatients smoked. Several factors may explain this high proportion: 1) a chance event on the survey day; 2) the fact that smokers suffer more illness and see their G.P.s more often than non-smokers[2]; or 3) outpatients simply reflect the proportion of smokers in the community. Repeated observations have convinced the author that the third factor is the most important.

A 1982 Canadian Cancer Society survey of 160 Nova Scotians over the age of fifteen, found that a total of 48% smoked (47% regularly and 1% occasionally). In contrast, the community hospital survey studied a select group of 240 people aged 11-70 years and found that 58% smoked. Presumably, of all the groups, the non-medical staff (65% smokers) and the outpatients (74% smokers) are most representative of the community at large. If this assumption is valid, then the proportion of smokers in the community (between the ages of 11-70 years) may well approach 70%, leaving the area with a very serious public health problem. The importance of this is further underlined because in this coal-mining town many men will be at a high risk of C.O.P.D. from both coal dust and cigarette smoking.

In this regard, there are several steps the hospital and its staff can take to assert its leadership role in promoting community health.

1. Health Education Programs

Extensive and repeated quit-smoking campaigns should be conducted amongst the hospital staff, with information pamphlets, seminars and support groups made available. As well, a coordinated effort should be made to establish smoking education courses in nurse's training programs and public schools.

2. A Smoking Ban in the Outpatients Department

The outpatient department is where a large proportion of the public makes its contact with the hospital. The community must be made clearly aware that the hospital and its staff are for health, not for smoking. Besides irritiating non-smokers, we have already seen that second-hand smoking may actually exacerbate those prone to angina, asthma and C.O.P.D.

3. A Smoking Ban for Hospital Visitors

Again, for all the reasons stated in recommendation two, the hospital must firmly demonstrate it stands for health, not smoking.

4. A Ban on the Sale of Cigarettes in the Hospital

5. A Smoking Ban in Patients' Rooms

Inpatients must not smoke in their rooms, as this may aggravate other patient's conditions, and pose a fire hazard as well. (There are several reports in the medical literature of patients who have set either themselves, their beds or their hospitals on fire). Physicians should endeavour to encourage their patients to quite smoking whilst in hospital. This is especially relevant for patients with respiratory, cardiovascular or surgical disorders. Minor tranquilizers or nicotine chewing gum may be prescribed for a short period if necessary to overcome any nicotine withdrawal effects.

6. Restricted Staff Smoking

Staff smoking should be highly discouraged, and if permitted at all, only in restricted areas well away from all patient contact areas.

7. Enforcement of Rules

If the hospital is to assert itself as a credible leader of health promotion, its non-smoking policy must be firmly enforced . "No-smoking" signs must be clearly posted throughout the hospital, and hospital staff must enforce the rules diligently. This should be monitored regularly by staff supervisors. With such a high prevalence of smoking amongst staff, patients and members of the general community, there is bound to be considerable resistance to such rules. However, strong resolve and firm leadership on the hospital's part must overcome this, for the public health problem is too serious to ignore.

There are 9000 people living in the community survey area, roughly 7000 of whom are between the ages of 11 and 70 years. Even if only 60% of this age group smoke (a conservative estimate), and knowing that one in four smokers die prematurely form their habit, then 7000 x 60%

x ¼ = 1050 people are smoking themselves to an early death. The hospital and its staff must set an example for the whole community to see. They must stand for health, and not for smoking and its deadly consequences. As we have just seen, there are at least 1000 reasons why this is so important and necessary.

8. Re-evaluation

Staff supervisors and administrators should meet on a regular basis to assess and improve the no-smoking policy, and they in turn should report to the hospital board on their progress. A repeat survey should be conducted a year after implementation of the no-smoking policy, in order to assess the effectiveness of the above measures.

9. A Total Ban on Hospital Smoking

The above measures still allow for restricted smoking in hospital, but this should serve only as an initial policy of change. After a period of time (ie 6 to 12 months), a total ban of smoking in hospital must be instituted. This is vital if the philosophy of health promotion is to be adopted in a credible and consistent manner.

One hundred thousand Nova Scotians are currently smoking themselves to a premature death. It is the author's hope that administrators and hospital boards across the province will review their smoking policies in light of the above recommendations. □

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Temporal Arteritis

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Temporal arteritis is a granulomatous arterial inflammation that characteristically involves the temporal and ophthalmic arteries, but may be part of a more widespread involvement of medium and large vessels. The patient usually presents with headache, constitutional symptoms (fever, malaise, anorexia, weight loss and anemia), and rheumatic complaints (polymyalgia rheumatica). The diagnosis should be considered in any patient over 55 years of age who develops these constitutional symptoms, or has recent onset of headache, sudden visual loss or localized arterial involvement.

The diagnosis is supported by a sedimentation rate over 50 and confirmed by a biopsy of the temporal artery. Visual loss may occur in 50% of untreated patients; other serious complications are less common. Because of seriousness of long-term steroids in the elderly, it is mandatory to carry out a biopsy in all cases even if the urgency of the situation requires initiation of therapy first. Although a self-limited disease, treatment with steroids is imperative to prevent blindness and it must be maintained for a prolonged period.

Temporal arteritis (giant cell arteritis; cranial arteritis) is a granulomatous inflammatory disease of medium sized arteries manifesting primarily with temporal and ophthalmic artery involvement. It was first described by Jonathan Huthchinson in 1890[1] and was clearly defined as an entity by Horton *et al* in 1932[2] as a syndrome in an elderly patient of headache, visual disturbance and constitutional symptons of malaise, fever, anorexia and weight loss.

Incidence

Studies suggest an average annual incidence rate of 3 per 100,000 with women affected four times as frequently as men.[3] The incidence over age 50 is 17.4 per 100,000 and appears to be increasing[4] although this may reflect better diagnosis.[5] Clearer definition of the syndrome, along with descriptions of its milder variations, suggest that the incidence may be considerably higher; a number of cases do not come to the physician's attention and others are not diagnosed, largely because no serious complications develop and the disorder may be self-limiting. At autopsy 1% of the population have changes of temporal arteritis.[5]

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Etiology

Temporal arteritis is part of the spectrum of autoimmune collagen vascular diseases and may involve a foreign body reaction to some non-specific agent or irritation.[6] Anti-IgG activity of IgA, together with complement in the biopsies of patients with this disorder, strongly indicates that antigen/antibody complexes or otherwise denatured immunoglobulins are present in the vessel wall.[7] There seems to be a definite relationship between the clinical-histological stages and the presence of anti-IgG activity in the tissue lesions. Although many vessels can be involved, it is puzzling that the temporal artery is the major vessel involved. One interesting suggestion was that the temporal artery is the target vessel because of the local pressure and trauma of wearing eye glasses.[8]

Pathological features

The granulomatous arteritis primarily involves medium and large sized arteries, usually the temporal artery and ophthalmic arteries on both sides. Bilateral involvement is frequent despite the unilateral presentation of most patients.[9] Any branches from the aorta may be involved including the coronary, subclavian, carotid, renal, mesenteric and iliac arteries.[6] Although the arteritis may be focal in that it involves only certain vessels, "skip lesions" also occur in the involved artery[10] although this is probably an overrated phenomenon. Bilateral involvement is usually present[9] but occasional unilateral involvement may occur.[10]

Granulomatous arteritis involves the inner layers of the media of the vessel with cellular infiltration of lymphocytes, and some neutrophils, plasma cells, eosinophils and macrophages. The number of characteristic giant cells is variable. Some areas show necrosis and others show connective tissue repair. The connective tissue thickening may obstruct the vessel lumen and enlarge the temporal artery to give it its cordlike, pulseless feel on palpation. In some cases, there is fragmentation of the internal elastic lamina. Although the vessel may totally occlude, it can recanalize later when the disease burns itself out. The vessel changes are characteristic and allow the diagnosis when vessels involved elsewhere are biopsied and show the same changes.[11]

The changes occurring in the temporal artery from normal aging can be differentiated from arteritis, and the residual changes of temporal arteritis with characteristic zonal arrangements of the intima can be identified years later.[12]

Clinical manifestations

The typical picture is that of an elderly patient, who presents with a history of many months of general malaise, weight loss, anorexia, and mild recurrent fever. Most such patients initially present with unilateral headache and scalp tenderness. Examination shows a generally unwell person with a pulseless, tortuous, cordlike temporal artery and redness and tenderness over that area of scalp. Laboratory studies show a high sedimentation rate, often over 100, anemia, and other hematogolical characteristics of chronic disease. A biopsy of the temporal artery shows the characteristic giant cell arteritis and the patient responds dramatically to steroids.

It is important to recognize, however, that many patients are not typical and the diagnosis is then made by having a high level of suspicion in elderly patients with part of the clinical picture, using appropriate tests to confirm or exclude the disease.[13] It is a constellation of protean manifestations, and no simple clinical sign, symptom or test can be relied on alone.[14] Indeed, Hamilton *et al* listed 36 different clinical presentations of temporal arteritis.[15]

Headache

The headache varies but is usually unilateral and felt superficially in the scalp. The pain may be described as aching, jabbing, or throbbing. Headache may also be experienced over the frontal and occipital regions; onset is acute in some cases but gradual in most. Although the headache is not specific, temporal arteritis should be suspected in any elderly patient who presents with a recent onset of headache, particularly if the patient has constitutional symptoms. Early in the course the palpatory abnormalities may not be present but biopsy will still be diagnostic.[16] Jaw pain associated with talking or chewing (jaw claudication), is seen in half of the cases and may occur without the temporal headache. It is a reliable indication of temporal arteritis as it rarely occurs in other forms of vasculitis.[17]

Visual Symptoms

If untreated, about half of the patients with temporal arteritis develop visual symptoms and sometimes blindness. Sudden blindness is the presenting symptom in a few cases. Although the syndrome may otherwise be a benign and self-limited disorder, the possibility of blindness makes temporal arteritis an important disease to recognize and treat as an emergency. The arteritis in the ophthalmic artery causes blindness by causing an ischemic palpillitis or a central retinal artery occlusion. An ophthalmoplegia may occur.[18] Occasionally, there are recurrent transient visual symptoms, but the loss of vision is usually acute, and very often becomes bilateral. When the visual symptoms are fully developed, response to therapy is poor and residual visual deficit or blindness usual. Meadows has emphasized the relatively minor fundus changes in most cases of blindness so you cannot be reassured by a negative examination in these cases.[18]

Mental changes

Occasionally, confusion or dementia develop as part of the progressive symptomatology of temporal arteritis, particularly if the patient has borderline cerebral function which is aggravated by the constitutional symptoms of mild fever, malaise, anorexia and weight loss. Involvement of the intracranial vessels, and ischemia due to involvement of the carotid and vertebrobasilar arteries are possible.

Fever

Although some patients with temporal arteritis present with fever of unknown origin, this symptom is usually mild.

Cerebrovascular symptoms

Acute cerebral infarctions may occur, and with myocardial infarction, accounts for 12% of deaths among patients with temporal arteritis.[19]

Polymyalgia rheumatica

Polymyalgia rheumatica is part of the syndrome of temporal arteritis. 30-50% of the affected patients will have positive temporal artery biopsies, even if they have no headache. These patients present with widespread muscle and joint aching, joint stiffness, and the other constitutional symptoms associated with temporal arteritis.[20] Actual joint swelling and muscle weakness are unusual, and stiffness is the chief symptom and finding. The syndrome is often migratory and aggravated by damp weather, and worse at night. The laboratory findings are the same as those in typical temporal arteritis. Patients respond to anti-inflammatory agents, but since temporal arteritis may still develop with these drugs[21], steroids must be used.

Skin lesions

Local changes over the temporal arteries are common, with redness, tenderness and slight swelling. Actual necrosis of the scalp and even the tongue, occur rarely.[22]

Cardiac involvement

Involvement of the coronary arteries is another cause of death in these patients. Occasionally, recurrent angina is a manifestation and this may be refractory to treatment until steroids are used.

