THE NOVA SCOTIA MEDICAL BULLETIN

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The Bones of the Matter

This, the third issue of *The Bulletin* sponsored by the Halifax Infirmary, is devoted to a symposium on the subject of "thin bones". Although those super-specialists who confine their interest to one part of the human body, such as the left ear lobe, may not find this symposium invaluable for the efficient conduct of their art, we trust that for the majority of practising physicians these articles will be both informative and rewarding.

As the life span of the population increases, so do the problems of bone demineralization. Probably almost 20 per cent of the population over the age of fifty have radiological evidence of loss of bone density in the dorso-lumbar area. One wonders whether this should then be considered a normal physiological finding or whether we are being negligent in our prophylactic management of one out of every five people in this age group.

The statement that a patient has X-ray evidence of osteoporosis is no more a diagnosis of his fundamental problem than saying he has heart failure. It is incumbent on the physician, once he has uncovered the fact that his patient is suffering from a demineralization syndrome, to investigate the problem thoroughly.

The case may be one of simple osteoporosis; but this is essentially a diagnosis by exclusion and the problem may prove to be one of osteomalacia secondary to gastrointestinal malabsorption, hyperparathyroidism or renal tubular disease. There may be an endocrine disorder such as Cushing's Syndrome or hyperthyroidism or perhaps multiple myeloma or metastatic carcinoma. In this symposium we have tried to cover all these matters in a practical, rather than an esoteric manner.

We have tried, too, to detail the prophylactic management of persons at risk such as pregnant and lactating women and the aged and to indicate the pitfalls in the physiotherapeutic and orthopedic management of spinal fractures

Bone demineralization may be a sign of serious disease of some other system and as such deserves careful consideration with respect to both diagnosis and treatment. A disorder which may have taken several years to develop may require several further years of therapy. The problems that arise are disabling to the patient and frustrating to the physician. Could it be that a more vigorous approach on the part of the doctor would leave both parties somewhat happier? □

I.D.M.

This issue has been contributed by
the Medical Staff of the
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The Pathology of Bone Demineralization

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Halifax, N. S.

Bone is not static like the framework of a building, but is in a constant state of flux even in old people, with microscopic fragments constantly being added and subtracted.

Just, as in building a house, first the scaffolding and framing go up and later are added the fabric of the walls, the bricks and the mortar; so in the laying down of bone there is first the osteoblastic deposition of a rubbery organic osteoid matrix which is followed by the (probably also osteoblastic) mineralization of the matrix.

The house of bone however, is at the same time being torn down by osteoclastic lacunar resorption involving the removal of both mineral and organic matter together; the two processes of construction and destruction accompanying and balancing each other.

The osteoblasts are probably responsible for all the components of the osteoid matrix, collagen, reticulin, elastin, and mucopolysaccharide ground substance (the "mortar") as well as the elaboration of alkaline phosphatase and probably, as suggested above, the subsequent deposition of compound calcium and magnesium salts upon this matrix to form true, mineralized bone.

It appears that the osteoclasts, which some regard as modified osteoblasts, remove the ground substance resulting in the collapse of the other components to form Howship's lacunae.

Whether or not osteoclasts are really rogue osteoblasts or are innocent bystanders watching the house wreckers it is slightly helpful to regard them as closely related to osteoblasts and to appreciate that the serum alkaline phosphatase level is to some degree an index of both osteoblastic and osteoclastic activity; in other words that it will only be elevated if there is an excessive rate of formation or destruction of bone or decreased excretion by the liver. Specifically, phosphatase will not be raised if the excess of destruction over construction has arisen as the result of reduced formation rather than as the result of increased resorption.

Reduction in bone bulk, signifying paucity or undue slenderness of trabecula, is known as *osteoporosis*. It may arise either from inadequate formation of osteoid as in states of senescence, starvation, scurvy or some endocrinopathies or from increased resorption due to either primary or secondary hyperparathyroidism. In either case, what bone that is present, though scanty will be *normally mineralized*.

Poor calcification of bone on the other hand, that is *osteomalacia*, develops from a defect in one (albeit complex) process; namely the failure of the organic matrix to become mineralized as the result of deficiency of available calcium, vitamin D deficiency or of renal tubular dysfunction (de Toni-Fanconi Syndrome, cystinosis etc). Poor calcification *per se* cannot be due to simple resorption, that is pure osteoporosis, though both osteoporosis and osteomalacia may be associated with one another particularly in renal rickets.

Laboratory Findings

HISTOLOGIC: The histologic differentiation of simple osteoporosis, osteitis fibrosa cystica of hyperparathyroidism and osteomalacic states is relatively easy and specific but all too seldom attempted. In view of the difficulty in making a positive differential diagnosis on clinical and radiological grounds it is strange that so little use has been made of bone biopsy. The iliac crest is a readily accessible site.

Osteoporosis In view of aging changes and individual variations, assessment of minor degrees of osteoporosis may be somewhat subjective but in more marked cases the compact bone becomes thinner; the alteration being mainly from the endosteal side[1] and trabeculae become attenuated and gradually disappear; in advanced degrees the scantiness and minute size of these is very striking.

Histological differentiation of the various osteoporotic states from one another is not usually possible except in *scurvy* where biopsy of the metaphysis reveals the diagnostic deeply calcified cartilage of the scorbutic lattice in children.

Hyperparathyroidism: In normal adult bone and in simple osteoporosis, formation and dissolution of bone are progressing at a low level and both osteoclasts and osteoblasts are relatively inconspicuous.

In hyperparathyroidism, however, the normal process is speeded up and active formation and destruction occur hand-in-hand. The slender bony trabeculae are lined by osteoblasts, shoulder to

From: Department of Pathology, Halifax Infirmary.

shoulder but resorption outstrips formation and clumps of osteoclasts, sometimes forming giant cell tumors, may be a prominent feature. Active proliferation of new fibrous tissue sometimes, but not invariably showing cystic change, fills the widened marrow spaces and fails to undergo metamorphosis to osteooid matrix. The picture is usually quite different from that of simple osteoporosis but may resemble fibrous dysplasia. It is easily differentiated from this on clinical grounds.

roidism, metastatic bone disease or multiple myeloma but it is seen in some cases of osteoporosis due to Cushing's syndrome.

Plasma Phosphorus Plasma inorganic phosphate levels are normal in osteoporosis, and low either in uncomplicated primary hyperparathyroidism or in osteomalacia from any cause. High plasma phosphorus suggests renal failure but it is also seen in some cases of osteoporosis due to thyrotoxicosis.

The product of serum calcium and plasma

TABLE I
LABORATORY DIFFERENTIATION OF GENERALIZED RADIOLUCENCY

	Trabecular size	Calcification	Osteoblastic Activity	Se. Ca^{++}	Pl.PO_4"	Ca · P Product	Urine Ca^{++}	Se. Alk. Phosphatase	SPECIAL FEATURES
Osteoporosis	↓	N	Low	N	N	N	↑	N	**Thyrotoxicosis** Pl.PO_4" ↑ Alk. Phos ↑ **Cushing's Syn.** Se. Ca^{++} ↑ **Scurvy** Metaphyseal changes
Hyperparathyroidism	↑	↑↓	High	↑	↓	N	↑	↑	**With renal failure** (Pl.PO_4" ↑ U. Ca^{++} ↓
Osteomalacia	N or ↑	↓	N(↑)	N or ↓	N or ↓	↓	↓	N or ↑	**Hyperchloremic Acidosis** U. Ca^{++} ↑ **Hypophosphatasia** U. Ca^{++} ↑ Ca · P ↑

N — normal, ↓ reduced, ↑ increased.

Osteomalacia In rachitic children osteomalacia produces abnormalities in the growth and calcification of cartilage cells causing a thickened, widened, very uneven epiphysis which is diagnostic. Even in adults Snapper[2] has found changes in the costochondral junctions of the ribs but the hallmark of osteomalacia is the presence of increased amounts of uncalcified osteoid matrix. The recognition of this in regular histologic sections requires careful technical control of the decalcification process but is, we feel, possible. Although undecalcified sections are vastly better for this differentiation[3], preparation of these imposes technical problems in the routine laboratory. Biochemical assay of the calcium content of bone biopsies is relatively easy and, if controlled by simultaneous histologic examination of representative material, should prove of value. Not enough use has been made of isotopic techniques; Lea and Vaughan[4] have shown that there are quantitative differences in the uptake of ^{35}S by normal and osteomalacic osteoid. Employing strontium as a non-isotopic marker of calcium metabolism, Fraser[5] has found differences in the uptake of this by normal, osteoporotic and osteomalacic bones.

BIOCHEMICAL FINDINGS -

Serum Calcium As a general rule serum calcium levels are normal in simple osteoporosis and normal or low in states of osteomalacia. Elevation of serum calcium suggests hyperparathyphosphorus usually lies in the normal range of 35 - 45 in osteoporosis and primary hyperparathyroidism but it is depressed below 27 in nearly all states of osteomalacia.

Urinary Calcium Although blood levels of calcium and phosphorus are usually normal in osteoporosis, urinary calcium excretion is raised. It is raised also in hyperparathyroidism, metastatic bone disease, multiple myeloma and thyrotoxicosis but nearly always depressed in osteomalacic states other than renal rickets.

Serum Alkaline Phosphatase Alkaline phosphatase levels parallel osteoblastic and osteoclastic activity; they are elevated therefore in hyperparathyroidism, primary or secondary Paget's disease of bone, thyrotoxicosis, metastatic bone disease and some cases of osteomalacia, seldom in states of osteoporosis.

Summary

The term *osteoporosis* implies bone which is delicate but normally mineralized. In *osteomalacia* bone bulk is usually normal or actually increased but mineralization is poor.

Although radiological differentiation of the two groups may be difficult or impossible, the histological and biochemical findings are distinctive, enabling cases to be placed into two broad groups.

Further subdivision on an etiological basis by laboratory means alone is not usually possible. □

References on page 309

The Role of Radiology
in the Investigation of Skeletal "Demineralization"

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Halifax, N. S.

The purpose of this presentation is not to summarize the oft-repeated details of radiographic appearances in states of skeletal rarefaction; rather, it is an attempt to impart some perspective regarding the role of radiology in dealing with individual cases where skeletal "demineralization" is of concern, and to offer some practical suggestions in respect to its use.