Limb involvement

Peripheral arterial insufficiency can occur. Gangrene may be more frequent than recognized because some cases are regarded as Buerger's disease in which giant cells are also found. In Hutchinson's original report in 1890 he commented on the complication of gangrene. Other possible sysmptoms of temporal arteritis are intermittent claudication, paresthesia and Raynaud's phenomenon.[10]

Diagnosis

The diagnosis must be suspected in more subtle clinical situations than had previously been recognized for this disorder. Any of the above symptoms or problems in a patient over 55 years of age (and more often over 65), particularly if assoicated with a sedimentation rate over 50, should make one suspect temporal arteritis. The two most reliable signs that correlate with a positive biopsy, are jaw claudication and a thickened, pulseless temporal artery. Although the clinical picture may be characteristic, and the findings of a pulseless, cordlike and inflamed temporal artery may be present, some laboratory investigations are necessary to confirm the diagnosis.

The erythrocyte sedimentation rate (ESR) is the simplest and most useful indicator initially; it is almost over 55 mm/hour and often over 100. Rarely it is normal in biopsy proven giant cell arteritis.[23] Other less specific laboratory findings include anemia, hypochromasia, rouleaux formation, low serum iron level, mild leukocytosis, toxic changes in the leukocytes and low serum albumin with an elevated IgG, IgA, total complement and complement factors C3 and C4.[22,23] Other observations have included increased blood fibrinogen, thrombocytosis, and eosinophilia. Selective arteriography of the external carotid artery may show very characteristic segmental narrowing of the temporal artery.[25]

The most important diagnostic procedure in temporal arteritis is the temporal artery biopsy. This should be carried out in every suspected case and a long segment of the temporal artery should be taken. Although the biopsy procedure may relieve the headache, the patient must still be treated with steroids to avoid possible blindness later. As steroid therapy is a long term commitment with a high complication rate it must be certain that the patient indeed has the disorder. Often a neurologist is asked to evaluate a patient treated with steroids for temporal arteritis who now has complications and the physicians have second thoughts about the diagnosis that was never proven.

Treatment

If there is a positive biopsy the patient should be started immediately on steroids. In fact, if the clinical suspicion is strong and there is concern about visual complications, the patient can be started on steroids when first seen and biopsy done within 24-48 hours without interfering with the results.

If the biopsy is negative there is a 94% chance that in long term follow-up the patient will not require steroids.[17] Such a reliable prediction assumes the surgeon always removes a large segment of the artery.

In suspected cases of temporal arteritis the biopsy should be the guide to determine the need for long term steroids.[26] If the biopsy is negative, follow and continue to investigate the patient for other causes of the symptoms. If positive, a long course of daily steroids is necessary with close follow-up over a number of years.

Although temporal arteritis is sometimes benign and self-limiting, the threat of blindness in up to half of the patients makes treatment imperative and urgent. A reasonable regimen is 60 mg/day for four weeks, followed by a reduction of 5 mg every two weeks to a maintenance dosage of 15 mg/day. Maintenance therapy is continued for up to two years, but further slow reduction in therapy can be made if they remain well and if the ESR remains low. If the doses are reduced too rapidly, or therapy discontinued prematurely, recurrence is possible.[26] The patients must also be on an ulcer regimen during steroid therapy, and other steroid precautions must be followed.

Unfortunately, if alternate day steroids are used to minimize side effects, the therapeutic response is poorer with recurrence of symptoms on the "off" days and more patients will continue to suffer ongoing symptoms of the disease.[27]

The ESR can be used as an indicator of the success of treatment. If the rate drops to less than 15-20 mm/hour the initial 60 mg of prednisone per day can reduced sooner. The ESR should be tested monthly after the cessation of therapy for a few years, even though the disease seldom recurs after a prolonged period.

Steroid therapy is necessary, not only to decrease symptoms and shorten the course of the disease, but also to prevent serious complications. Unfortunately, blindness may still occur in a few patients. There is little likelihood of reversing the blindness when therapy is started after visual symptoms are well established. Kearns has suggested that hyperbaric oxygen could be useful in emergency treatment of visual loss from temporal arteritis but it would be impractical in most instances, as retinal infarction occurs so rapidly.[28] It would be worth trying if symptoms were progressive and a chamber were immediately available.

Prognosis

A mortality rate of 12% has been recorded in the disorder[19] and there may be many patients who have progressive temporal arteritis and die of coronary or cerebral involvement without being diagnosed. The next most serious problem is that of visual loss. Otherwise, the disorder tends to have a very good prognosis. It seems facetious to say that if the patient doesn't die or go blind they will likely do well, but that is the case. Patients respond dramatically to steroid therapy and often the disease has a self-limited course. It may persist for may years, however, so long term follow-up is required.[29]

Unfortunately, steroid therapy is associated with many complications in the elderly and these patients are all elderly.[27] If the patient is diabetic or has other relative contraindications to steroids, a lower dose (40 mg daily or 80 mg on alternate days) can be used initially and the dosage reduced to maintenance levels more quickly if the response is good. □

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Non-Invasive Evaluation of Arterial Disease in the Lower Extremity

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During the past decade, the development of non-invasive vascular diagnostic techniques has expanded at a rapid pace. The advantages provided by these techniques are: a) localization of occlusive lesions and their functional repercusions; b) objective follow-up of surgical and non-surgical patients; c) help to determine the level of amputation; and d) differentiate patients with leg pain of vascular and non-vascular origin.

In most cases patients can be selected and referred for angiography only when surgery is contemplated.

A detailed history and physical examination provide the physician with sufficient data to suspect peripheral vascular disease of the lower extremity. These patients usually present with signs and symptoms that belong to one of the three following groups: a) intermittent claudication; b) rest-pain (pre-gangrene); c) gangrene.

The magnitude and precise location of the obstructive lesions can not be accurately determined by the above methods alone. Adjuvant diagnostic methods are non-invasive vascular tests and angiography, these two techniques compliment each other.

Angiography visualizes the morphology and precise location of these lesions. Non-invasive vascular studies provide objective information of limb performance (physiological repercussion of the lesion). These studies range from simple bedside or office techniques to sophisticated specialized vascular laboratory monitoring.

The purpose of this article is to describe the most common non-invasive techniques for all those physicans involved in the management of vascular patients.

CONTINUOUS-WAVE DOPPLER ULTRASOUND (CWD)

The most commonly used method for functional arterial evaluation is CWD. Portable devices are easy to use, readily available in most hospital areas and relatively inexpensive. Their pocket size makes them ideal for bedside or office use. The larger units are used in the vascular laboratory and allow recording of the signal, thus visual and numerical interpretations are possible.

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These devices operate by emission of a continuous ultrasound beam. The hand held probe is equiped with two crystals in its tip. One crystal emits the beam and a second crystal receives it. A water-soluble gel is applied between the patients skin and the probe. This gel is used as a coupling agent to avoid air interface. The ultrasound wave is reflected from the erythrocytes in proportion to their velocity. The pitch of the audiosignal is proportional to this velocity, i.e. a high-pitched sound indicates high velocity, a low-pitched sound indicates low velocity. These signals can be visualized in the laboratory as waveform tracings or by spectral analysis (sonogram), the former being the most commonly used. Both are reliable methods for qualitative analysis.

Quantitative measurements of the above signals can be obtained by the following techniques: a) pulsatile index (PI); b) damping factor; c) spectral analysis; and d) velocity measurements. These values are obtained by formula or computer analysis and are beyond the scope of this paper.

SEGMENTAL PRESSURE MEASUREMENTS

Significant and valuable information can be obtained by measuring pressures at different levels in the lower extremities. A CWD is used to obtain the blood velocity signal. Blood pressure cuffs and a regular blood pressure manometer are used as well to obtain these sequential pressures. It is important that the size of the cuff be appropriate to avoid false readings. In most patients the regular size cuff (arm) can be used for pressure measurements at the calf and ankle levels. The air-bladder size should be 20% wider than the diameter of the limb. The cuff is inflated above the patients systolic pressure and then slowly deflated. The audiosignal will be heard with the resumption of flow, this equals the systolic pressure in the artery underlying the cuff.

It must be remembered that the obtaining pressure is not an indication of the pressure or flow under the probe but the one under the cuff. Thus with the probe at the level of the dorsalis pedis artery, by simply applying the cuffs at the thigh, calf and ankle levels values can be obtained for these three different segments. The difference in pressure between cuffs can be calculated as a gradient; for instance, if a given patient has a segmental pressure at the level of the thigh of 120mm Hg and at the calf level has only 80mm Hg, the gradient between the two is 40mm Hg. This indicates an area of stenosis between both cuffs that induces such a reduction (Table I).

TABLE I

EVALUATION OF STENOSIS BY DIFFERENCE OF PRESSURES BETWEEN TWO CUFFS. (I.E. THIGH AND CALF)

Pressure Drop Between Cuffs	Indicates:
> 30	Stenosis
> 50	Tight Stenosis or Occlusion

Occasionally, due to severe distal disease, the Doppler signals may be difficult to obtain. In cases of such low flow a strain gauge plethysmograph may be used in the vascular laboratory because of its higher sensitivity.

ANKLE PRESSURE INDEX (API)

The upper extremities are rarely affected by occlusive disease, and thus the pressure in the arm vessels can be used as baseline indicators for comparison with the lower limbs. Doppler ultrasound pressures recorded at the calf or ankle level normally are slightly higher than arm pressures. This occurs as a consequence of the pulse wave progressing distally.

The ankle pressure should be divided by the arm pressure to obtain the index (API). This ratio is normally one or greater than one, and lower values indicate occlusive disease. API between 0.7 and 0.5 are present in claudication and usually indicate signal lesions. Values below 0.5 are seen in patients with rest pain ulcers or gangrene (Table II). It must be remembered that in diabetic patients, API may have an abnormally high value due to calcified arteries that are difficult to compress. In average, a 10% increase may be seen in the lower extremities.

TABLE II

EVALUATION OF LOWER EXTREMITIES BY API

Ankle/Brachial Index (API)	Indicates:
1 or higher	Normal
0.7 to 0.5	Arterial Claudication
below 0.5	Rest Pain Ulcers or Gangrene

Calculation of API will also provide an accurate and objective follow-up. Decrease of API indicated worsening of the lesions while an increase signifies development of collateral circulation.

EXERCISE TESTING

Exercise testing is the most valuable test for assessment.

The patient is allowed to rest on the supine position for 15 minutes. The resting pressure is recorded. Then the patient walks on a treadmill with a 10 to 15° inclination at 2.5 km/hr. When severe pain ensues the exercise is terminated. Brachial and ankle pressures are recorded immediately and at one minute intervals until they reach the baseline values.

The onset of claudication can be accurately determined and reproduced. The test provides an objective indication for operation and reliability in follow-up and results. In patients in which the pressure decreases to zero, multiple occlusions should be suspected. If the patient does not experience pain after five minutes of exercise, the degree of arterial disease is not significant and the test should be terminated. If there is a previous history of angina, the test should be done with EKG control, or not at all in the presence of severe disease. □

Extensive Bibliography available from the author.

Homeopathy

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INTRODUCTION

Today's professions of Medicine and Medical Research follow essentially the same guiding principles and beliefs that inspired Hippocrates in his famous oath. He believed that a physician's duty was to heal and to procure knowledge in order to heal better. Although this statement is easily understood, disagreements on its interpretation have led to heated conflicts throughout medical history. If one applies Darwin's theory of Natural Selection to this case, it can be said that the doctrine of today (ie. modern medicine) has survived because it is best suited for our needs. Homeopathy was one of the most formidable foes of modern medicine or allopathy. This conflict still continues today, but homeopathy is a declining doctrine and is now classified as a "fringe medicine". An understanding of homeopathy would be incomplete without some knowledge of its historical development.

HISTORY OF HOMEOPATHY

Samuel Hahnemann

Christian Gottfried Samuel Hahnemann was born in the town of Meissen, Germany in the year 1755. From here, in twenty-five short years, he set out to do battle with contemporary medical tradition. By 1779, after wandering through many parts of Germany, he graduated with a degree of Doctor of Medicine from the University of Erlangen. Soon, after he started his general practice, he became dissatisfied with the customary traditional practices of his time. This dissatisfaction translated into a restlessness that sent him travelling constantly over Europe for the next five years.

Origin of the Basic Homeopathic Principle

Eventually, Hahnemann gave up practising medicine and instead, took up chemistry and writing. He began to dispute the "heroic" treatments of his contemporaries through letters and publications. In one such correspondence, he wrote on the reason why cinchora (China or Peruvian bark) had curative powers in malaria. After trying the drug on himself while he was in a healthy state, he simply stated his observation that it induced symptoms similar to those presented by malaria patients. This observation is said to be the first beginnings of the homeopathic principle.[1]

Remove Cause and Contraria Contraris Versus Similia Similibus

After this first incident, Hahnemann continued testing drugs on himself while he continued to travel extensively. Finally, in 1796, he settled in Königslutter, Germany and published a paper in a medical journal entitled, "Essay on a New Principle for Ascertaining the Curative Powers of Drugs, and Some Examinations of Previous Principles". In this, he discussed the two principles which guided traditional medical thought. The first was the "Remove Cause" concept which was the belief that if one removes the cause of a disease, then there would be no further damage to the body. Though he admitted that it may be true, he quickly dismissed it as being a futile quest because, in his time, no one had ever been able to establish the fundamental cause of any illness. The second line of thought was to treat opposites with opposites, "Contraria Contraris". Hahnemann argued that this mode of treatment (which often involved bleeding and purging) was always unsuccessful in curing diseases. Although it relieved the patient temporarily, it did more harm than good in the long run.

And so, Hahnemann suggested the alternative that he had been experimenting on for years:

> "I must ask my colleagues to desert this way; it is the wrong one ... One should imitate nature which at times, heals a chronic disease by another traditional one. One should apply, in the disease to be healed, particularly if chronic, that remedy which is able to stimulate another artificially produced disease as similar as possible; and the former will be healed - Similia Similibus—likes with likes".[2]

So evolved the basis of homeopathy, "Similia Similibus Curantur", let likes be treated with likes. From this central dogma of homeopathy, several corrollaries were produced by Hahnemann and his immediate followers.

Homeopathic Principles

The following is a short list of Hahnemann's main guidelines for homeopathy.[3]

1) Origin of Disease

Hahnemann believed that the diseases of man are not brought on by any material substance or disease-matter, but that they are solely spirit-like derangemants of the spirit-like power (the vital principle) that gives life to the human body.

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2) Curing the Disease

He explains that a cure can only take place by the reaction of the vital force against the correct remedy that has been ingested. The patient's recovery will be certain and rapid depending only upon the strength with which the vital force still prevails in the patient.

3) Physcian's Goal

The only task of the physican is to cure rapidly, gently, permanently, not to construct theoretical systems, and not to attempt to explain phenomena.

4) Practical Definition of Disease

The disease consists only of the totality of its symptoms (ie. cure symptoms, cure disease).

5) Duties of the Homeopath

a) Investigation of the disease and determining the totality of its symptoms.

b) Investigation of the effect of the medicines (tested on a healthy body, usually the physician himself).

c) The appropriate application of the correct medicine.

6) Homeopathic Therapeutics

Only one, single, simple medicine should be given to the patient at one time and in appropriate amounts. (It should be noted here that one of the mainstays of homeopathic therapeutics is the concept of "potentiation". This involves many serial dilutions of the medicine to finally obtain the "infinitesimal dose" or "microdose". This microdose is often in the order of 10^{-60} or even 10^{-402} which, according to Avagadro's Law, contains essentially no molecules of the original medicine).

7) Two Types of Diseases

Chronic diseases take a longer time to cure than acute ones because they are of a more complicated nature and have had a chance to root in the patient for a longer time.

8) Explanation of the Homeopathic Cure

As stated before, the natural disease is thought of as a spirit-like agent disturbing the normal course of life which is in turn controlled by a similar spirit-like vital force. The homeopathic administration of a medicine which produces similar symptoms (ie. alters the vital force in a similar manner) to the natural disease is, in effect, a way of masking or exceeds the disease in energy. Then, the influence of the natural disease on the life principle is lost during the action of this stronger, similar, artificial disease. Thus the disturbance is no longer caused by the natural disease which can no longer sustain itself and disappears. The symptoms caused by the medicine (artificial disease) also subside after a period and the patient is cured.

(This traditional explanation is vague and today's homeopaths deviate from this. They believe that the method of the homeopathic cure of similar medicines relies on awakening or stimulating the body's own defense mechanisms against the disease.)

9) Incurable Disease

A disease is incurable if the vital force of the victim has been weakened beyond the point of return.

With these ideas, Hahnemann and his followers set out to convince the allopaths that the methods they were using at the time produced more adverse side-effects than the desired treatment-effects.

RECENT HISTORY AND PRESENT STATUS

Homeopathy slowly spread from its origins in Germany to the rest of the European countries and eventually to Russia, Great Britain, Cuba, Mexico, India, and the United States of America, among others. Canada also was touched by its influence through the U.S.A. It enjoyed so much popularity in North America that in the late 1960s, one-sixth of all medical practitioners in the United States followed this doctrine.[3] Homeopathy was even more successful in India, where it ws introduced first by an anonymous German geologist and subsequently by a London missionary, an English doctor by the name of Mullins. It was adopted very quickly in this country which was probably due to the rarity of well-educated orthodox doctors. All over the world, many hospitals, institutions and societies were started under the name of homeopathy. However, even at its peak, homeopathy never dominated over allopathy and soon, it started to decline in popularity. This degeneration can be attributed mostly to advances in orthodox medicine which promoted better hygiene, prevention of diseases, and research into understanding of pathophysiology. Currently homeopathy is relatively unpopular and is restricted to a handful of medical colleges, hospitals, and institutions. These are usually found only in countries where this doctrine had once attained widespread acceptance.[4]

ANALYSIS OF RISE AND FALL OF HOMEOPATHY USING T.S. KUHN'S PARADIGMS

Kuhn's Paradigm Concept

A useful method of analyzing the course of homeopathy in medical history is that of T.S. Kuhn's. He devised the concept of a paradigm to explain the process by which scientific research progresses. A paradigm is "... the set of assumptions and methods that guide research in a given domain for a particular period of time".[5] Dominant paradigms are successful because they share two essential characteristics: first, the paradigm produced an achievement that was sufficiently unprecedented to attract an enduring group of adherents away from competing modes of scientific activity and second, it was suficiently open-ended to provide a variety of problems for the redefined group of practitioners to ponder and solve. Kuhn explains that an established paradigm can evolve into a modified one or a totally new one if a crisis takes place. A crisis occurs when a

number of anomalies have accumulated so that the old paradigm cannot explain the new facts satisfactorily. Thus, a modified or new paradigm supercedes the old one.

Explanation of the Emergence and Decline of Homeopathy

These observations of Kuhn can be used to explain the emergence of homeopathy in the scientific realm of medicine. Before the rise of homeopathy, medicine was guided by the paradigm of "heroic practice" which involved antagonistic processes such as blood-letting, blistering, purging, and sweating. The ignorance of the patients as well as the physicians allowed such harmful and barbaric practices to flourish. However this paradigm failed in providing a satisfactory explanation and logical cures for diseases, and thus a crisis was at hand. This might have been realized eventually, but the credit must be given to Hahnemann who first saw this crisis and formulated his own paradigm, that of homeopathy.

Kuhn's paradigms also suggest reasons for the eventual decline of homeopathy. Hahnemann had the insight to see that the "heroic practice" paradigm violated the underlying principle of all medicine, that of *primum nil nocere* or "first do no harm". This means that whatever cure is applied, the most important property of that cure is that it should inflict no further injury to the patient. As a result of Hahnemann's arguments, the orthodox practitioners were grudgingly becoming aware of its inadequacies and so modified their paradigm. This improved version based its therapeutics on knowledge gained from medical research or from clinical experience. Now, the stage was set with the two competing paradigms, the homeopathic one and the orthodox or allopathic one. Kuhn states that the dominant one must be able to boast unprecedented achievements and supply a multitude of problems for the scientific community to solve. As it will be shown, homeopathy was lacking in both of these areas.

Ruthaven Mitchell explains in his book on homeopathy that when Napoleon's shattered army returned from Moscow in 1812, they brought back with them the deadly plague of typhus. He claims that homeopathic cures proved superior to allopahic cures in the treatment of this plague.[6] Incidents such as these allowed homeopathy to seem successful, but this was only relative to the old "heroic practice" paradigm. The new orthodox paradigm did not have such cures to offer, but it was not long before it obtained its own healing powers (eg. antibiotics and surgery). The second condition of a dominant paradigm is where homeopathy failed miserably. The followers of homeopathy were not encouraged to investigate how medicines healed. This left them to investigate what medicines healed which diseases by simple trial and error. However, the orthodox practitioners could conduct research into many different problems of how drugs worked and how the body itself functioned. In this way, allopathy fulfilled the two conditions and became the dominant paradigm to reign over the medical profession.

Another possible reason for the demise of homeopathy is its intrinsic spiritual quality. It was hard to argue scientifically in support of the homeopathic principle of disturbances in the vital force of an organism. This left room for doubt and indeed required a certain amount of faith from its followers. Homeopathic historians often quote incidences where homeopathy had performed unbelievable feats of medical healing. For example, it is said that Hahnemann, "...contracted cholera and cured himself in six hours with homeopathic ipecacuanha"[7] and that a certain famous London doctor by the name of Hervey Quin, "...became fatally ill as a young man, but even as death seemed certain, ... Dr. Romani (a homeopath) ... prescribed five powders and the youth made a rapid and uneventful recovery".[8] Although isolated events such as these may have occurred, the "religious" quality and its lack of explanatory evidence prevented homeopathy from being accepted universally.

Reasons for the Persistence of Homeopathy

Why then did homeopathy enjoy the partial success that it did and why is it still surviving today? It can be safely stated that within a population of followers of a system, there will always be a small group of members who will not be content. If someone offers this population an alternate and seemingly perfect system, which claims to relieve their anxieties, and also offers some superficial proof in support, a few of this small group will be easily convinced to convert. Such was the situation with many of the allopathic converts to homeopathy.[9] Allopathic treatments of that time failed to heal and often did more harm than good. Hahnemann offered a holistic doctrine where the patient was cured gently, rapidly, and completely. This attracted and converted some allopaths to homeopathy. Homeopathy probably gained most of its followers in this way because most homeopaths, then and now, were once allopaths and very few have practised homeopathy from the onset of their medical career.

However, it is unfair to say that this is the sole reason for the existence of this doctrine. Some of the experiments and trials that have been done to date suggest that homeopathy could be of value in curing diseased crops and animals and even as a supplement for allopathic treatment of humans (discussed below).

THE MEDICAL DEBATE

Homeopathy in Great Britain

A very good example of the persistence of homeopathy can be seen in Great Britain. Here, homeopathy is very well organized and there are many homeopathic institutions still remaining including a million-dollar pharmaceutical company which mass-produces homeopathic medicines. Some of the most valid discussions and arguments concerning this controversy can be found in many of the British medical journals.