Diagnosis of the disease responsible for skeletal "demineralization" can rarely be made radiologically. The kinds of help to be expected from X-ray studies include:

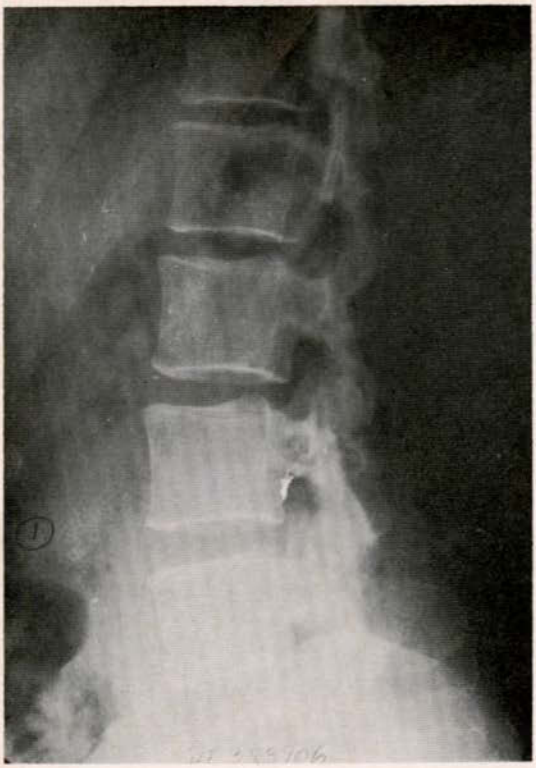
1 recognition of a significant degree of skeletal rarefaction,
2 broad categorization of the type, which may suggest the probable kinds of underlying disease and so be useful in directing further investigation,
3 evaluation of the extent and degree of the process as it affects the skeleton,
4 recognition of associated fractures or other sequelae that may be producing symptoms,
5 discovery of related or unrelated conditions that may be responsible for symptoms, and in some cases
6 follow-up examination may help in assessing the results of therapy.

In regard to the follow-up for evaluation of therapy, it should be emphasized that this is of help only in ccases where definitive therapy for specific conditions has been instituted; eg. following removal of overactive parathyroid tissue, specific therapy for rickets, scurvy; etc. . . Some authorities[1] state that radiologic improvement *never* occurs in osteoporosis. Whether or not this degree of dogmatism is justified, it is a fact that such recognizable reversal of the osteoporotic process would require years and is seldom if ever observed radiologically.

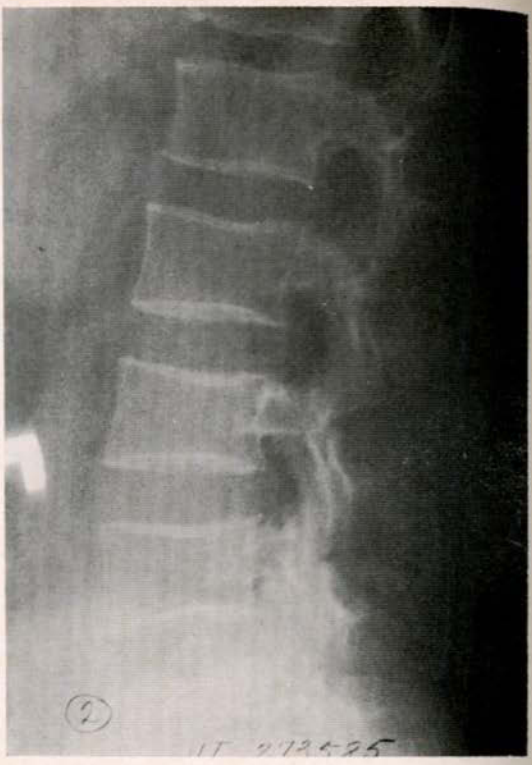
The term osteoporosis is used by the radiologist to mean a reduction in the amount of bone tissue in the whole or part of the skeleton without any radiologic findings to suggest a change in its chemical composition; osteomalacia (bad bone) to suggest the presence of abnormal and poorly mineralized osteoid tissue as a part of the process. Less than the normal amount of bone salts is depicted radiographically as lack of bone "density" or "increased radiolucency" and is common to both the above-mentioned categories of osteoporosis and osteomalacia. In reporting radiologic findings it is common practise to use the term "osteoporosis" when the only significant departure from a normal appearance is increased radiolucency and other findings compatible with this category such as thinning of bone cortex, thin smudgy trabecular patterns, central vertebral body compression; etc. Only when findings of a more specific nature are present such as "pseudofractures" in the adult or a widened appearance of the growth plate in childhood rickets, can one postulate, on radiologic evidence, poorly mineralized osteoid tissue and so place the process in the category of osteomalacia. On occasion even more specific findings such as the small sub-periosteal cysts of the phalanges in hyperparathyroidism might be present. When seen, such findings are of great diagnostic significance; their absence in any individual case can *never* "rule out" anything. This is to point out that in a radiologist's report the terms "demineralization", "osteoporosis", "lack of bone density" or "increased radiolucency" may all have much the same meaning.

Categorization as outlined above does not constitute a diagnosis. One recent classification[1] lists some thirty-two conditions that give rise to the radiographic complex referred to as osteoporosis; there are probably as many entities responsible for osteomalacia.

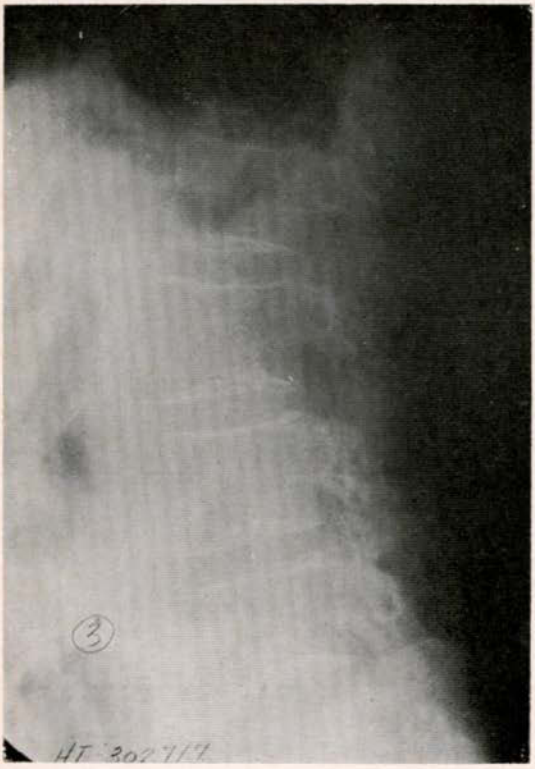
From the Department of Radiology, Halifax Infirmary.



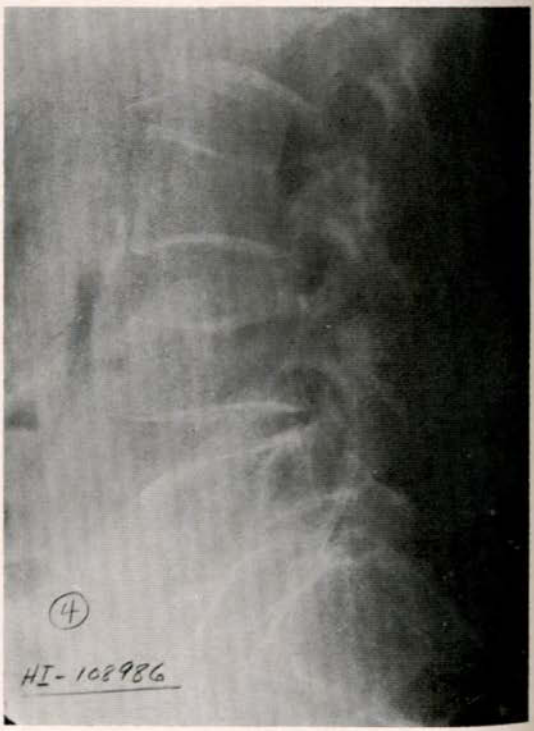
1. Normal - White female, age 38 years.



2. Cushing's Disease - White male, age 19 years.



3. Malabsorption Syndrome - White female, aged 64 years.



4. Plasma cell myeloma - age 51 years.

We should also point out that while even the broad categorization outlined above can be very helpful in directing investigation it is often difficult or impossible to go even this far radiologically. Since the most common complaint resulting in X-ray examination that reveals skeletal demineralization as the major significant finding is back pain, we have chosen to illustrate the difficulty by showing four lateral views of the lumbar spine. "Demineralization" or "osteoporosis" is recognized in three of these; categorization (to say nothing about diagnosis) is not possible from this study alone.

Once skeletal demineralization of significant degree is recognized, how then should one proceed? The most important point here is to emphasize that one *must* proceed to attempt a diagnosis; the clinical and laboratory aspects are outlined in other presentations in this issue of the Bulletin. As far as further X-ray studies are concerned, these should be planned in consultation with the radiologist. In general the most helpful views include: lateral skull, lateral dorsal and lumbar spine, frontal view of the pelvis to include the upper femora and postero-anterior view of the hands. Special views of the teeth for "loss of the lamina dura" and repeated or serial views of the same region to look for "changes" are rarely helpful and for the most part a waste of time and effort.

Of paramount importance is that the radiologist be fully informed of all available laboratory data regarding the case, and that he be made aware of the clinical thinking at the time when X-ray studies are being planned and interpreted. It is only by such an integrated approach that any real help can be expected from radiological studies.

Summary

1 Radiology can be most helpful in the recognition and categorization of states of skeletal rarefaction.

2 The terms "skeletal rarefaction", "demineralization" "increased radiolucency" and "osteoporosis" can all have much the same meaning in a radiologist's report. The term "osteoporosis" should not be construed as a diagnosis.

3 X-ray studies can be most helpful in diagnosis only when planned in consultation with the radiologist as an integral part of all other modalities of investigation. □

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The Pathology of Bone Demineralization

continued from page 306

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2. Snapper, I. Medical Clinics on Bone Diseases. New York, Interscience Pub. (1943).
3. Ball, J. *J. Clin. Path.* 10: 281 (1957).
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WINTER SOLSTICE

December 22, when the sun is at the farthest point south, marks the beginning of winter.

While December is the month with the shortest day in the northern year, the Canadian Highway Safety Council reminds us that December may seem the darkest month for accident victims.

Shorter daylight hours, and bad weather conditions are some of the factors contributing to the poor visibility and generally hazardous driving conditions of December.

Foul weather, distracted drivers, and preoccupied pedestrians can add up to an accident.

FORTY YEARS AGO

From the Nova Scotia Medical *Bulletin* December, 1926

"ORTHOPAEDIC DEFECTS OF THE GREAT

Aesop suffered from Pott's disease

Alexander the Great - Torticollis

Talleyrand - Right Equino Varus

Walter Scott - Talipes Equinus following Infantile Paralysis

Lord Byron - Equino Varus

Hawthorne - Osteomyelitis

It may be assumed that the celebrity of such cases was largely laboriously and painfully attained by way of compensation for inferiority complexes. Physical defects doubtless serve useful purposes with greater frequency than we are apt to imagine.

A deformity may decide the issue of greatness or mediocrity."

DECK THE HALLS WITH DISINFECTANT

The following items are excerpts from the Christmas message to THE BULLETIN issued by the Information Service of the Department of Health and Welfare, Ottawa, December 1966.

BON APPÉTIT, BUT WATCH THE TRICHINOSIS

Pork should never be eaten raw. unless otherwise treated to destroy trichina, should be cooked sufficiently to reach an internal temperature of at least 165°F. Cooks who are in the habit of tasting their meat preparations before cooking should keep this risk in mind, especially in the Christmas Season.

WAS IT THE BIRD OR THE STUFFING?