Clinical Trials in Arthritis Treatment

One such debate involves a comparision of allopathic and homeopathic treatments of rheumatoid (or osteo-) arthritis. In 1978, a preliminary clinical trial was done to test the value of homeopathic remedies in the management of rheumatoid arthritis.[10] This study began with 195 patients and was conducted for one year. Despite the large sample and the length of the trial, several intrinsic faults in the methodology did not allow any concrete conclusions to be made. As it was only a preliminary study, the researchers acknowledged its deficiencies but, they maintained that there was enough evidence in the results to indicate that patients receiving homeopathy responded better statistically (based on articular index, limbering-up time, grip strength in each hand, and pain relief) than those that received salycilates which was the allopathic treatment. They claimed that the undertaking of another, more controlled study was justified.

Allopaths wasted no time in criticizing this study and rightly argued that the data could not be used to make any inferences about the efficacy of homeopathy in arthritis treatment.[11] They concentrated on two components of the trial. First, the patients receiving homeopathy were allowed to continue their previous orthodox anti-inflammatory drugs whereas the patients receiving salycilate had to discontinue theirs. Thus, any improvements shown by the homeopathically-treated patients could be attributed to the continuation of their orthodox drugs and any lack of improvement in the salicylate group could be due to the continuation of the same drugs. Secondly, each of the two groups of patients were treated by different doctors which allowed bias to enter the study.

In reply, the original researchers conducted a more controlled, double-blind, cross-over therapeutic trial.[12] This trial had forty-six patients only and lasted three months only, but its technique showed a great improvement over its predecessor. The carefully selected arthritic patients were divided randomly into two groups; one group was given conventional therapy of non-steroidal anti-inflammatory drugs, supplemented with homeopathic remedies prescribed according to the patient's individual symptoms; the other was given the same conventional therapy but the homeopathic remedies were replaced by placebos. The results again indicated that patients receiving the homeopathic remedies as a supplement to their orthodox treatments showed a greater improvement than the control group. In addition, the researchers emphasized that homeopathic remedies were much safer (ie. less side-effects) than orthodox, second-line drugs commonly used in the treatment of rheumatoid arthritis. This study has not received much criticism.

Subsequently, another double-blind crossover trial comparing Rhus tox. 6X (a homeopathic remedy) with fenoprofen (an allopathic drug) in the treatment of osteoarthritis was undertaken.[13] Again, the research technique was good, but the results contradicted the previous trial as it showed fenoprofen was beneficial to the patients and Rhus tox. 6X did not differ significantly from placebo.

The homeopaths disregarded this study for two reasons. First, the trial lasted two weeks only, but chronic cases such as osteoarthritis require some time to react to homeopathic remedies. Second, one of the basic principles of homeopathy states that the medicine should be prescribed specifically and individually to each patient as his unique symptoms dictate; but in this study, Rhus tox. 6X was given to all patients regardless of their varying symptoms.

The Allopathic Side

Small arguments such as this have been ongoing for several centuries but the same points seem to be discussed everytime. The allopaths point out that homeopathic nostrums are at times administered in very high dilutions. Some of these dilutions are often greater than 10^{-24} which means than, according to Avagadro's constant, it is by chance alone if there is one molecule of the active substance in it or not. Essentially, none of the original medicine is present in these "high potency microdoses". Allopaths accuse homeopathic clinical trials of not withstanding scientific scrutiny (in terms of inadequate controls, not performed under double-blind conditions, or other experimental design defects). Also, the orthodox practitioners state that the empirical form of medicine practised by the homeopaths offers no logical explanation that can be verified satisfactorily. Finally, homeopathic principles dictate that detailed study of the pathology of diseases and how they affect normal physiological processes is irrelevant to the duty of a physician. However, medical research, conducted by allopaths, into pathophysiology has uncovered valuable knowledge that has been used to scientifically deduce treatments and cures. For example, biochemical research into diabetes has led to the use of the hormone insulin in its treatment. Even if new knowledge cannot be used immediately, it contributes to a growing knowledge bank which can be utilized in the future.

The Homeopathic Side

Homeopaths accuse conventional medicine of not having a concise set of principles dictating its therapeutic methodology whereas homeopathy does (see *Homeopathic Principles* section). Thus, they argue that homeopathy is the only truly scientific medical doctrine. They also show that the nature of homeopathic remedies (simple substances often in low concentrations) allow them to be taken very safely during pregnancy and breast-feeding, to be nonaddictive, and to be less likely to produce adverse side-effects; the powerful, concentrated medicines of orthodox doctors can do more harm than good if administered improperly and sometimes even when administered properly. Allopathic testing of homeopathic therapeutics have been criticized by homeopaths for the lack of individual attention given to each patient, for inaccurate administration of the remedies, and for not providing enough treatment time. Finally, they point out areas in which allopathic treatments have been less than satisfactory, areas such as arthritis, common cold, and cancer. In

fact, it is in these areas on which homeopaths concentrate because there is a better chance of obtaining relatively successful results in favor of homeopathy when clinical trials are performed. Also, most patients received by homeopaths usually have ailments in these areas and are seeking alternate methods since allopathy has failed them.

DEFINITION OF A CONTROLLED CLINICAL TRIAL

A controlled clinical trial is one that allows a true comparison of two or more different treatments of similar patients by controlling or balancing all variables. There are certain minimum criteria to be met before a clinical trial can be described as effectively controlled:[14]

1. The therapeutic objective must be identified clearly. This allows the experiment to be designed adequately, with its underlying goal as its foundation.

2. The criteria for the measurement of improvement in the patient's condition should be predetermined.

3. Using these criteria, the threshold for success should also be preset so that it cannot be compromised during the course of the trial.

4. Finally, it is necessary to have an understanding of the disease to be studied and its clinical behaviour, as these are usually the sole basis on which the treatment groups are equally divided.

These guidelines describe the ideal clinical trial which is strived for, but they are often compromised to make the trial practical.

CONTROL OF BIAS

In the past, a common problem that plagued both allopathic and homeopathic clinical trials is the placebo effect. A placebo is normally used to keep hidden the identity of the drug given to a particular patient. However, even if one applies a "pure" or "true" placebo (one that has virtually no effects compared to the real drugs) bias can be introduced. Real drugs will invariably produce some symptoms or signs that are easily recognized by the experienced patient or physician and thus, a "true" placebo may give itself away by not producing these signals. In an attempt to control this type of bias (and also other types of biases), the double-blind technique is used. Here, every precaution is taken to insure that both the subject and investigator do not know whether a placebo or an active drug is being taken until the trial is completed.

CONCLUSION

The solution to the homeopathy versus allopathy controversy may lie in the simple-minded idea of co-operation. What is suggested is that a team of researchers, consisting of both homeopaths and allopaths, supervise a clinical trial in a particular area such as arthritis or cancer. The terms of the trial would have to be mutually satisfactory but all the elements of a controlled clinical trial (as stated before) should be included, and every attempt should be made to eliminate bias of any sort. Such a study could not be rejected by either doctrine and thus seems to be the best line of action to resolve this disagreement.

Some factions of orthodox medicine believe that homeopathy has absolutely no role to play in modern medicine whereas others believe that it does have some value. It is my opinion that homeopathy has already played its most important role in medical history by opening the eyes of the "heroic practice" followers and inadvertantly setting them on the right track. I believe also that present-day homeopathy does indeed have some practical value that should be filtered and absorbed by modern medicine. For example, it has already been mentioned that conventional medicine is at a loss in certain areas.

Homeopathy and other forms of holistic medicines can be of help here in at least soothing the patient's mind, if not his body, with their gentle and harmless medicines. Indeed, today's physicians are faced with more and more barriers to progress as numerous "twentieth-century" diseases have sprung up. In light of this, help from any "fringe medicine" should be accepted gratefully if their methods prove to be successful. It seems that the British Medical Association has also realized this and has recently undertaken the task of evaluating various forms of "fringe medicines" (including homeopathy) and how they might be of use as supplements to orthodox therapeutics (as stated above). This is definitely a step in the right direction. □

ACKNOWLEDGEMENTS

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MORALITY AND MEDICINE

Three distinguished scholars and health educators will examine questions of medical ethics and morals during the 1985 Dorothy J. Killam Lecture series at Dalhousie University in October.

October 10 - Dr. Robert Evans, economics professor, University of British Columbia speaks on "By What Warrant? Ethical Bases of Intervention in Medicine and Economics".

October 17 - Dr. Vivian Rakoff, chairman, psychiatry department, University of Toronto, speaks on "High Technology, Will, Ethics and Medicine".

October 24 - Dr. Edmund Pellegrino, director, Kennedy Institute of Ethics, Georgetown University, Washington, D.C., speaks on "The Anatomy of Clinical Ethics".

All lectures are open to the public, free of charge and will be held in the Rebecca Cohn Auditorium, beginning each evening at 8 p.m.

Sarcoidosis: An Historic Perspective

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It is of some interest that the first cases which we would now consider as sarcoidosis recorded in medical literature were cases of diseases of the skin. *Jonathan Hutchinson* described what was likely sarcoidosis of the skin in 1877 in his report of a case of "anomalous disease of the skin of the fingers (papillary psoriasis)". Twelve years later, *Ernest Besnier*, a French dermatologist, described a 34 year old male with skin lesions (lupus pernio) and polyarthritis which was probably sarcoidosis. This patient also had swelling of the nose, cheeks and ear lobes. (Pernio is a Latin word for chilblain.) In 1892 *Tenneson* described "epitheloid tubercules" in a case of lupus pernio. A tuberculous etiology was suggested by *Hutchinson* in 1898 when he reported the case of Mrs. Mortimer, a middle-aged patient of his. "Mortimer's Malady" later became the common English language description for this dermatitis.

Figure 1

Figure 2

1. Sir Jonathan Hutchinson, surgeon, syphilologist and opthalmologist.

2. *Cesar Boeck* whose name is still attached to the word sarcoidosis.

The name of *Cesar Boeck*, a Norwegian dermatologist, is still associated with sarcoidosis as it was he who in 1899 described the histology in a similar case, calling it "multiple sarkoid of the skin". The association of sarcoidosis with *erythema induratum* was suggested in 1919 by *Sweitzer* and with *erythema nodosum* by *Morgensen* in 1933.

The first description of lung changes in 1921 by *Kuzoritsky* followed the introduction of chest radiographs, and *Valenti* described the chest X-ray changes in greater detail in 1928.

Multiple organ involvement had been suggested in 1904 by the Czeck dermatologist, *Karl Dreihich*. Involvement of the eye was reported with iritis and optic neuritis and the syndrome of uveo-parotid fever became a part of the syndrome of sarcoidoisis. 1941 was an important year in the history of sarcoidosis because of four significant reports: 1) an association with uveitis by *Guyton* and *Woods*, 2) the description of asteroid bodies in sarcoid granulomas by *Schaumann*, 3) the association with hypercalcemia *(Schuaback* in Germany and *van Crevald* in Switzerland); and 4) *Kveim's* report of "a new specific cutaneous reaction in Boeck's sarcoid". By injecting a lymph node suspension from a patient with sarcoidosis, *Kveim* had produced a skin nodule in 12 of 13 similar patients. Biopsied after four weeks, this skin nodule showed non-caseating epitheloid granulomas with giant cells. Since that time such suspensions have been shown to produce a positive reaction in 85-90% of patients with active sarcoidosis especially those with bilateral hilar lymphadenopathy. The test is now regarded as variably selective rather than specific for active sarcoidosis.

Between 1942 and 1944, other important contributions to knowledge of the disease included the report of electrophoretic changes in serum proteins *(Fisher* and *Davis)*, an association with splenomegaly *(Malgras* and *Pasquel)*, the value of closed liver biopsy in diagnosis *(van Beck* and *Haex)* and finally, the finding of posterior pituitary infiltration as part of the syndrome *(Glass* and *Davis)*.

In 1949, the value of scalene node biopsy was reported by *Daniels*. In 1951, *Simkins* described a case of complete heart block with sarcoidosis and a year later two reports *(Davis* and *Crotty* as well as *Myers, et. al.)* stressed the importance of polyarthritis as a manifestation of sarcoidosis.