To avoid food poisoning after eating your Christmas bird, watch your timing preparations and cook the beast thoroughly. Never dress the bird several hours before cooking or leave it overnight in the refrigerator. The cold mass of stuffing acts as an insulation to cooking heat. To destroy salmonella germs the bird should reach an internal temperature of at least 165°F.

HANGOVER HEADACHE

Some of the factors causing hangover headache are the hyperemia due to ethl alcohol and sensitivity to some chemical by-products of alcohol. Home care of rest or a combination of pain-relievers and a stimulant, such as coffee, in time restores many patients. If necessary a physician may be called for the replacement of fluids and relief of emotional difficulties.

Calcium Requirments of Pregnancy and Lactation

J. T. DUNNE, BSc, MD, AND M. G. TOMPKINS, MD, FRCS (C)

Halifax, N. S.

That calcium is a necessary nutrient in the diet of pregnant and lactating females has been known for some decades; however, specific correlation between calcium requirements of the fetus, daily quantitative intake of the mother, and the further needs during lactation have not been reported in the recent literature.

The calcium requirements for the standard fetal skeleton has been calculated to be of the order of 30 gms. total, or approximately 250 mgs. daily in the last trimester when the demand is highest[1]. Since calcification of the fetal skeleton is progressive from the beginning of the third month, it may be taken that the fetal parathyroid is active from then also.

Hytten and Leitch[1] reviewed a number of studies of dietary records of a number of pregnant females of different socio-economic classes in several large cities of the world and found that the average calcium intake from diet amounted to approximately 840 mgs. daily (range 500 - 1,200 mgs.). It was noted by Hankin and Symonds[2] that the calcium intake paralleled both the protein intake and total caloric intake. The Canadian Council on Nutrition has recommended that daily calcium allowance for women in the second half of pregnancy be 1,500 mgs. A retention of one third of the allowance for pregnancy is implied, though absorption and retention will be much higher relatively where intake of calcium bears a closer relation to need. Evidence from farm animals shows that calcium is stored in pregnancy and mobilized for milk secretion.[1] It seems likely that if the supply is adequate women also will store calcium but further information is needed in this regard.

Hankin and Symonds[2] in a comprehensive study of diet and lactation in 174 women found the length of lactation to be higher in those women with better prenatal and postnatal diets. It is difficult to assess the influence of diet *per se* on lactation for other factors such as psychological, socio-economic, etc., are known to exert some influence. Nevertheless, these authors seem to be able to draw some valid conclusions. They found that the critical intake levels of protein and calcium for the maintenance of lactation are approximately 70 gms. per day and 800 mgs. per day respectively. No apparent increase in length of lactation occurred with intakes in the range of 110 gms. per day of protein and 1,600 mgs. per day of calcium. These levels are lower than the N.R.C. (1958) allowances[2] which are 98 gms. per day of protein, 2,000 mgs. per day of calcium and 3,300 calories.

These aspects of nutrition, however, are not wholly determinants of successful lactation for in underprivileged countries where poor economic conditions make breast feeding essential, women on near starvation diets have been found to continue breast feeding over long periods. Presumably a large amount of ingested calcium is absorbed and calcium balance is kept at a minimum of negativity with highly efficient conservative measures, which keep osteoporosis at a minimum.

Consider the evidence then of calcium requirements in late pregnancy and lactation and the calcium available in the average diet. One 8 oz. glass of whole milk supplies 290 mg. calcium. The calcium content of some of the present-day vitamin and mineral preparations is listed below.

Beminal - (Ayerst).....10 - 50 mg. calcium pantothenate
Osteofer - (Anca).....125 mg. calcium gluconate
Dayalets filmtabs - (Abbott)....5 mg. calcium pantothenate
Adelflor prenatal tabs - (Upjohn) 250 mg. calcium carbonate
Obelins - (Mead-Johnson)....250 mg. calcium carbonate
5 gm. calcium pantothenate

These preparations fall short of the daily requirements and are inadequate for significant calcium supplement if needed. Calcium can be supplied adequately with Calcium Sandoz effervescent tablets, each tablet supplying 4 gm. of calcium gluconate.

From the Department of Obstetrics and Gynecology, Halifax Infirmary.

To summarize then we may conclude that:

1 An adequate diet supplies sufficient calcium and supplementary calcium is not needed where diet is adequate.

2 There is no evidence to substantiate the claim that leg cramps in pregnancy are due to a calcium deficit.

3 The fetus is never in jeopardy from calcium deficiency.

4 It would seem that lactating women do need extra calcium and protein and this could be adequately supplied with whole milk (24 ounces daily).

5 Calcium supplementation is otherwise best achieved with Calcium Sandoz effervescent tabs 4 grams, one twice daily. □

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1. Hytten, F. E., and Leitch, I. The Physiology of Human Pregnancy - Oxon. Blackwell's Scientific Pub. (1964) pp 319 - 353.
2. Hankin, M. E., and Symonds, E. M. *New - Australia Zealand J. Obstet.* and *Gynecol.* **5**: 86, 1965.

Comment:

The authors base their conclusions on the premise of "an adequate diet". According to the U.S. Dept. of Agriculture, the mean intake of calcium in the United States is about 1 gram per day.[3] In Canada as a whole, the average intake during 1962 was 1.002 grams a day, but a limited study in Newfoundland showed that 82 per cent of the population consumed less than 0.85 grams per day.[4]

If the recommendations by the Canadian Council on Nutrition (1.5 grams) or by the N.R.C. (1958) (2.0 grams) quoted supra are accepted as realistic, the conclusion is inescapable that an additional daily supplement of from 2 to 4 glasses of milk should be prescribed for the *average* pregnant or lactating woman. As, presumably, some 50% of the population will be taking less than the average amount, this recommendation is even more urgent.

Ed.

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Osteoporosis in General Practice

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Osteoporosis is one of the most important and confusing problems facing the family physician today. As the patient's life span increases, so does his chance of developing osteoporosis: approximately 30 per cent of the female and 10 per cent of the male population over the age of 50 have X-ray evidence of demineralization. The family physician sees the end result of an illness (physiological or unphysiological) which has been going on for many years, and finally presents itself either as backache or bony fracture. It may be asked: "Should we wait until the complications occur or attack the problem at an earlier stage?" There is marked variation of opinion in the world literature. Some of the main opinions are presented and discussed below.

Osteoporosis is essentially a diagnosis made by exclusion. It is not uncommon, in fact the rule in some areas, for radiologists to report any form of diffuse demineralization involving the dorso-lumbar spine as "osteoporosis." This is similar to reporting all "coin lesions" of the lung as granulomas. Although they may be correct in most cases, they may mislead the physician. The best that one can say is that the bone is demineralized; the exact reason will remain unknown until the patient has been properly investigated. It is impossible to say from an X-ray of the spine whether the patient has osteoporosis, osteomalacia, hyperparathyroidism, multible myeloma, Cushing's Disease, thyrotoxicosis or metastatic bone disease since all can, on occasion, give identical X-ray pictures.

It is not the purpose of this paper to discuss the differential diagnosis of bone demineralization, but it is important for us to realize that X-ray evidence of demineralization may reflect a serious and sometimes curable underlying disease. All patients with demineralization of bone deserve the benefit of more investigations than just an X-ray of the spine. Hemoglobin, sedimentation rate, complete urinalysis, serum calcium, phosphorus and alkaline phosphatase, total serum protein with albumin/globulin ratio, blood urea nitrogen and CO_2 combining power should all be studied. X-rays of the skull, long bones, pelvis and hands, as well as those of the lumbar-dorsal spine should be done. In simple, uncomplicated osteoporosis, all of the above with the exception of the lumbar-dorsal spine X-ray should be normal. If this is not so, then another cause for the demineralization should be sought. The term osteroporosis, generally speaking, is applied to any condition where there is a reduction in the amount of bone tissue per unit volume without change in its composition (that is, what bone present is normal), compared to osteomalacia where the composition of bone is changed (that is, what bone present is abnormal). One can divide osteoporosis into two major groups:

Primary - where hormone imbalance and calcium imbalance play a major role.

Secondary - where such things as immobilization, hyperthyroidism, steroid excess either endogenous or exogenous, Vitamin C deficiency, and protein deficiency are of importance.

Secondary osteoporosis is self-explanatory. The primary group is most confusing. The pathogenesis has not been firmly established, but there is general agreement that the main defect is one of increased bone breakdown rather than one of decreased bone formation. It is evident that there is a progressive reduction in bone density after middle age (40-50) and that all elderly people are osteoporotic when their bones are compared with young adults. Why should this occur?

In the early 1940's, Albright suggested that gonadal deficiency leads to failure of bone formation and that this failure can be reversed by the use of estrogens and androgens. Recently, workers in this field have shown that estrogen deficiency leads to a slight elevation of serum calcium (but still within normal range) thus to hypercalciuria and therefore, over a long period, to negative calcium balance. Estrogen replacement, therefore, should lower serum calcium slightly thus preventing hypercalciuria and thereby conserving body calcium. Estrogen deficiency may be a factor in the etiology of osteoporosis, but is certainly not the single determining factor: not all post-menopausal women develop

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osteoporosis. Induction of artificial menopause does not lead to increase in premature osteoporosis. Removal of gonads in animals does not always lead to osteoporosis. Some males develop osteoporosis. Nevertheless, in support of the estrogen theory, several investigators claim that estrogen alone has never been shown to cause remineralization of the bone, or improvement of bone density on X-ray.

Over the past ten years, Albright's theory has received much criticism, particularly since the advent of radioactive tracer techniques for measuring bone metabolism. Several workers have postulated that osteoporosis is the end result of prolonged calcium deficiency leading to a continuing negative calcium balance. In support of this, the following points are noted. The osteoporotic individual consumes approximately 25 per cent less calcium than the non-osteoporotic individual. Since the faecal loss of calcium is quite constant and similar in both groups, this discrepancy in intake will lead over a long period to demineralization of the bony skeleton; a negative balance of 50 to 100 mg. of calcium per day is often found by metabolic studies. If this be the case, then in one year, 18 to 36 grams of calcium will be lost from the skeleton. Since the skeleton contains approximately 1,000 grams of calcium and has to be depleted of at least 30 per cent of its contents before there is X-ray evidence of demineralization, it then follows that a period of at least 10 to 20 years must pass before osteoporosis becomes evident on X-ray. In females, one must also consider an average loss of 20 grams of calcium per baby and for breast feeding each child (3 months) an average loss of 15 grams. One can then see the relatively strong position this group of workers is in, especially since it has been shown that low calcium diet in animals leads to the classical picture of osteoporosis and that this is reversed by increasing calcium intake. On the other hand, there is no evidence that osteoporosis occurs more frequently in those countries where calcium intake is low; and some investigators have found no significant difference in the calcium intake of osteoporotic, compared to non-osteoporotic individuals in our country.

In this confused situation, the physician may be in some doubt, not only as to whether the osteoporotic patient should be treated, but at what stage, and how. We would recommend that only the following patients be treated:

All those of any age group with symptoms that are attributable to osteoporosis.