The etiology is still a mystery. *Cummings* and *Hudgins* in 1958 suggested a relationship between sarcoidosis and exposure to pine pollen. Since that time, manifestations of the disease have led to the theory that the disease is an immunologic reaction to some unknown antigen or antigens. □

References are available from the author.

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Sarcoidosis of The Skin

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INTRODUCTION

Sarcoidosis is a systemic inflammatory granulomatous disease of unknown aetiology. It may affect a variety of internal organs and may or may not be associated with cutaneous lesions. In the past, any noncaseating granulomatous process involving the skin was diagnosed as sarcoidosis. However, as clinical and laboratory analysis improved, many of these processes were found to be related to foreign body reactions such as talc or zirconium, tuberculosis, fungal infections, berylliosis, leprosy, syphillis, and even some types of lymphoma. As these processes were removed from the sarcoid spectrum, however, a uniform characteristic process of cutaneous involvement remained.

As with systemic sarcoidosis, sarcoidosis of the skin may be a great imitator and masquerade in a variety of clinical presentations.

CLASSIFICATION OF CUTANEOUS DISEASE

Cutaneous Sarcoidosis is seen in approximately 20-35% of patients with systemic disease.[1] Patients with cutaneous disease have either specific or non-specific lesions. Specific lesions refer to those which histologically reveal a noncaseating granuloma. These are seen in approximately 10-35% of patients and are usually associated with an unfavorable prognosis.[1] Non-specific lesions are referred to as lesions in which the biopsy fails to reveal granuloma formation, but which are seen in association with sarcoidosis elsewhere. The classic example of this is erythema nodosum which histologically shows a panniculitis without showing any specific sarcoidal pathology. Erythema nodosum may be seen as a manifestation of diseases other than sarcoid and for that reason is also referred to as a non-specific lesion.

Non-Specific Manifestations

The early stage of sarcoidosis is frequently associated with erythema nodosum, especially in young females. It is present in approximately 17% of patients with systemic disease.[2] This usually presents as subcutaneous erythematous tender nodules frequently seen on the shin regions. Young individuals are more frequently affected with fever, fatigue, polyarthralgia, uveitis, an elevated sedimentation rate, and bilateral hilar lymphadenopathy (Lofgren's Syndrome). There is no therapy indicated unless they become quite painful. Erythema nodosum usually tends to spontaneously resolve in 80% of those affected, within six months.[3] Frequently, as these lesions resolve, they may become ecchymotic and this presents clinically as focal areas of bruising on the anterior tibial surface. Immune complexes and their deposition in the subcutaneous tissue has been suggested by some authors as a cause for their cutaneous manifestation.

Specific Manifestations

Specific manifestations refer to those cutaneous lesions which show a typical sarcoid granuloma histologically. (Fig. 1). The commonest cutaneous expressions are a maculopapular eruption (Fig. 2), plaques (Fig. 3), cutaneous and sub-cutaneous nodules, and diffuse erythematous infiltration of the skin. One may clinically see different types of lesions present at the same time.

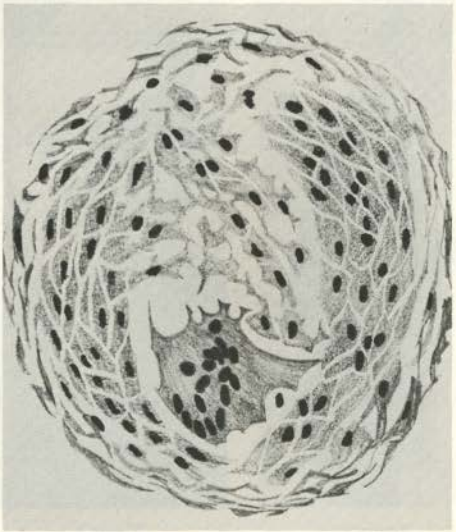

Figure 1. Sarcoidal granuloma showing a collection of epitheloid cells with a Langhans giant cell and a sparse lymphocytic infiltrate. (from Ackerman A B, Histologic Diagnosis of Inflammatory Skin Diseases).

The papular lesion may range in size from 1 to 10 mm and may coalesce to form plaques which occassionally may clear centrally to become annular in nature. (Fig. 4). The color may initially be erythematous or violaceous and then gradually assume a yellow-to-brown hue.

The nodular lesions are larger than papules and, like the papular lesions, may be seen predominantly on the face, especially about the eyelids and ala nasi as well as on the

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Figure 2. Forearm showing well defined papules of varying size.

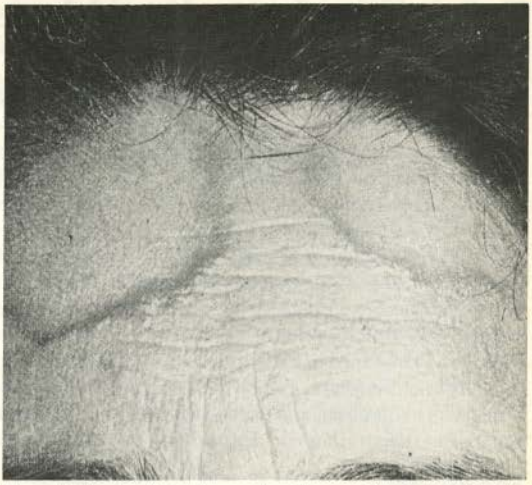

Figure 4. Forehead showing annular plaques with an elevated border and central clearing.

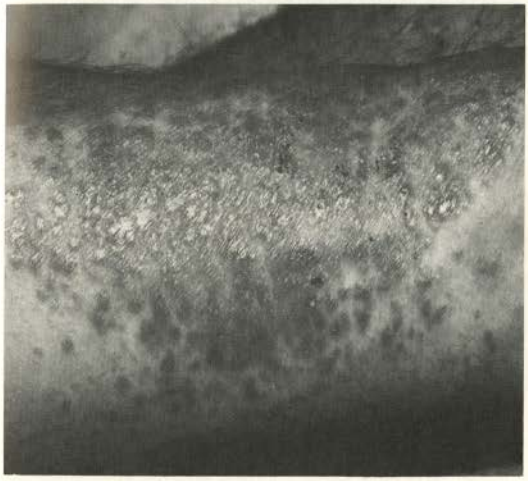

Figure 3. Forearm showing discrete papules, many of which are forming confluent plaques.

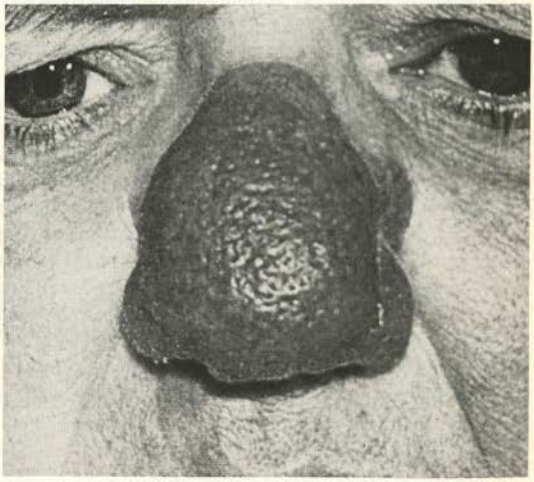

Figure 5. Lupus Pernio of the nose.

posterior aspect of the neck and shoulder and the extensor aspects of the extremities.

Lupus pernio is a distinctive cutaneous plaque-type lesion occurring acrally, particularly on the cheeks, ears, dorsum of the hands, and may characteristically affect the nose, (Fig. 5) which may be bulbous with dilated venules. Nasal mucosal involvement may also occur. Lupus pernio may also be seen in association with bone cysts, particularly of the digits of the hands and feet. This form of sarcoidosis may resolve leaving atrophy and, in some cases, may be the most disfiguring form of cutaneous sarcoidosis.

Other types of sarcoidosis include sub-cutaneous, erythrodermic, verrucous, ichthyosiform, psoriasiform, ulcerative, etc. It may present as scarring alopecia of the scalp or may even mimic lupus erythematosus-like lesions. Among the miscellaneous and rather uncommon lesions, are scar sarcoidosis in which the sarcoid granulomas appear in scars.

PATHOLOGY

The essential pathologic feature in sarcoidosis is the so-called hard tubercle (Fig. 1) which does not show central caseation, although occasionally fibrinoid necrosis may occur. These tubercles or granulomas are discrete aggregates of epithelioid cells and occasional Langhans giant cells which may be surrounded by lymphocytes, macrophages, and fibroblasts. Inclusion bodies are frequently observed within giant cells but this finding is non-specific. Three types of inclusion bodies have been described: the Schaumann body which consists of calcium carbonate, phosphate, and iron; the Asteroid body which is made up of lipoprotein; and the residual body which probably represents lipomucoprotein granules.

The Kveim-Siltzbach Test

The Kveim-Siltzbach Test is an immunological event peculiar to and diagnostically significant for sarcoidosis, but is not usually performed in Canada. The test involves injecting an intradermal suspension of heat-sterilized sarcoid tissue, waiting a period of one to six weeks, and then taking a biopsy of the inoculated site. The presence of typical sarcoid granulomas within the dermis is a positive Kveim test. Although the test has been of great value in the past, some controversy persists as to its sensitivity and specificity. It is possible that its questionnable diagnostic efficacy stems from poorly prepared antigen.[4] Systemic steroids may suppress the reaction and negative test results may be obtained in patients who have had the disease for more than five years. The Kveim Skin Test is reportedly negative in the absence of hilar adenopathy and is thus of little value in extrathoracic disease.[5]

PROGNOSIS

Sarcoidosis, in general, becomes chronic in only a very small percentage of patients. The prognosis is stated to be better when patients present with skin lesions rather than pulmonary symptoms. Negros tend to have a more chronic progressive type of disease.[6] A favourable outcome is also seen in young persons, especially women.[1] Chronic forms of the disease are seen more commonly in older individuals and those whose disease has an insidious onset and tends to be progressive. The disease tends to run a more chronic course in patients with specific cutaneous disease. The papular and nodular lesions clear in 6 months to 3 years or more. Plaque lesions tend to be more persistent and lupus pernio is the most chronic and resistent form. Patients with erythema nodosum tend to have a very acute and therefore relatively "benign" form of the disease. The prognosis for any individual patient, however, is always unpredictable.

TREATMENT

Since the cause of sarcoidosis remains unknown, therapy in general should be aimed at preventing progression, promoting regression, or alleviating symptoms with the creation of minimal side effects. In 70-80% of patients with acute sarcoidosis, the disease resolves spontaneously and therapy is seldom indicated.[1] Even though systemic steroids are the main-stay of therapy, other treatments such as intralesional steroids for skin lesions, steroid eye drops for acute uveitis, and indomethacin or other nonsteroidal anti-inflammatories for erythema nodosum and arthritis have a role in therapy.

Anti-malarials such as chloroquine may have an effect upon cutaneous disease although relapses apparently are common on discontinuation of therapy.[7] Allopurinol has been known to be of some value in treating patients with chronic skin and mucosal lesions but relapses may also occur when these are discontinued.[8] Methotrexate may be effective in cutaneous and ocular disease.[9] Other immunosuppresives such as immuran, and chlorambucil, have also been employed as cortieosteroid-sparing agents in advanced or progressive disease which has been unresponsive to steroid therapy. More recently, oral retinoids have been used in treating the cutaneous manifestations of this disease.[10] In general, sarcoidosis of the skin usually remains untreated. Only if sarcoidosis becomes a disfiguring condition, particularly of exposed surfaces, does one intervene with the therapies mentioned.

□

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"Examples would indeed be excellent things were not people so modest that none will set them, and so vain that none will follow them."

— Julius Charles Hare

Sarcoidosis: Neurological Manifestations

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Halifax, N.S.

Neurological involvement in sarcoid disease has become an increasingly recognized entity. The incidence, ranging from 3.5-17%, indicates that many patients with this disorder will at some time exhibit evidence of such a complication. These patients usually have other stigmata of sarcoid; however, neurological dysfunction may be the sole presenting feature of this disease.

Any portion of the neuraxis may be affected the commonest however being that of cranial and/or peripheral nerve involvement. Such involvement, conveying a more favourable prognosis, usually develops during the chronic stage of the disease. Central nervous system involvement rather tends to develop early and conveys with it a poorer outlook.

The course of such involvement is unpredictable i.e., may be transient, recurrent or chronic. However, overall, although exhibiting remissions and exacerbations, not infrequently this aspect of the disorder is of a relatively benign nature.

Steroids have been the main thrust of therapy but the response is exceedingly variable. Large doses may be useful in inducing remissions. Usually there is a response with cranial and peripheral nerve involvement. The parenchymal lesions of the central nervous system however are much more resistent to therapy and much less likely to exhibit spontaneous remissions.

The following constitute the principle manifestations of involvement of various portions of the neuraxis.

CENTRAL NERVOUS SYSTEM

Sarcoid takes the form of granulomatous infiltration of the meninges and underlying parenchyma there being a marked predilection for this to occur in the region of the base of the brain most particularly in the vicinity of the optic nerves, chiasm, hypothalmus and pituitary gland particularly its posterior aspect. This leads to visual failure and/or endocrine dysfunction primarily diabetes insipidus. It is to be recalled that not every instance of visual dysfunction need be on such a basis as this disorder also preferentially involves the anterior portions of the eye.

Cortical involvement may be manifested by features of confusion, personality change, dementia, alteration of the level of consciousness, seizure activity and motor and sensory dysfunction. A similar encephalopathic picture may also arise secondary to the hypercalcemia which often occurs in this disorder or may represent an adverse effect of steroid therapy either directly or as a result of rendering the patient increasingly susceptible to the occurrence of opportunistic infections. Occasionally focal signs, including seizure activity, predominate due to the presence of a solitary granulomatous mass which behaves as does any space occupying lesion.

Meningeal involvement, productive of stiff neck, may also obstruct spinal fluid pathways giving rise to hydrocephalus leading to the usual features of raised intracranial pressure. Such involvement may be reflected by changes in the spinal fluid ie., increased protein, increased cells of the lymphocytic variety usually being modest in nature ie., 20-200, and either normal or very frequently a decreased sugar. Such alterations are identical to those which occur in other forms of chronic meningitis i.e., tuberculosis or cryptococcosus the difference being that in sarcoidosis the stain will reveal no organisms and the culture will be sterile.

CRANIAL AND PERIPHERAL NERVOUS SYSTEM

This is the commonest manifestation of sarcoidosis facial neuropathy being the most frequent manifestation of single peripheral nerve involvement. The facial dysfunction may be unilateral or bilateral. It is of sudden onset and usually after a variable time sequence spontaneously resolves though residual impairment may be present i.e., contracture or abnormal movements. It has often been attributed to involvement of the parotid gland by granuloma however such a cause-effect relationship is by no means invariable. Indeed facial nerve impairment may occur without, before or after such parotid involvement. Any cranial nerve may be affected and one may see multiple nerves involved at the same or different times.

Peripheral nerve involvement occurs as a result of granulomatous infiltration imbedded in reactive connective tissue scattered along the course of the nerves. This may result in a subacute or chronic polyneuropathy usually of an asymmetrical type. Neuropathy may occur in isolation as involvement of a single nerve or in combination with involvement of other peripheral or cranial nerves the picture being that of asymmetrical mononeuritis multiplex. The clinical features consist of weakness, atrophy, suppressed reflexes and impaired sensation in the distribution

Continued on page 92.

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Sarcoidosis and Tuberculosis

Helen M. Holden,* M.D.,

Kentville, N.S.

Today it is generally accepted that sarcoidosis and tuberculosis are two distinct diseases. In the past, sarcoidosis was considered by some to represent a special phase of tuberculosis and the term noncaseating tuberculosis was applied to the condition. As the result of postmortem studies, it was also realized that both could exist together.[1] Furthermore, it was stated that tuberculosis was not just an incidental complication when it occurred in the course of sarcoidosis but that the potentiality of its development was inherent in the nature of sarcoidosis.[2]

After World War II and especially following the advent of effective treatment for tuberculosis, it became clear that sarcoidosis and tuberculosis are of different etiology. Therefore, when tuberculosis develops in a patient with sarcoidosis, it represents a new disease and not a step in the evolution of the sarcoidosis itself.

Sarcoidosis and tuberculosis can precede or follow one another by short or long intervals in the same individual, or both can be active at the same time. Israel and Sones reviewed a series of 360 patients with sarcoidosis: two had tuberculosis first, thirteen developed tuberculosis after sarcoidosis and four were still in the active phase of sarcoidosis when tuberculosis was discovered. All 13 reacted to tuberculin during the active phase of tuberculosis although ten had been nonreactors when sarcoidosis was diagnosed and eight reverted to the anergic state on recovery from tuberculosis.[3].

Chusid *et al.* also point out the tendency towards tuberculin anergy or hypergy in sarcoidosis in their series of 350 sarcoidosis cases in whom only 49 (14%) were reactors. Eight developed active tuberculosis and reacted to 10 units of tuberculin, including six who had been non reactors and two who were weak reactors before the onset of tuberculosis. This illustrates the fact that the development of a positive tuberculin test during sarcoidosis should alert one to possible infection with tuberculosis rather than lulling one into thinking that the reaction signals recovery from sarcoidosis.[4] The significance of such a change is even greater today with the lower incidence of tuberculin reactivity and of tuberculosis.

Scadding reported a series of 275 patients with sarcoidosis and there were fifteen who had tuberculosis at varying intervals before sarcoidosis, inculding four who developed sarcoidosis during the active phase of tuberculosis. In eighteen cases tubercle bacilli were isolated when the findings were considered typical of sarcoidosis, not tuberculosis. In addition five patients showed a transition from sarcoidosis to tuberculosis after intervals of two to twelve years.[6]

With the declining incidence of tuberculosis, chiefly due to the advent of modern antituberculosis chemotherapy, there has been no corresponding drop in the number of sarcoidosis cases. Indeed, it has been noted that sarcoidosis occurs in the wake of tuberculosis as in the West Indian population of Great Britain and among South African natives.[5]

At times the differential diagnosis between pulmonary tuberculosis and the pulmonary form of sarcoidosis may be difficult, especially if there is a question of both being present at the same time. If tubercle bacilli are found, tuberculosis is present. Almost every patient with tuberculosis reacts to tuberculin. The patient with advanced tuberculosis is usually quite ill but responds dramatically to appropriate chemotherapy. There is no response to antituberculous drugs in sarcoidosis.

In contrast to tuberculosis, a patient may be asymptomatic in spite of advanced sarcoidosis. At the same time there are sometimes acute manifestations of sarcoidosis such as erythema nodosum and polyarthritis that are not characteristic of tuberculosis. The presence of uveitis and skin lesions may be of help in distinguishing the two conditions. Spontaneous recovery is common in sarcoidosis; less so in tuberculosis. There is no evidence of person to person spread in sarcoidosis, in contrast to the infectious nature of tuberculosis. Furthermore, tuberculosis tends to involve the socially disadvantaged groups in society while sarcoidosis occurs in all with equal frequency.

As to the radiological findings, a diffuse bilateral miliary infiltration can occur in both conditions. Apart from this, the typical patient with sarcoidosis has a bilateral infiltration and the well known hilar and mediastinal adenopathy is a fairly frequent finding.

In tuberculosis, the spread tends to be apicalcaudal, and in sarcoidosis caudoapical. A burnt out case of sarcoidosis may present with apical disease, making differentiation more difficult. Teirstein and Siltzbach report a series of 616 patients in whom 54 had upper lung field disease simulating tuberculosis.[7] Cavitation is much more common in tuberculosis.

Between January 1, 1944 and December 31, 1974, 5029 patients with tuberculosis were admitted to the Nova Scotia Sanatorium for the first time. During the same 31 year period, 102 patients were admitted in whom the diagnosis

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proved to be sarcoidosis. Six of these patients were referred to the Sanatorium as miliary tuberculosis but sarcoidosis proved to be the diagnosis.[8] All six made a satisfactory recovery. However, one female patient, then 17 years of age, was readmitted sixteen years later at the age of 33 years with advanced cavitary tuberculosis and required a left upper lobectomy.

There was a second patient in whom sarcoidosis preceded tuberculosis — a 25 year old woman in whom lung and pleural biopsies were compatible with sarcoidosis but she was readmitted fifteen months later with advanced tuberculosis and required surgery.

There were six other patients with both conditions making a total of eight. Three had tuberculosis in the remote past. One was a 48 year old woman who was admitted with a diffuse pulmonary mottling that proved due to sarcoidosis. She had been treated for tuberculosis of the elbow at the age of 7 years.

Another was a 40 year old woman, known to have had primary infection calcified tuberculosis who was admitted with marked hilar adenopathy due to sarcoidosis.

A third woman was treated for cavitary tuberculosis at the age of 20 years. Ten years later at the age of 30 years, she was admitted with marked mediastinal adenopathy and pathological examination of resected glands established the diagnosis of sarcoidosis.

The sixth patient with both conditions was admitted at the age of 23 and had already undergone four biopsies due to recurrent cervical and hilar adenopathy over a 3 year period. The first biopsy was considered diagnostic of sarcoidosis, the second of tuberculosis and she was treated accordingly. The third was considered diagnostic of tuberculosis and she received another course of drugs. However, the glands enlarged and she developed a diffuse pulmonary nodulation. A fourth biopsy was done, and a portion was sent to two pathologists in different centres; one reported tuberculosis and the other sarcoidosis. A fifth biopsy was done at the Nova Scotia Sanatorium and the findings were considered diagnostic of sarcoidosis. She went on to complete recovery. The tuberculin test fluctuated over the 3 year period. Originally negative at the 250 unit PPD level, she reacted to 5 units at the time of the third biopsy, and was negative again on admission to the Sanatorium. Quite likely this woman had sarcoidosis with superimposed tuberculosis which cleared up on treatment, the sarcoidosis again became dominant and then resolved spontaneously.

In both the seventh and eighth cases, tuberculosis and sarcoidosis were active at the same time. A 20 year old man was admitted for treatment of pulmonary tuberculosis; sputum was positive for tubercule bacilli, the tuberculin test weakly positive (250 unit PPD). After 14 months of treatment with streptomycin and PAS, there was a surprising change in the chest film which now revealed the marked bilateral mediastinal and hilar adenopathy so typical of sarcoidosis (Fig. 1). He had become anergic to tuberculin. A scalene node biopsy was inconclusive and he was booked for an exploratory thoracotomy. However, the operation was cancelled owing to progressive improvement, and he was discharged home. Sixteen years later he was admitted with reactivation of the tuberculosis; sputum again revealed tubercle bacilli but he remained anergic to tuberculin. He responded to treatment with isoniazid and ethambutol.