All those under age 60 without symptoms.

Specific Therapy

HORMONAL: Estrogens are best reserved for women, and should be given in physiological doses. Treatment should be continued until the patient is 75 years old. With such prolonged therapy, an inexpensive product is preferred: Stilbestrol, 1 mg. daily is satisfactory. It is best given for the first three weeks of every month, unless the patient has had a hysterectomy, when it may be given continuously. If side effects occur, the dose can be halved.

Androgens - an androgen plus estrogen is probably a better "bone builder" than estrogen alone. A product with very little masculinizing effect should be used. There are several preparations of equal potency on the market, Danabol 2.5 mg. daily is inexpensive and fulfils the requirements. Men with lack of secondary sexual characteristics, unless senile, are probably best treated with a sexually potent androgen. The intramuscular route gives better results than any present-day oral preparation, although after a couple of years of intramuscular therapy, the patient may be carried on oral preparation. Testosterone Cyclopentilpropionate 200 mg. intramuscularly every two weeks for one year and then every month thereafter will prove satisfactory. Osteoporosis developing in otherwise normal males requires an androgen without secondary sexual effect and Danabol in a dose of 5 mg. per day is recommended.

CALCIUM THERAPY - This should be considered a must for every osteoporotic patient regardless of sex, unless there is a contraindication such as renal disease, renal failure or lithiasis. An intake of approximately 1 gram of calcium per day should be adequate. Both skim milk and whole milk have approximately 300 mg. of calcium per 250 ml.; therefore two to three glasses per day would give an adequate calcium intake. Supplements are available for those who dislike milk, but this is not advisable as the protein derived from milk is also beneficial in the laying down of bone. Calcium gluconate effervescent tablets (Sandoz) contain approximately 350 mg. of calcium per tablet. Each tablet has slightly more calcium than a glass of milk but is also more expensive (approximately 10 cents per day more).

Nonspecific Therapy

Adequate nutrition, rest, exercise, physiotherapy, and on occasion, the use of a back brace are of importance. Lumbar supports, while valuable in disabling osteoporotis with or without fractured vertebrae, should be used for only short periods as prolonged use may aggravate the underlying condition. They may cause progression of the osteoporotic process due to prolonged immobilization of the spine.

Summary

Osteoporosis remains a partially unsolved problem, but with the information available today, we, as physicians should be attempting to prevent this disabling disease and also to treat it energetically when it arises. What the results of therapy will be are as yet uncertain, although many patients obtain dramatic relief of their symptoms within weeks of initiating therapy. This is only subjective evidence, but recently special techniques have demonstrated recalcification of osteoporotic bones in some patients. Increasing knowledge with regard to osteoporosis may influence our future concepts concerning etiology and thus our form of management. □

Physiotherapy in Osteoporosis

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It has been recognized for many years that disuse produces osteoporosis. Disuse may be due to paralysis of a limb, immobilization by a cast, or inhibition of use because of pain on movement. X-rays of the immobilized parts taken at various stages during the immobilization reveal that the appearance of demineralization increases over a period of months or years, then seems to stabilize even though paralysis or immobilization continues. As active movement of the part is resumed the X-ray appearance of demineralization gradually abates and eventually the bone assumes a normal appearance. It is recognized that the period of recovery may be quite prolonged depending on the severity of the osteoporosis.

The phenomenon of disuse osteoporosis has been explained for years by a decrease in osteoblastic activity and it was assumed that bone resorption continued unchanged, resulting in imbalance between formation and resorption of bone leading to a net reduction in bone mass.[1] Although there appears to be little reason to doubt that disuse produces bone atrophy, the mechanism of production of this bone atrophy has been the subject of considerable controversy and questioning in recent years. Dietrick et al[2] showed that immobilization of normal subjects resulted in negative calcium balance and that restoration of weight bearing stresses produced a reversal of this effect, but they also observed that mechanical stress did not influence the duration of negative calcium balance in cases of actual paralysis.[3] However studies of bone metabolism in disuse employing radiocalcium indicate that bone formation during the process of disuse osteoporosis is normal but bone resorption rate is 2 to 3 times normal. In those patients having long established osteoporosis due to disuse the rates of bone formation and bone resorption were either normal or slightly low.[4]

Even though we recognize that the older concepts regarding the mechanism of development of disuse osteoporosis are open to question we are still left with the recognized fact that osteoporosis does occur with disuse and it is possible to reverse this osteoporosis by the resumption of activity including the stress of muscle contraction, strain on ligaments and weight bearing stress on bones. In the presence of paralysis it does not appear possible to prevent or reverse to any significant degree the X-ray appearance of demineralization of bones. In other words externally applied stresses have not been observed to reverse the process of bone atrophy and it appears necessary to have a resumption of active muscle contraction and joint movement.

It is not the purpose of this paper to discuss the treatment of osteoporosis of other types where hormone or calcium imbalance play the major part, even though these types may lead to immobilization of the patient because of pain from collapsed vertebral bodies, etc., giving a chance for disuse bone atrophy to be added to the picture and complicate it.

Prevention of Disuse Osteoporosis

In the case where a fractured arm or leg is immobilized in a cast or placed in traction, the basic principle for prevention of osteoporosis is active exercise of the parts of the limb not immobilized or those parts which can be safely moved without jeopardizing the healing of the fracture - in some cases it may be possible for the patient to carry out isometric or muscle setting exercises of the injured part, even when the part is immobilized by plaster, in an attempt to reduce the degree of inevitable osteoporosis. A typical example would be exercise of the quadriceps by straight leg raising or muscle setting exercise for a patient with a long leg cast following fracture of the tibia with the hope of reducing to some extent the degree of osteoporosis of the femur and maintaining all possible quadriceps strength. It is also important to remember that the patient may develop a habit of relative inactivity of the uninjured parts during the period of healing of the injured part. This is prone to occur in apprehensive or debilitated patients, and these debilitated patients may have a pre-existing degree of osteoporosis of post menopausal or senile type. Here the principle of active exercise of the uninjured limb is particularly important if the period of

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relative immobility will be prolonged. Even patients who find it difficult to follow a program of therapeutic exercise because of senility can usually carry out simple active exercises of the unimmobilized limbs under the direction of the doctor, nurse or physiotherapist. The provision of sand bags, springs or other methods of resisting active movement certainly enhances the value of active exercise in preventing disuse osteoporosis.

Treatment

This is simply an extension of the technique used in prevention. It is presumed that the tissues have healed sufficiently to allow active movement and this active movement should be performed through the fullest possible range and against some form of resistance. In reliable patients with a fracture of the lower limb, partial weight bearing should be allowed when appropriate and gradually increased as the stability of the fracture allows. In unreliable patients, or in those not able to handle crutches or other supports skilfully it may be necessary to forego weight bearing until the fracture is strongly healed and rely entirely on resisted active exercises, non weight bearing. The use of the walking cast in selected cases has been shown to encourage the earlier improvement in bone mineralization presumably because of muscle contraction and weight bearing even though the injured parts are encased in plaster.

It is recognized that pain in a part and consequent immobilization can produce disuse osteoporosis but some practitioners feel that osteoporosis itself is a source of pain, particularly in the foot and ankle, and although the resumption of weight bearing does produce varying degrees of discomfort it must be tolerated as the very act of resuming functional use of the limb restores the normal balance between bone production and resorption and in the end it appears to relieve the degree of discomfort due to osteoporosis.

Osteoporosis of the spine from various causes may produce vertebral collapse, considerable pain on movement and cause the patient to reduce activity to a great extent thereby inviting the further complication of disuse osteoporosis. A good many of these patients are helped by a well fitted corset which allows them to become ambulatory again. In addition to ambulation as tolerated some of these patients with anterior wedging of vertebral bodies can tolerate trunk exercises in the reclining position with particular emphasis on contraction of trunk extensors and avoidance of trunk flexion. □

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Hyperparathyroidism

continued from page 324

Management

1. Treat the primary disease, namely, chronic renal failure.
2. Negative calcium balance should be treated with large doses of vitamin D as stated above. Rarely, subtotal parathyroidectomy offers gratifying results.
3. Measures should be taken to reduce serum phosphate, namely, aluminium hydroxide in adequate dosage. □

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The Aging Spine

Fractures and Osteoporosis

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There is a significant increase in fractures of the spine due to osteoporosis after the age of 60. The ratio of the occurrence of osteoporotic compared to malignant vertebral collapse undergoes a complete reversal between 55 and 60 years of age. Published figures give ratios of 1:2 below 55 years and 12:1 above 60 years. Vertebral collapse over the age of 60 has a 10:1 chance of being osteoporotic in origin.[1] Smith and Frame[2] radiographed the spines of over 2,000 "normal" women and found that the incidence of wedging or fractures of vertebrae due to osteoporosis rose from 6.8 per cent at 60 - 64 years to 20.3 percent at 70 - 74 years. In the general population of Nova Scotia only 8.6 percent are 65 years of age and over, but the various problems of this age group are emphasized by the fact that they occupy 1 out of every 4 beds in the wards of the Victoria General Hospital.[3] Epidemiologic studies of the problem of fractures of the aging spine, with or without osteoporosis, should enable us to provide better treatment for the patient and also help to relieve the load on our "acute" hospital beds.

Age and Sex Distribution

Rowe and Sorbie[4] indicated an apparent preponderance of women experiencing fractures of the aging spine. On enquiry, however, this is less convincing when compared with the preponderant female population on which they based their study.

Etiology

Age: - Since the time of Francis Bacon, the problem of senescence has been approached in three different ways and has been summarized aptly by Comfort:[5] "Do we age because of the loss of cells we are unable to replace? Do we age because the cells we produce late in life are 'less sound' than those we produce when young - in other words, are somatic cells an unstable clone? Do we age because of colloid changes? Or is the process a mixture of all these, or is it the product of yet another cause? Finally, is there a single 'cause', other than an evolutionary one, for aging at all, or is it a ragbag of effects with which evolution has been unable to deal?" There are no complete answers to these problems and none has been investigated by convincing experiment.

In 1918, Alexander Graham Bell reported the results of an exhaustive study on 8,907 members of a family in a monumental work entitled "Duration of Life and Conditions Associated with Longevity".[6] He was intrigued by the problem of "Who Shall Inherit Long Life,"[7] the title of another paper which he published a year later. In his study of the pedigree of the Hyde family, leaving out deaths in pre-adult life, he found that of 184 people whose parents reached the age of eighty or more, the average duration of life was 52.7 years; for the 127 Hydes whose parents died before reaching the age of sixty, the average duration of life was only 32.8 years.

In 1824 Sir Astley Cooper[8] in his "Treatise on Dislocations and on Fractures of the Joints" suggested that the skeleton becomes brittle (because of changes in skeletal metabolism) with age. He noted that ". . .Old age, however is a very indefinite term; for in some it is as strongly marked at sixty, as in others at eighty years. That regular decay of nature which is called old age, is attended with changes which are easily detected in the dead body; and one of the principal of these is found in the bones, for they become thin in their shell, and spongy in their texture. The process of absorption and deposition varies at different periods in life. . . ."

Even Cicero[9] in 44 B.C. referred to "Old age . . . .which all men wish to attain and yet reproach when attained."

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External Causes: - Brittle vertebrae require very little stress to produce a fracture. Rowe and Sorbie[4] found that 33 per cent of their patients fractured their spines in the course of ordinary daily activities such as raising a window, lifting a weight, after sneezing or coughing or following a minor fall. Many fractures occurred spontaneously (Fig. 1)

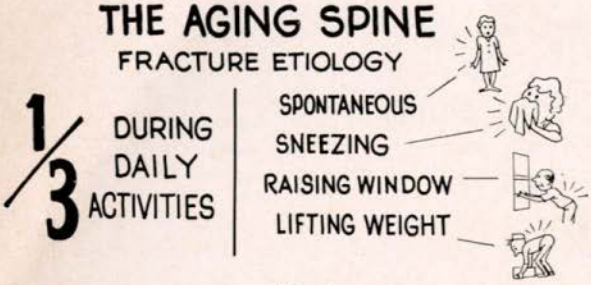

Fig. 1

Minor falls may produce fractures and six common causes of such falls have been listed by Sheldon.[10]

General weakness.
Arthritis of the lower limb joints.
Minor pyramidal tract lesions.
Paralysis agitans.
Syncopal attacks.
"Premonitory falls."

Other factors that play a part in falls are:

Unexplained falls, so-called "drop seizures."
Failing eye-sight.
Bifocal spectacles.
Defective hearing.
Slow reaction-time.
Vertigo.
Impaired vibration sense in the lower limbs.
Obstacles in the house, such as furniture, rugs and pets.
Slippery floors and slippery baths.
Impaired coordination in the dark.
Leaning backwards.

Internal Causes: - 1. *Osteoporosis or Osteopenia*

The principal cause of fractures in the aging spine is osteoporosis which may be regarded as a disease of too little bone without alteration in its chemical composition. In 1962 Holdoway, Buhr and Little[11] reported the results of a study of the X-ray diffraction patterns of cancellous and cortical bone and it was found that in all age groups, other than infants, only hydroxyapatite lines were noted, both before and after heating, whether the specimens were "normal" or osteoporotic; this unchanged mineral composition suggested that the mineral crystallites have a purely passive role in any changes which may occur in osteoporosis.

Pathologically there is a loss of internal lamellae of cortical bone and a reduction in the number of trabeculae of cancellous bone. Microscopic study fails to show either the increased osteoclastic activity indicative of osteitis fibrosa or the uncalcified osteoid seams of osteomalacia. The levels of serum calcium, phosphorus and alkaline phosphatase are normal in osteoporosis in contrast to osteomalacia and primary hyperparathyroidism, the accepted prototype disorders of defective mineralization and excessive resorption respectively. Robinson[12] has pointed out that the water content of the matrix of old bone is less : 30 per cent in young bone and 20 per cent in old bone. This process of dehydration with age is common to all connective tissue.

2. *Immobilisation.* Disuse osteoporosis is a well-recognized entity and it is generally held that a person confined to bed for upwards of three weeks goes into negative nitrogen, calcium and phosphorus balance from atrophy of muscles and bones, and that these processes are accelerated by injury and sepsis.

The reality of this problem is reflected in the great concern of NASA (National Aeronautics and Space Administration) over the effects of weightlessness and decreased activity on the astronaut's musculoskeletal system during prolonged space flight missions. Bone X-ray densitometry experiments are scheduled on several of the Gemini flights to determine any change in bone mineral content of the astronauts.[13]

3. *Nutrition.* Information on the relationship of nutrition to osteoporosis is badly lacking but it would seem obvious that there is a danger of bone atrophy if the basic ingredients for making bone are not available. Various reports have been published to show that the incidence of osteoporosis is higher in starvation,[14] in low calcium intake,[15] and in multiparous as compared with nulliparous women;[16] however, in the patients studied, the presence of other contributory factors cannot be excluded. There appears to be a correlation between long-continued calcium intake and bone density values[17] but this relationship has not been observed consistently.[18] A large number of disorders associated with protein depletion are also associated with osteoporosis, suggesting that it is basically a disease of osteoid; for practical purposes, decreased collagen production. In a geographically localized group of women, a highly significant correlation has been noted between ascorbic acid intake and the os calcis density coefficient.[19]

4. *Endocrine Causes.* These causes were discussed in an earlier paper (Buhr and Cooke, 1959)[10]. In essence, there are considerable species differences in the action of the sex hormones on bone; but it happens that in man, as in the pigeon and dove, both oestrogens and androgens have a bone-building effect. These facts were first applied to the treatment of human beings by Albright, Bloomberg, and Smith[20] in 1940, and hormonal treatment has been extensively used for patients with postmenopausal and senile osteoporosis of the spine.

In a subsequent "Report to the M.R.C. Working Party on Fractures in the Elderly" by Knowelden, Buhr and Dunbar[21] an attempt was made to discover the absolute incidence of fractures in older people in Britain. In the 5 years from 1954 to 1958, 4,260 Dundee residents and 2,213 Oxford residents aged 35 years and over were treated by the fracture services of those cities. The fracture patterns were carefully studied and it was concluded that, ". . .nothing in these patterns suggests a sudden change in trend coincident with the menopause or senile deterioration. Since these hormonal events are more abrupt and earlier in women than in men, it would be expected that hormonal-dependent events would show earlier in women. No such trend is seen, and it seems more likely that the etiology of these fractures is multi-factorial and that, if changes in bone play a large part, they have their origin far earlier than the conventional boundaries of old age."

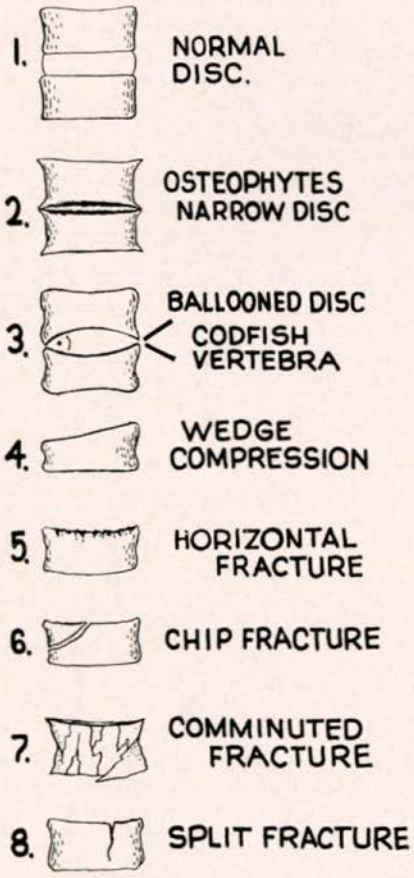

Fig. 2

Level of Fracture

The most frequently involved vertebra is L1 which accounted for 26 per cent according to Key and Conwell[22] and 24 per cent in the series reported by Rowe and Sorbie.[4]

Types of Fracture

In the series of 237 fractures noted above 209 were of the compression variety. Fig. (2) shows the common types of fracture of the bodies of vertebrae.

Fish-mouthing, biconcavity or "cod-fish vertebra" occurs far more commonly in the lumbar region whereas anterior wedging is seen more frequntly in the thoracic area. In osteoporosis there is commonly an increase in vertebral biconcavity which may be interpreted as a gradual fracture resulting from the expansion of an intact disc into a vulnerable softened vertebral body.

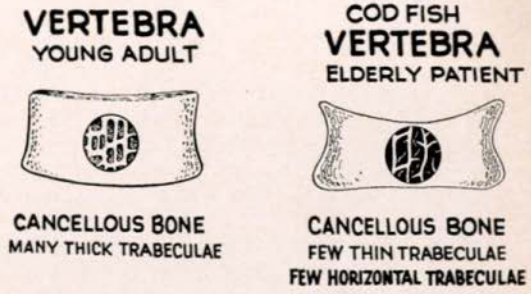

Fig. 3

Symptoms and Signs

Most patients complain of back ache and have local tenderness and are reluctant to move in bed in order to avoid further pain. A fair number have root pain and a much smaller group sustain injury to the spinal cord. About 15 per cent of patients develop paralytic ileus, particularly in association with fractures of T12 and L1. (Fig. 4) It is gen-

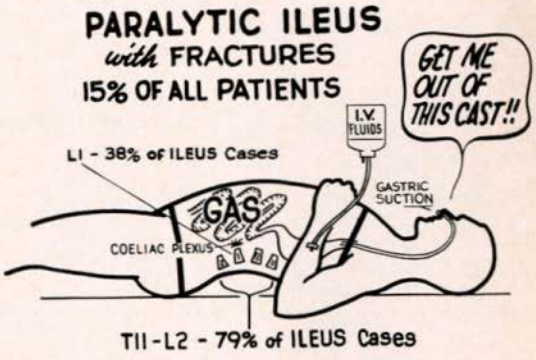

Fig. 4

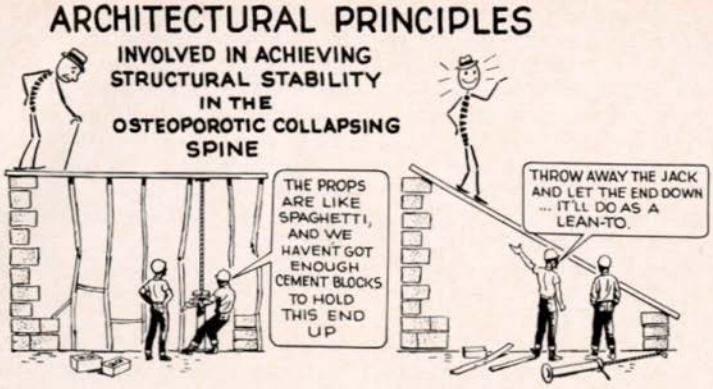

Fig. 7

erally considered that retroperitoneal hemorrhage is the underlying factor in the production of ileus, either by the size of the hemorrhage or irritation, disturbing the coeliac plexus. This plexus, through which motor impulses to the small bowel are metered is situated opposite the body of L1. Another possibility is a local pain reflex arc which may explain the increased incidence of ileus on extention. *Ileus may develop in 50 per cent of patients treated with hyperextension and only 11 per cent of the remainder.*[4]

Treatment

Management of the patient with complicated fractures including spinal cord injury is beyond the scope of this paper which is mainly concerned with simple compression fractures of the spine which are generally associated with osteoporosis.

The management of osteoporosis is discussed in considerable detail elsewhere in this Symposium and the general principles of treatment are summarized in Fig. 6. Some of these measures will be discussed very briefly.

(a) DIETARY: - Some investigators advocate a high-calcium diet (2.0 to 2.5 Gm. per day as calcium lactate or gluconate) because there is evidence that a low-calcium diet or malabsorption of calcium, or both may be important in the development of osteoporosis. However, until more complete information is available, one should probably ensure a daily calcium intake of at least 1.0 Gm. daily; this need is met by an average diet supplemented by one or 2 glasses of milk. If calcium lactate is used instead of milk one should remember that a 0.3 Gm. tablet contains only about 40 mg. of calcium.