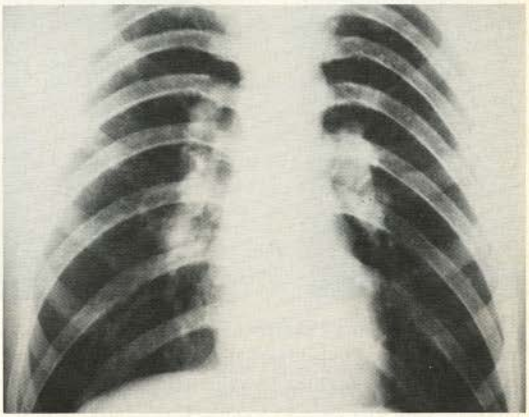

Figure 1. Hilar adenopathy

The final case was that of an asymptomatic 60 year old woman who was admitted with a diffuse pulmonary nodulation that had gradually extended over a three year period (Fig.2). She reacted to tuberculin only at the 250 unit PPD level and a scalene fat pad biopsy revealed noncaseating granulomas considered typical of sarcoidosis. Steroid therapy along with INH and PAS was begun and she was discharged from hospital. Subsequently two gastric lavages carried out on admission were reported positive for tubercle bacilli on culture, indicating the combination of active tuberculosis and sarcoidosis. She was readmitted a few weeks later with superimposed pneumonitis that resolved promptly, and she was discharged in 14 days. The original pulmonary infiltration was undergoing satisfactory improvement. However, she died one year later at another hospital after a short illness and death was attributed to pneumonia, sarcoidosis and cor pulmonale. Unfortunately an autopsy was not done.

These eight cases illustrate some of the problems that may arise in the differential diagnosis of tuberculosis and sarcoidosis. In any given case, it is most important to rule out the presence of tuberculosis which is a progressive and possibly fatal disease if it remains untreated, whereas sarcoidosis usually runs a benign course. At the same time it is also important that the patient with pulmonary sarcoidosis not be misdiagnosed as tuberculosis and be given unnecessary treatment. The contribution of mediastinoscopy towards the diagnosis of difficult cases cannot be overemphasized, but even so there are still patients who may require lung biopsy. If there is any doubt about the presence of tuberculosis in a case of sarcoidosis, chemoprophylaxis should be administered if steroid treatment is to

be given. Indeed there are those that believe antituberculosis therapy should be give to any patient with sarcoidosis who is to receive steroids, regardless of the tuberculin test or other findings.

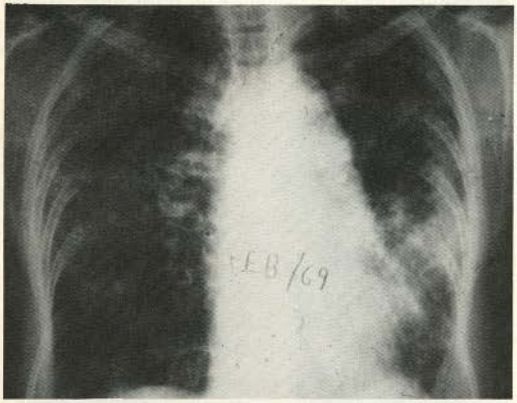

Figure 2. Diffuse pulmonary disease and adenopathy

Today, there is really no doubt that tuberculosis and sarcoidosis are of different etiology. Tuberculosis is due to infection with the mycobacterium tuberculosis but sarcoidsis remains an enigma. The similarities in the two conditions can certainly create diagnostic problems as illustrated in the literature and in this series of eight cases. Above all, let it not be forgotten that even with the decline in tuberculosis, both conditions can exist together. □

ACKNOWLEDGEMENTS

Thanks to Mr. Hector McKean, R.R.L. for his invaluable assistance.

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SARCOIDOSIS: NEUROLOGICAL MANIFESTATIONS

Continued from page 89.

of the affected nerve. The occurrence of large irregular zones of sensory loss over the trunk, reflecting involvement of intercostal nerves or thoracic roots, distinguishes the neuropathy of sarcoid from other forms of mononeuritis muliplex ie., diabetes mellitus or polyneuritis nodosa.

Meningeal involvement which may occur in the region of the spinal cord producing a picture of chronic adhesive arachnoiditis, may also affect, within the subarachnoid space, the adjacent dorsal and ventral spinal nerve roots giving rise to the development and progression of a diffuse polyradiculoneuropathy. This would however be reflected by the usual alterations, as outlined previously, in the spinal fluid.

MUSCLE

Such involvement may be asymptomatic i.e., granulomas detected in muscle either by biopsy or at autopsy in patients without features of muscle dysfunction. It may however be symptomatic either as an acute granulomatous myositis but more often as a chronic myopathy productive of weakness and wasting with or without the presence of palpable nodes in various muscle groups. Muscle biopsy has often been pursued as a means of establishing the diagnosis of sarcoidosis. This presumes however that there is evidence of disease elsewhere as sarcoid involvement of skeletal muscle and of no other organ, though it has been reported, is exceedingly rare. When specifically investigated 50 percent of patients with no muscle or joint symptoms have muscle granulomas. When myalgia, arthralgia or erythema nodosum are present the incidence of the detection of such granulomas increases to 90 percent. Thus as there is a association between the systemic dissemination of sarcoid and the presence of muscle granulomas muscle biopsy may be very helpful in patients with suspected disease in several organs. □

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Current Topics in Community Health

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REPORT OF THE SELECT COMMITTEE ON HEALTH

Volume I of the Report of the Nova Scotia Legislative Select Committee on Health addresses some of the most important issues and concerns facing Nova Scotia's health care system. The Report includes chapters on health care financing, prevention, hospitals, home care, and medicare/hospital insurance. The Report contains forty-two (42) recommendations on these topics for consideration by Government.

After reviewing health care financing the Committee concluded that as a percentage of total provincial government expenditures, Nova Scotia's expenditures for health services have been remarkabley stable over the past twenty years. Compared with other provinces, this same measure shows that Nova Scotia's emphasis on health care has been strong, placing fourth on average among the provinces. However in recent years, provincial revenues have not kept pace with health expenditures. In particular, Federal transfers for health services have lagged behind increases in health expenditures. Hence the Select Committee recommends that the Federal Government be encouraged to provide appropriate funding to help Nova Scotia cover the costs of additional required health services.

The Committee noted that prevention — defined as programs and services which avert the occurrence of disease, illness or dysfunction and which promote the optimal health of the population — has long been recognized as a major theme in health care. However, emphasis in the health care system has been placed on curative medicine and treatment. Based on its investigations, including evidence from recent medical studies, the Select Committee concluded that the health of Nova Scotians can be improved and significant savings in health care expenditures can be obtained, through preventive programs. The Committee therefore urges government to give prevention a high priority and recommends that a Committee be established to devise a preventive health strategy for the Province. The Committee also makes specific recommendations regarding preventive action in areas such as health education, prenatal care, discouragement of the use of tobacco, alcohol and drug abuse, seat belt legislation, and drunken driving.

Nova Scotia's hospitals are a key component of the health care system in the Province, accounting for over two thirds of current operating expenditures on health. The Select Committee concluded that a very high standard of care is provided in Nova Scotia's hospitals, but felt that improvements could be made to the hospital system. Recommendations pertaining to hospitals were made by the Select Committee in the following areas: capital funding for hospitals; target ratios for the provision of acute and extended care beds; the geographic distribution of hospital beds; policy changes to promote flexibility and innovation in hospitals; and the promotion of regional cooperation in the operation of hospitals in Nova Scotia.

More recommendations were made to the Select Committee regarding the development of a home care program in Nova Scotia than for any other specific type of program. One of the most important reasons for this interest is that institutional health care is relatively expensive. By contrast, a co-ordinated home care program involving health and social services personnel and other agencies represents significant savings while continuing to ensure high quality care for the patient. The Committee therefore urges the government to implement a comprehensive, coordinated, home care program for Nova Scotia. As a first step, an advisory committee of health and social services professionals should be established to identify specific public needs and to advise the government on planning a coordinated program.

The Select Committee's investigations into certain aspects of Nova Scotia's Health Insurance System were to some extent overtaken by the passage of the Canada Health Act, which became law on April 7, 1984. Nonetheless, the widespread concern voiced by Nova Scotians regarding direct charges (user fees, premiums and extra billing) prompted the Committee to address the subject in this Report. After reviewing the effects of direct charges in other provinces the Select Committee recommends that user fees should not be introduced in this Province, and that Nova Scotia legislation with respect to extra billing by physicians be amended to comply with the Canada Health Act. If additional provincial revenues are required to finance health care in Nova Scotia, progressive taxation instruments should be used instead of health care premiums. Many recommendations were made to the Select Committee concerning the extension of benefits under the Health Services and Insurance Act to include additional services and supplies. The Committee recommends that, subject to the availability of funding, benefits should be extended to include a number of additional areas which are specified in the last chapter.

Source: *Report of the Nova Scotia Legislative Select Committee on Health. Volume I. Executive Summary.* Halifax Province of Nova Scotia, 1985.

FISHING VESSEL CASUALTIES

In 1984 a study on marine casualty investigations in Canada was concluded by Bernard M. Deschenes, Q.C., for the Minister of Transport. The full report contains information on all types of marine casualties, including those involving fishing vessels, commercial vessels and pleasure craft. The evolution of the accident and casualty investigation systems in Canada receives particular attention, and the legislation and practices of other countries are reviewed. Several recommendations are made with emphasis on the need to make public the marine accident investigation process. As expressed at a London seminar of the Nautical Institute "many lives and ships are lost each year simply because the lessons learned from accident investigations do not reach those who are most concerned, the mariners".

To illustrate the extent of the problem, the following information on fishing and other vessel losses has been extracted from the report. Table I reveals a total of 242 deaths as a result of accidents aboard ship during the period 1975 to 1982, of which 77 (32%) were aboard fishing vessels.

TABLE I

REPORTED DEATHS AS A RESULT OF AN ACCIDENT ABOARD SHIP

Year	Commercial and Other Vessels	Fishing Vessels	Total
1975	17	6	23
1976	21	14	35
1977	16	5	21
1978	15	9	24
1979	26	8	34
1980	23	9	32
1981	22	10	32
1982	25	16	41

Source: Marine Casualty Investigations, Canadian Coast Guard.

Although accidents aboard fishing vessels are known to be underreported, 330 were recorded during this time period. Table II provides a breakdown of fishing vessel total losses by type of casualty. The number of losses during this 8-year period (693) is impresive. The dramatic increase in 1976, when total losses appear to have been relatively constant from 1975 to 1979 was attributed to a severe storm affecting the Gulf of St. Lawrence and the Atlantic coast in that year. Enforcement of the Casualty Reporting Regulations has been improved since 1980. Nonetheless, there is concern that the substantial increase during the period 1980 to 1982 may correspond with a true increase in total losses.

Source: Deschenes BM, *Study on Marine Casualty Investigations in Canada.* Ottawa Ministry of Transport, 1984.

TABLE II

FISHING VESSEL TOTAL LOSSES CLASSIFIED BY CASUALTY TYPE

Year	Fires	Sinking	Ground-ings	Colli-sions	Others	Total
1975	16	20	9	6	—	51
1976	24	20	19	5	23	91
1977	21	24	7	5	—	57
1978	26	16	7	4	4	57
1979	18	30	6	4	5	63
1980	53	35	16	4	3	111
1981	68	29	20	16	8	141
1982	63	22	19	14	4	122
TOTAL	289	196	103	58	47	693
%	41.7%	28.3%	14.9%	8.4%	6.8%	100%

Source: Marine Casualty Investigations, Canadian Coast Guard.

ABSTRACT

CUMULATIVE EFFECTS OF LIFETIME PASSIVE SMOKING ON CANCER RISK

Cancer risk from cumulative household exposure to cigarette smoke was evaluated in a case-control study. Overall cancer risk rose steadily and significantly with each additional household member who smoked over an individual's lifetime. Cancer risk was also greater for individual's with exposures during both childhood and adulthood than for individuals with exposures during only one period. These trends were observed for both smoking related and other sites. These findings are preliminary and must be confirmed with other studies. Nonetheless, they suggest that effects of exposure to the cigarette smoking of others may be greater than has been previously expected.

Source: Sandler DP, Wilcox AJ, Everson RB. *Lancet* 1985; **1**:312-314. □

Notice Re: By-Law Amendments

The By-Laws of the Medical Society stipulate that amendments to them may be proposed at an Annual Meeting of the Society provided they are published in the Bulletin at least one month prior to the Annual Meeting.

The following amendments will be presented by the By-Laws Committee at the 1985 Annual Meeting of the Society.

PROPOSED

THAT Article 6 of the amended By-Laws of The Medical Society of Nova Scotia be amended as follows:

(a) Renumber Articles 6.7 and 6.8 as 6.15 and 6.16 respectively.

(b) Add Articles

6.7 First Year Practice, an ordinary member in his/her first year of practice following initial licensing in Canada.

6.8 Post-Graduate, a medical practitioner whose name is entered in the Medical Register or the Temporary Medical Register.

6.9 Interne/Resident, an ordinary member undertaking postgraduate training in the Dalhousie Program.

6.10 Non-Resident (Nova Scotia Only), a member residing in a province of Canada other than Nova Scotia, or outside Canada and not a C.M.A. member.

6.11 Non-Resident (Conjoint), a member residing outside Canada or Canadian Territory beyond the jurisdiction of any division.

6.12 Retired, an ordinary member no longer in active practice, defined as less than ten hours per week in the practice of medicine.

6.13 Non-practising Scientist, an ordinary member employed as a basic scientist in Dalhousie University and not practising medicine except on an emergency basis only. The dues for this category are 50 percent of the dues for an ordinary member plus $15.00 for the C.M.E. levy, plus C.M.A. dues at 50 percent.

6.14 Over Sixty-Five, an ordinary member not retired; effective the fiscal year following the fiscal year in which the age of 65 was attained, the dues for this category to be 50 percent of ordinary dues. Category M to apply for fiscal year 1985.

EXISTING

6. MEMBERSHIP AND DISCIPLINE

This article includes definitions for Ordinary Members (6.2), Senior Members (6.3), Honorary Members (6.4), Courtesy Members (6.5), Medical Student Members (6.6), Discipline (6.7), and Resignation from Membership (6.8).

Continued on following page.

PROPOSED

THAT Article 6.1 of the amended By-Laws of The Medical Society of Nova Scotia be amended by rewording 6.1 as follows:

The Society shall be composed of regularly qualified Physicians, Internes/Residents, and Medical Students. The Medical Act requires that every duly qualified medical practitioner shall pay the Annual membership dues on or before October 1st each year. Every duly qualified medical practitioner who fails to pay the annual membership dues ceases to be in good standing and thereupon becomes suspended as a qualified medical practitioner. Any physician not wishing to be a Society member should communicate that wish to the Society in writing. Categories of membership are: 1st Year - of actual practice, Ordinary, Post Graduate, Internes/Residents, Non Resident (N.S. Only), Non Resident (Conjoint), Retired, Senior, Honorary, Student, Non-Practising Scientist, Courtesy, and Over Sixty-Five.

EXISTING

6.1 The Society shall be composed of regularly qualified physicians and medical students. Categories of membership are: Ordinary Members, Senior Members, Honorary Members, Special Members, and Student Members.

PROPOSED

THAT Article 9.2.1 (n) of the Amended By-Laws of The Medical Society of Nova Scotia be amended by rewording as follows:

9.2.1 (n) To increase general practitioner representation on Council representatives as follows: From each Branch having one hundred members, two members.

EXISTING

9.2.1 (n) To increase general pratitioner representation on Council representatives as follows: From each Branch having fifty members or less in good standing in the Society, one member; and for each fifty 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.

PROPOSED

THAT Article 12.4.1 of the amended By-Laws of The Medical Society of Nova Scotia be amended by rewording 12.4.1 as follows:

The voting members shall be:

President
President-Elect
Immediate Past President
Treasurer
Honorary Secretary
Chairman, Executive Committee
Vice-Chairman, Executive Committee
and a maximum of two representatives from each Branch Society, and that to qualify for a second representative on the Executive Committee a Branch must have in excess of one hundred members. A Branch member will be interpreted as a Medical Society member who resides and/or practises in a Branch jurisdiction (Regulation 1.1).

EXISTING

12.4.1 The voting members shall be:

President
President-Elect
Immediate Past President
Treasurer
Honorary Secretary
Chairman, Executive Committee
Vice-Chairman, Executive Committee and all elected representatives of the Executive Committee from the Branch Societies.

PROPOSED

THAT Article 12.4.2 of the amended By-Laws of The Medical Society of Nova Scotia be amended by rewording 12.4.2 as follows:

The non-voting members shall be the Executive Secretary, and all observers.

EXISTING

12.4.2 The Non-voting members shall be:

The Executive Secretary
Editor
All observers

PROPOSED

THAT Article 12.5 and 12.5.1 (Discipline Committee) be deleted from the amended By-Laws of The Medical Society of Nova Scotia.

EXISTING

12.5 Discipline Committee

12.5.1 Terms of Reference. The Discipline Committee members are the Medical Society President (Chairman), Immediate Past President and President-Elect. The Committee is charged with the responsibility of investigating charges of unprofessional conduct or of conduct unbecoming to a member of the Medical Society. In conducting the proceedings of the Discipline Committee the principles of natural justice shall be observed. Proceedings of the Discipline Committee may only be instituted by written complaint following which a hearing or due inquiry shall ensue. Full and reasonable notice of any such inquiry shall be communicated to the member, or his counsel, to permit him the opportunity to question the complainant and any other witnesses and to argue as to the merits of the complaint. The proceedings shall be recorded by a competent and duly-sworn stenographer. The decision shall be reserved, then rendered in writing with reasons, a copy being forwarded to the accused, but not to the complainant. The Executive Committee is required to review all decisions of hearings of the Discipline Committee. □

Personal Interest Notes

1985 CONVOCATION
DALHOUSIE UNIVERSITY
FACULTY OF MEDICINE

The Dalhousie University Faculty of Medicine Convocation was held on May 17, 1985, when 90 M.D. degrees were conferred. By place of residence, these graduates were from: Nova Scotia — 52; New Brunswick — 20; Prince Edward Island — 11; United States — 3; and one each from British Columbia, Ontario, Quebec and Malaysia.

Dr. Joseph Francis Horan, Hyde Park, Massachusetts, was awarded the Dr. C.B. Stewart Gold Medal. Honorary degrees were conferred on **Dr. G. Enid MacLeod,** Professor Emeritus, Dalhousie University, and on **Dr. Robert B. Salter,** orthopaedic surgeon, University of Toronto, who gave the Convocation Address.

Dr. Aden C. Irwin, Professor of Epidemiology and Associate Editor of the *Bulletin*, was named Professor of the Year by the Graduating Class. The award, which is a trophy in the form of a small shovel with a silver handle and an inscribed blade, was presented at the convocation exercises.

Dr. Aden C. Irwin, recipient of the Professor of the Year Award and Dr. Cynthia Ann Forbes, class president.

The valedictory address was given by **Dr. Anil Kumar Sharma,** Truro, Nova Scotia.

Drs. Joseph F. Horan, Moira M. Cooper, G. Enid MacLeod and Robert B. Salter.

Dr. Anil K. Sharma delivering the valedictory address. □

PLAN TO ATTEND

The Medical Society of Nova Scotia
132nd ANNUAL MEETING and 21st MEETING OF COUNCIL
Friday, November 22 and Saturday, November 23
Nova Scotian Hotel, Halifax, N.S.

OBITUARIES

Dr. Hermann H. Felderhof, (78) of Pictou County, N.S. died on June 24, 1985. Born in The Netherlands he received his medical degree from the medical school of Utrecht. He was a member of The Medical Society of Nova Scotia and a past member of the Aberdeen Hospital. He is survived by his wife, three daughters, and nine sons. Our sympathy is extended to his family.

Dr. John S. Robertson, (77) of Halifax, N.S. died on July 4, 1985. Born in Churchville, Pictou County he received his medical degree from Dalhousie in 1934. He was the former Deputy Minister of Health of Nova Scotia, held positions on provincial and national committees, and represented Canada at the World Health Organization meetings in Geneva. In 1972 he was made Senior member of The Medical Society of Nova Scotia and the Canadian Medical Association. He is survived by two sons, two daughters, and five grandchildren. Our sympathy is extended to his family.

Dr. Gordon Holmes, (60) of Dartmouth, N.S. died on July 11, 1985. Born in Toronto he received his medical degree from the University of Toronto in 1950. He served for 30 years with the Royal Canadian Navy. He is survived by his wife and four daughters to whom we extend sincere sympathy. □

MEDICAL DEVICES SEMINAR

Health and Welfare Canada is organizing a seminar on the regulatory requirements and use of medical devices in Canada. Scheduled on Oct. 17, 1985 in Halifax, this seminar is open to health professionals and medical devices importers, distributors and manufacturers in Atlantic Canada. To register, phone Vernon Greene, supervisor of the Nova Scotia District Office, at (902) 426-7553 or write to:

Health and Welfare Canada
Health Protection Branch
Nova Scotia District Office
5th Floor, Ralston Building
1557 Hollis Street
Halifax, N.S.
B3J 1V5

Correspondence

To the Editor:

I enclose a copy of the revised guidelines for prevention of bacterial endocarditis. These have been approved by the American Heart Association, and the American Pediatric Society. The major change, which should be noted, is the reduction in duration of preventive measures with regard to dental and upper respiratory procedures.

Formerly, the regime for antibiotic coverage was as follows: A large dose of penicillin one hour before the procedure, followed by a maintenance dose four times a day for two days. The present recommendations are based on invitro studies, clinical experience, and experimental animal data. The duration of preventive antibiotic therapy is reduced to a large dose, one-half hour before the procedure, and a single smaller dose six hours later. It is expected that better compliance will be the result of this reduced dosage schedule.

It should be recognized that endocarditis is a significant risk for virtually all patients with congential heart disease, operated or unoperated, at any age. We therefore bring this to the attention of all physicians.

While I enclose the complete risk of recommendations, you may wish to publish only the summaries, which are included in Tables 1-4. Pediatric doses are given at the bottom of Tables 3 and 4.

Thank you very much for distributing this information.

Sincerely,

John P. Finley, M.D., F.R.C.P.(C)
Department of Cardiology
The Izaak Walton Killam Hospital for Children
Halifax, N.S.

□

EDITORS NOTE: Inserted in this issue is the revised guidelines for Prevention of Bacterial Endocarditis courtesy of the American Heart Association and the Nova Scotia Heart Foundation.

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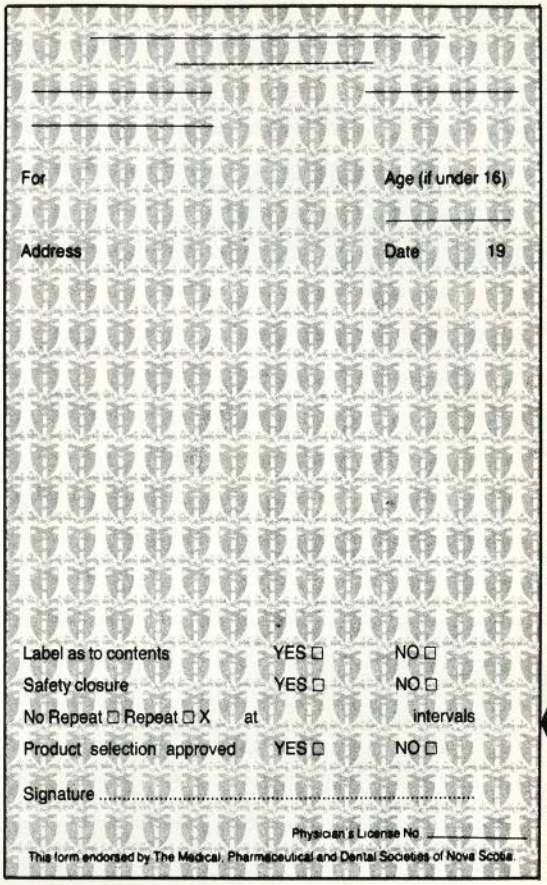

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