Osteoporotic patients have been fed small amounts of fluoride which causes an increase in calcium retention after a lag of 2 to 3 months.[23] However, we do not know the long-term toxic effects of this element and more investigation is needed before fluoride can be confidently recommended as a safe and useful therapeutic agent.

(b) HORMONAL: - Symptomatic relief of bone pain, cessation of loss of height as an index of vertebral collapse, and a feeling of well-being have been reported following the use of estrogens and androgens in the treatment of spinal osteoporosis, but there is as yet no evidence of any correction of the skeletal disorder.

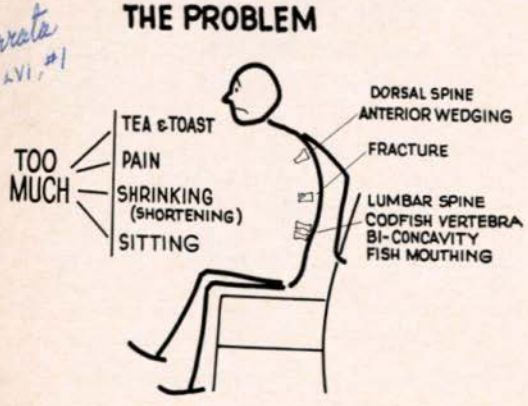

Fig. 5

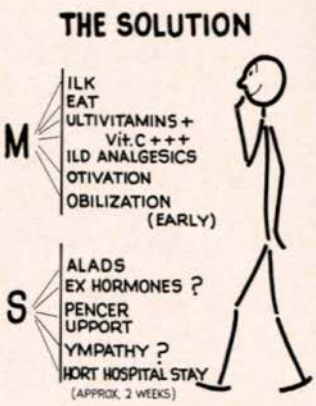

Fig. 6

(c) ORTHOPAEDIC MEASURES: - Little can be gained in either function or comfort by trying to obtain an anatomic reduction of the vertebral body fracture in the aged. The compressed state of the osteoporotic vertebra is the most stable (Fig. 7) and attempted reduction by hyperextension is unlikely to succeed and unnecessary. The high incidence of paralytic ileus in patients treated by hyperextension has been referred to earlier. (Fig. 4).

The patient with osteoporosis should be immobilized as little as the severity of his pain allows, and external supports should be kept to a minimum. A light, well-fitting support may be very useful, but hyper-extension plasters and heavy braces should be avoided.

(d) TREATMENT OF PARALYTIC ILEUS: - Intravenous fluids and gastric suction are used until bowel activity returns. One must avoid hyperextension.

Duration of Hospital Stay

In their study of 160 patients over the age of 60 admitted to the Massachusetts General Hospital with fractures of the spine, Rowe and Sorbie[4] pointed out the short period of hospitalization required in a large percentage of patients. (Table I).

TABLE I

Type of Spine Fracture	Percentage (etc) DISCHARGED Within 2Weeks
All Cases	76%
Uncomplicated Cases	91%

Discussion

When Browning[24] wrote of old age as "the last of life for which the first was made" he did not temper his philosophy by including some of the hard facts of aging described in Ecclesiastes:[25] ". . .the years draw nigh, when thou shalt say, I have no pleasure in them. . . . . *.and the strong men shall bow themselves*, and the grinders cease because they are few. . . . .when they shall be afraid of that which is high, and fears shall be in the way. . . . . .and the grasshopper shall be a burden, and desire shall fail: because man goeth to his long home. . . ." No modern description of aging and its problems can match this magnificent word-picture dating back to the 5th Century B.C.

We still do not know whether aging is simply the result of deliberate "biological programming" and cytoplasmic deterioration closely related to the total developmental history. Medicine has made considerable progress, but it is not increasing our life span; we are prolonging life a little but vigour hardly at all. Because of the altered composition of the "mushroom crowd" we are now confronted with the growing problem of the diseases of degeneration and the management of structural defects due to aging.

Summary and Conclusions

Compression fractures of the osteoporotic spine are common in the elderly patient. Most of these fractures are simple and can be treated simply (Fig. 6). Attempted reduction by hyperextension is unlikely to succeed, unnecessary and undesirable and very frequently associated with paralytic ileus. The compressed state of the hollow-shelled vertebral body is the most stable. The patient with osteoporosis should be immobilized as little as the severity of his pain allows and external supports should be kept to a minimum. Very few of these patients need to be in hospital more than two weeks. □

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MAN. P. 46

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Hyperparathyroidism

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Hyperparathyroidism or generalized osteitis fibrosa cystica as described in 1891 by von Recklinghausen can be either of the primary or secondary variety. The primary type accounts for about 85-95 per cent of all cases of hyperparathyroidism. In this article most of the space will be alloted to the commoner form and only brief mention will be made of the secondary type.

The author is abundantly aware of the fact that this article is primarily intended for the general practitioner, who is usually too busy to read the controversial material on this subject; yet, when indicated, it is essential that he have enough relevant information at his fingertips so as to have a high index of suspicion, and then to conduct the pertinent investigations in a logical fashion. With this in mind, the author is compelled to make a few statements which may appear dogmatic, but which represent the opinion of the majority of respected physicians in this field of medicine.

A not too extensive bibliography is included to serve as a ready source of reference for the physician who may be interested in further expanding his knowledge of this disease.

PRIMARY HYPERPARATHYDOIDISM

Though rare, the exact incidence of this disease is unknown. There is a female preponderance (3:1)[1,2,3] and the majority of cases are diagnosed during the third to fourth decades. A familial incidence is rare.[4]

About 90 per cent of the cases are due to adenoma. Ninety percent of these involve only one of the four parathyroid glands, the remaining 10 percent, multiglandular adenomata. Hyperplasia of one or usually more than one gland is rather infrequent, and less than 1 percent is due to primary carcinoma.[5] The syndrome of multiple endocrine adenomatosis (parathyroid, pituitary, and pancreas) giving rise to hyperparathyroidism is rare indeed.[6]

Clinical Features

Symptoms

It should be underlined that the late diagnosis of hyperparathyroidism after the appearance of cystic bone changes and multiple renal stones should be a thing of the past. There are numerous clinical clues that, when carefully evaluated, should suggest the diagnosis much earlier in the course of this disease.

The main symptoms are:

1. *Skeletal* Historically, skeletal symptoms were the first to be described in this disease. Today, due to earlier diagnosis, these are much less common and consist of: - vague local or generalized bone pain, bony enlargements due to cysts which are commonest in the mandibles, skeletal deformities and pathological fractures. These bone changes are most likely due to the effect of parathormone on the skeleton.

2. *Renal* Clinical features of renal disease are by far the commonest manifestations of hyperparathyroidism[2,3]. About 5 percent of all urinary calculi and 15-60 percent of recurrent calculi are on this basis.[2,5] Even without known calculi, the presence of painless hematuria, recurrent urinary tract infections, or any picture of chronic renal failure may indicate nephrocalcinosis due to hyperparathyroidism.[8]

With chronic renal failure, polyuria, nocturia, polydypsia, and occasionally a pitressin-resistant diabetes insipidus-like syndrome can occur. The pathophysiological basis for these symptoms can be explained as follows. The hypercalcemia *per se* has a slight diuretic effect[9] and persistent hypercalcemia causes damage to that area of the kidney responsible for concentration of urine[10]. This leads to polyuria, nocturia, hyposthenuria, and polydypsia. The defect in the renal concentrating mechanism is reversible in its earlier phases but, if hypercalcemia persists, chronic renal failure eventually occurs manifested by uremia, hypertension in about

From the Department of Medicine, Halifax Infirmary.

50 percent of cases and normocytic normochromic anemia.

3. *Gastro-intestinal* The incidence of peptic ulceration associated with hyperparathyroidism varies from 9-25 percent.[2] Manifestations of peptic ulcer may precede other clinical features of hyperparathyroidism. Frequently, these features may disappear following surgery to the parathyroid gland, but peptic ulcer may recur.[12]

Pancreatitis is occasionally associated with this disease, and it is most likely due to the hypercalcemia *per se* since other causes of hypercalcemia are not infrequently associated with pancreatitis. Other gastro-intestinal symptoms include: - a bitter metallic taste, anorexia, nausea, vomiting and constipation. These are most likely due to hypercalcemia. Moreover, it has been fairly well documented that the symptom-complex of hyperparathyroid crisis: - acute onset of intractable nausea and vomiting, constipation, abdominal pains, circulatory collapse (focal myocardial necrosis), oliguria with rapidly progressive azotemia, lethargy, confusion, disorientation, deepening stupor and coma - is due to the markedly elevated serum calcium which may be in the range of 17-22 percent.[2,4,13-15]

4. *Neuropsychiatric* The clinical picture is usually that of apathy, delusions, hallucinations, mental sluggishness, often culminating in progressive confusion, coma, and even convulsions. These develop over the course of a few months in previously well-adjusted individuals, and are most probably due to the hypercalcemia. In such cases the serum calcium is usually above 13 mg percent and with much higher levels hypercalcemic crisis develops as noted above.[2,4,13-15]

Physical Examination

Usually there are no positive physical findings. Moreover, even when these are present they lack specificity.

Bony deformities and tenderness, bone cysts, and pseudoclubbing due to resorption of the terminal phalanges are sometimes present. Hypertension which is common[8] may or may not subside to within normal limits depending upon the magnitude of the renal damage.[16] Band keratopathy manifesting as whitish opaque bands on the lateral margins of the cornea is due to hypercalcemia. In the absence of obvious band keratopathy, slit lamp examination often reveals whitish opacities in the anterior chamber of the eyes.

Though uncommon, a palpable mass in the neck due to parathyroid tumour can sometimes be palpated.[2] Parathyroid carcinoma is more often palpable than is adenoma, and in the presence of a palpable tumour, carcinoma should be suspected.[5] In my own limited experience, careful palpation of the neck has been highly rewarding in one case of parathyroid adenoma which could not be demonstrated on barium swallow, and which was subsequently verified at surgery. It must of course be appreciated that most of these so-called "palpable parathyroid tumours" turn out to be thyroid adenomata.

Roentgenology

Frequently no skeletal abnormalities are demonstrable; about 25 percent of cases show no recognizable changes and only in about 33 percent of cases are the changes diagnostic. However, as discussed elsewhere in this bulletin, osteoporosis though present, may not necessarily be visualized on an X-ray film. Nevertheless, the following abnormalities are frequently found[17] and are most probably due to parathormone excess: -

1. Skeletal: - A spectrum of bone lesions are found and these include:
   (a) Subperiosteal and endosteal bone resorption, characteristically of the phalanges (middle), pubic rami, and end of clavicles.
   (b) Generalized osteoporosis.
   (c) Osteitis fibrosa generalisata is found in very advanced cases.
   (d) Osteoclastomas or giant cell tumours in mandible or calvarium.
   (e) Absence of lamina dura. This is very nonspecific and usually of no help.
2. Renal: - Nephrocalcinosis or calculus formation occur frequently as already mentioned. Some investigators teach that patients with nephrocalcinosis do not have nephrolithiasis, but this is not necessarily so. A flat plate of the abdomen is usually enough to demonstrate nephrocalcinosis. Only rarely is nephrotomograph indicated.
3. Tumour: - Barium swallow, especially with the aid of cinefluoroscopy will reveal indentations of the contrast medium along the esophagus. If, in case of hyperparathyroidism, this radiological abnormality is demonstrated, it is of great help to the surgeon in localizing the tumour.

Laboratory Investigation

This aspect of hyperparathyroidism is undoubtedly the most controversial in this disease. However, I will stress what I consider the most useful chemical methods of investigation.

The main criteria of diagnosis are:

Hypercalcemia
Hypercalciuria
Hypophosphatemia
Hyperphosphaturia
Elevated alkaline phosphatase and urinary hydroxypholine

By far the most important single criterion is hypercalcemia. In fact, for all practical purposes, the diagnosis of hyperparathyroidism must never be made in the absence of an elevated serum calcium, however other causes of hypercalcemia must be constantly kept in the forefront.[18] Frequently the elevation in serum calcium is unequivocal (normal is 9-11 mg per cent) but occasionally

minimal elevations of 10.8-11.5 mg per cent on repeated determinations may be the only clue. On the other hand, normal serum calcium levels have been reported in this disease. Renal insufficiency will cause lowering of the elevated serum calcium.

The 24-hour urinary excretion of calcium is elevated. It must be stressed that the interpretation of this parameter must take the following facts into consideration: -

(1) Calcium intake: The daily excretion can vary widely depending upon calcium intake. Nevertheless, on the average North American hospital diet, the 24 hour excretion of calcium is usually more than 250 mg. in patients with hyperparathyroidism. On a known calcium intake, about 50 per cent of these patients excrete more than their intake.[2]

(2) Accurate 24-hour urine collection: This may sound as a very simple procedure. In fact, it is. However, in the author's experience (at least in hospitals in Halifax and Montreal) the urine collection is frequently not accurate. One usually overcomes this by informing the patient about the importance of this procedure and holds him responsible for saving every drop of urine.

(3) Renal insufficiency will reduce urinary excretion of calcium.

Serum inorganic phosphorus (normal 3-4.5 mg) is almost invariably lower than normal. In the presence of renal insufficiency, a normal or even mildly elevated inorganic phosphorus may be found.

Many investigators have attached great significance to tests based on renal excretion of phosphorus To the general practitioner, these tests leave a lot to be desired and often add unnecessary confusion.

Management

Once the diagnosis of primary hyperparathyroidism is made, surgical removal of the tumor (usually an adenoma) becomes mandatory. In the case of hyperplasia removal of all except a portion of one gland is usually necessary. In those cases where surgery is delayed the calcium intake must be stringently restricted and generous fluid intake encouraged.

Following surgery, hypocalcemic tetany is occasionally encountered. It should be suspected when bone lesions are prominent and alkaline phosphatase significantly elevated. Treatment of this complication can best be discussed under three headings:

Prophylaxis

Acute hypocalcemia

Chronic hypocalcemia - a very rare occurrence, and will not be discussed further.

Prophylaxis If tetany is predicted from features as noted above, therapy revolves around generous dietary calcium, calcium lactate, and calciferol (vitamin D_2) or dihydiotachycholesterol (AT-10). Within one week adequate serum calcium level is usually attained. To assess adequacy of therapy 24-hour urine calcium or Sulkowitch test for urine calcium, and serum calcium must be done at frequent intervals. The appearance of calcium in the urine with the Sulkowitch test indicates a serum calcium of about 7 mg%.

Acute Hypocalcemia This constitutes an emergency and demands immediate therapy. 10 ml. of 10 percent calcium gluconate given I. V. corrects the hypocalcemia, but the effect lasts for only a few hours and additional doses are usually indicated. Concurrently, calciferol in doses of 50,000 to 150,000 units per day is also used. Evaluation of adequate therapy is assessed as noted above.

SECONDARY HYPERPARATHYROIDISM

This accounts for 10-15 per cent of all cases of hyperparathyroidism. By far the commonest cause is long-standing renal disease, viz: pyelonephritis and glomerulonephritis. Other forms of chronic hypocalcemia in the presence of intact parathyroids can also produce this disease.

The relevant pathophysiology in this form of hyperparathyroidism is as follows.[19-20] Long-standing pyelonephritis or glomerulonephritis leads to a negative calcium balance due to decreased absorption of calcium. The resultant hypocalcemia stimulates the parathyroids to produce parathormone. It is controversial whether or not phosphorus retention plays a part. Despite the increased parathyroid activity as evidenced by the significant hyperplasia of all four parathyroid glands the calcium balance remains negative and the serum calcium subnormal, though somewhat increased as compared to previous values. When serum calcium values are normal in this setting the possibility of primary hyperparathyroidism should always be suspected.

In an attempt to counteract this abnormal physiology of calcium metabolism, numerous investigators [19-21] have used very large doses (200,000-600,000 units) of vitamin D daily, and occasionally, subtotal parathyroidectomy[20] has been performed with very good results.

The clinical features are as discussed under primary hyperparathyroidism. In addition, features of the primary disease (pyelonephritis, glomerulonephritis) dominate the clinical picture.

Diagnosis

In contradistinction to the hypercalcemia, hypercalciuria, and hypophosphatemia of primary hyperparathyroidism, in the secondary variety, the serum calcium is subnormal or rarely normal, the 24-hour urinary excretion of calcium is very low (50-100 mg. per day), and the serum inorganic phosphate is invariably markedly elevated. The other biochemical features are evidences of severe chronic renal insufficiency. Roentgenographic features are as mentioned above.

continued on page 316

Diseases of the Kidney

Associated with States of Bone Demineralization

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The kidney is intimately concerned with calcium homeostasis mediated through parathormone and Vitamin D levels either directly or by virtue of the resultant calcium load. Urinary calcium is also increased in hyperthyroidism and hyperadrenocorticism. The sources for this urinary calcium are the gut and the skeleton. It is not surprising, therefore, that diseases of kidney and diseases of bone are closely linked and that it is sometimes not readily apparent which is the chicken and which the egg.

Ionized calcium passes the normal glomerular filter freely and is 99 per cent absorbed by the normal proximal convoluted tubule. Calcium complexed with other ions such as citrate, phosphate, sulfate or E.D.T.A. is also freely filterable but is poorly reabsorbed. Protein bound calcium is not normally passed by the glomerulus. There is no evidence of tubular excretion of calcium nor is there any evidence for a set level for maximal tubular reabsorption. (Tm_{ca}).

The homeostasis of phosphorus is quite different. The partition of acid and basic phosphates is one of the main factors in the maintenance of acid-base equilibrium, the tubular reabsorption reaches a maximum at approximately 150 μ mols per minute beyond which the excess is excreted (the actual level of maximal tubular reabsorption being under the direct control of the parathyroids) and, in addition, it is possible that actual secretion of phosphate by the tubules may take place.[1]

Although the regulatory mechanisms differ somewhat, the renal excretion both of calcium and of phosphate therefore is dependent on the plasma levels of these substances, on the glomerular filtration rate and on the integrity of tubular function as well as on hormonal regulatory mechanisms.

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Bone and the Kidney

PLASMA LEVELS: - States of bone demineralization associated with hypercalcemia and hypercaliuria constitute a recognizable cause of renal calculi and calcinosis. Thus renal calculi are common complications of primary hyperparathyroidism, Vitamin D poisoning, sarcoidosis, myelomatosis and Paget's disease as indicated elsewhere in this symposium. They are less commonly seen in cancer metastatic to bone and in hyperthroidism and hyperadrenocorticism. The tendency to calculus formation is well recognized in the osteoporosis of immobilization induced by fractures, poliomyelitis and paraplegia in which stasis and infection are added to hypercalciuria.

Renal infection tends to lead to renal lithiasis, and even more so, renal calculi from any cause are heavily contributory to pyelonephritis. Owing to a feed-back mechanism this resultant source of renal impairment if it interferes with tubular function may cause further stimulation of parathyroids, worsening of the primary condition and increase in the load presented to the kidney.

Treatment Early treatment of primary bone disease, if possible, therefore is indicated. This aspect has been covered already in the sections on hyperparathyroidism and on physiotherapy. In addition, it is wise to force fluids to two liters daily, limit calcium and Vitamin D intake and attempt a decrease of the gastrointestinal absorption of calcium by the administration of Amphogel 25-50 ml., q.i.d.[2] or sodium phytate such as Renical (Squibb) 3 gms., t.i.d.[3]

Prophylaxis or treatment of urinary infection should include chemotherapeutic or antibiotic agents dependent on the nature of the organism isolated. Chelating agents such as E.D.T.A. have been tried.[4]

The Kidney and Bone

I Glomerular Disease (*Impaired Glomerular Filtration Rate*)

In diseases of the glomeruli, either glomerulonephritis or hypertensive nephrosclerosis, disturbances of calcium and phosphate homeostasis do not occur until glomerular filtration has fallen to 25 per cent or less. This implies severe azotemia with phosphate retention and reduced serum calcium leading to secondary hyperparathyroidism, metabolic acidosis, and the disorder known as *renal osteodystrophy*. Uremia from any cause, even though not primarily glomerular in origin, such as chronic pyelonephritis, congenital hypoplasia, or bilateral hydronephrosis may lead to this condition. Either osteitis fibrosa or osteomalacia or both together develop in a minority of patients. There may be renal lithiasis or metastatic calcification, usually confined to the kidneys.

Treatment Disease of bone is a minor facet of chronic uremia and therapy will mainly be directed towards alleviation of the kidney ailment. Extra Vitamin D may be required and the acidosis should be corrected. One would question whether parathyroidectomy is indicated in these terminal patients.

II Tubular Disease (*Integrity of Tubular Function*)

In contrast to glomerular disease, chronic disorders primarily affecting the tubules tend to lead either to hyperphosphaturia and resultant hypophosphatemia or to hypercalciuria. In either case osteomalacia or "renal rickets" develops without secondary hyperparathyroidism unless there is uremia.

A number of these disorders are genetically determined and are grouped loosely under the term **Fanconi Syndrome** but it has become evident that not only are there many variants of this condition including cystinosis (Lignac-Fanconi Syndrome), cerebro-ocular-renal dystrophy (Lowes Syndrome), some cases of renal tubular acidosis (Butler-Albright Syndrome, Lightwood's Syndrome) and some forms of Vitamin D resistant rickets, but also on occasion the condition appears to be secondary to some other disorder and is seen in some cases of heavy metal poisoning, the use of out-dated tetracycline, Wilson's Disease, von Recklinghausen's Disease (neurofibromatosis) and multiple myeloma; all however are accompanied by phosphate diabetes together with defective tubular resorption of one or more aminoacids, glucose, potassium or sodium. Simple aminoacidurias, on the other hand, such as phenylketonuria and maple syrup urine disease are not complicated by phosphate diabetes or bone lesions.

Out of this rather heterogeneous mélange a number of broad groupings can be drawn, though as indicated above there is considerale overlapping in individual cases.

(a) deToni-Fanconi Syndrome -

As in the case of Trousseau and von Recklinghausen, Fanconi's name is applied to two quite unrelated syndromes; for this reason the name of de Toni should be included in the eponym of the renal tubular anomaly.

This syndrome presents in children and occasionally in adults as rickets or osteomalacia accompanied by renal glycosuria, phosphaturia and polyaminoaciduria, the pattern being that of the blood partition rather than of a simple amino-acid as in phenylketonuria and other metabolic aminoacidurias without phosphaturia. In one variety there is generalized deposition of cystine crystals throughout soft tissues (Lignac-Fanconi Syndrome). This must be sharply differentiated from the relatively innocuous condition known as cystinuria in which there is massive excretion of dibasic amino acids without the deposition of these in the tissues or phosphate diabetes. The Fanconi Syndrome may be accompanied by Vitamin D resistance or hyperchloremic tubular acidosis or by glaucoma, cataracts and mental retardation. In so heterogeneous a collection of signs and symptoms it would indeed be unreasonable to expect a common pathogenic lesion but such appears to be the case. In nearly all cases which have been studied adequately by microdissection a peculiar attenuation of the first part of the proximal convolute, known as a "swan-neck deformity" has been found.[5] The remainder of the proximal tubule is short and truncated. This lesion has been found not only in the kidneys of affected infants but also in adult patients who have developed the "acquired" condition late in life. Unfortunately, detection of this specific abnormality requires meticulous teasing out of indivisual nephrons under the dissecting microscope and this is not possible in needle biopsies taken during clinical practice as not only does the needle cut through the nephron in a haphazard manner but also the technique of microdissection is one for the research laboratory.

Treatment The loss of amino acids, though constant, is not great and there is no need to replace these. Calcium, phosphate and potassium levels in the blood should be maintained by supplements depending on laboratory findings. This is particularly important prior to glucose tolerance tests because of the danger of acute hypokalemia.

The primary anatomical abnormality is beyond our therapeutic armamentarium and clearly will remain so but as these patients are susceptible both to metabolic urinary calculi and to urinary infection it is important that infection be kept under control and that the diet be low in cystine. For correction of acidosis intravenous sodium bicarbonate or M/6 sodium lactate may be required as an emergency measure but maintenance of Shohl's solution *per os* is preferable. If there is concomitant hypoka-

lemia this solution may be modified as follows:

℞. Citric acid - 140 gms.
Sodium citrate - 75 gms.
Potassium citrate - 25 gms. Distilled water to one liter.
Sig. 2 to 6 oz. daily as required.

Alkali therapy is a two-edged sword, it may increase the tendency to form calculi or, if continued too long, the patient may slip into alkalosis. I well recall a patient who was placed, perhaps inadvertently and certainly without requisite concern, on two bottles of M/6 lactate daily as a routine measure. He lapsed into a moribund alkalosis requiring two litres of N/10 HCl intravenously to correct it.

These patients are losing water along with the other urinary constituents and infants rapidly become dehydrated. Death may follow a minor infection.

(b) Vitamin D. resistant rickets -

If Vitamin D therapy is long continued, as in primary hypoparathyroidism, the osteomalacia of steatorrhea or in renal failure, it may tend to lose its effect and the patient may become Vitamin D resistant to the specific preparation employed. Such persons usually respond, to a different form, such as dihydrotachysterol. There are, however, other forms of non-response to Vitamin D which appear to be genetically inherited either as an autosomal or sex linked dominant in 2/3 patients.[6]

One such form represents a variant of the Fanconi Syndrome and is a multisystem disorder apparently due on the one hand to a defective response of the G.I. tract to Vitamin D resulting in deficient absorption of calcium and on the other to abnormal renal tubular function resulting in aminoaciduria, hyperphosphaturia and hypophosphatemia, and, in some cases, renal glycosuria or fluid and electrolyte imbalance. Defective calcium absorption leads to secondary hyperparathyroidism which intensifies the hypophosphatemia.

In other cases there are no stigmata of the Fanconi Syndrome, no acidosis and no abnormality of the serum chemistry apart from hypophosphatemia and (usually) greatly elevated alkaline phosphatase levels. In these patients it would appear that the renal tubular defect is confined solely to a decreased ability to conserve phosphorus though there may be a concomitant intestinal malabsorption of calcium and phosphorus as in the first type.

Diagnosis Primary Vitamin D resistance usually appears in infancy and, if the other manifestations of tubular dyscrasia are not detected, patients may be considered to be suffering from simple dietary rickets. They do not respond however to prophylactic doses of Vitamin D, requiring much larger doses to prevent deformity. General health is otherwise little impaired and infants survive to adult life when signs of active osteomalacia appear again.

Treatment Whereas 1,000 to 10,000 I.U. of Vitamin D_2 (Calciferol) are usually adequate in the treatment of simple dietary rickets, in Vitamin D resistant rickets quantities of 50,000 to 150,000 units are required. These are within the range of proven toxicity in Vitamin D non-resistant persons. Unfortunately Vitamin D resistance refers to non-response of the skeleton to Vitamin D and in these patients therapeutic and toxic doses are uncomfortably close.

The aim of therapy is to restore serum phosphorus levels to normal without evoking hypercalcemia or hypercalciuria and renal calculi. Each case must be treated on an individual basis without a rigid rule-of-thumb, reducing Vitamin D therapy so far as possible on restoration of normal or near normal calcium, phosphorus and alkaline phosphatase levels. As soon as there is radiological evidence of healing serious consideration should be given to reduction of Vitamin D administration closer to the normal therapeutic levels keeping in mind that Vitamin D resistance persists throughout life.

If there are gross deformities of the limbs, osteotomy may be considered even before radiological healing is complete. The surgery and post-operative immobilization, according to Kajdi[7] may actually accelerate the return of phosphatase levels and radiologic findings to normal.

In view of the hereditary nature of Vitamin D resistance, the children of affected families should be checked for hypophosphatemia, hypercalcemia, raised alkaline phosphatase and amino-aciduria at regular intervals as a prophylactic measure.

(c) Renal Tubular Acidosis -

This may complicate the Fanconi Syndrome or occur without significant amino-aciduria. The condition has been described at all ages but is more common in infants and children. The fundamental defect appears to be an inability of the kidney to form ammonia or to secrete an acid urine leading to gross bicarbonate loss and hyperchloremia. As there is often vomiting associated with the condition it is readily complicated by loss of fluids and electrolytes particularly sodium, potassium, calcium and phosphate. Hypochloremia may develop together with gross dehydration. There may be periodic muscle paralysis (hypokalemia) or tetany (hypocalcemia). In the later stages there is a peculiar type of medullary calcification of the kidney resembling bristles.

Probably some cases represent a congenital tubular anomaly but this is not apparent in histological sections except in cases complicating the Fanconi Syndrome. Perhaps electron micrographs or wider use of histochemistry in this condition may reveal the fundamental cellular problem. Adult cases are constantly associated with pyelonephritis; this would appear to be the cause rather than the effect.[8]

Diagnosis The laboratory diagnosis rests on an inability to produce acid or concentrated urine, reduced alkali reserve, raised serum chloride, normal or low serum calcium or potassium, low serum phosphorus and high urinary phosphorus and calcium. Alkaline phosphatase is normal or elevated. Skeletal X-rays show the changes of osteomalacia or rickets and bristle nephrocalcinosis.

Treatment: Both infantile and adult cases respond to alkalinizing therapy particularly if supplemented by concomitant Vitamin D in therapeutic doses (1,000 - 10,000 I.U. Calciferol q.d.) and replacement of the calcium or potassium deficit.

(d) Hypophosphatasia

Although, in common with most of the other renal tubular dyscrasias it is a hereditary disorder, alkaline phosphatase deficient rickets differs in that the other features of the Fanconi Syndrome or its variants are not shown. The deficiency of alkaline phosphatase involves not only the serum but also the tissues of kidney, liver, spleen, lungs, intestinal mucosa, bone and polymorphs. It is accompanied by severe rachitic or osteomalacic changes in the skeleton, retarded growth, hypercalcemia, renal calcification and the presence of phosphoethanolamine in the urine. There may be renal failure.

The severity of the condition is in inverse ratio to the age of onset; only in infants or neonates are there severe skeletal defects or extensive renal calcification with hypercalcemia up to 15 mgms. per cent. At this age it is rapidly fatal. If the condition does not develop in the first six months, there may be no hypercalcemia, renal abnormality or growth retardation and if it makes its appearance later in childhood or in adult life, the level of alkaline phosphatase may be the sole abnormality. In these surviving patients the tissue defect of alkaline phosphatase persists throughout life.

Diagnosis Osteomalacic changes associated with an isolated deficiency of serum alkaline phosphatase are almost diagnostic. In all other osteomalacic states phosphatase is either normal or elevated and serum phosphate is abnormal. The diagnosis can readily be confirmed by finding a low score of leukocyte alkaline phosphatase or, less easily, by demonstrating negligible alkaline phosphatase in needle biopsies of liver, kidney or bone marrow by histochemistry. Routine sections of bone are almost indistinguishable from classical rickets or osteomalacia.

Treatment Vitamin D therapy has no effect on the rickets. Fraser and Laidlaw[9] have found cortisone therapy helpful in producing healing of bone. Apart from this nothing affects the phosphatase levels. Fortunately the defect produces little morbidity in most patients.

Summary

Bone and kidney, the kidney and bone, they are intimately associated with one another. We all appreciate that states of demineralization of bone, particularly in immobilization, are associated with renal infection and calculus formation. In addition, there is an interesting though rare group of renal disorders mainly but not entirely hereditary and familial in which lesions of the tubules inapparent by means of routine pathologic examination are associated with upsets in calcium, phosphorus or phosphatase balance producing profound changes in the body's economy. To a degree we can treat these disorders on a symptomatic level but we remain, in most cases, ignorant of what exactly has gone wrong. The answer perhaps lies within the field of competence of the electron microscope or within the area of cytogenetics which has been relatively unexplored in this regard. □

Acknowledgement in the preparation of this paper is given to Drs. H. B. Sabean and W. Nicholas for helpful comments and encouragement in deciding the criteria to be observed and the therapeutic regimens to be followed.

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Metabolic Bone Disease

and

The Gastro Intestinal Tract

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Osteomalacia (defective bone mineralization) and Osteoporosis (decreased skeletal mass) may be due wholly or in part to inefficient absorption of calcium and Vitamin D from the gastro intestinal tract. The primary patho-physiological defect in most cases is a failure in the intestine to adequately absorb dietary fat. Calcium combines with this intraluminal fat and forms unabsorbable calcium soap complexes. Furthermore, Vitamin D which is fat soluble is not absorbed and this results in a deficiency of two of the essential constituents required for normal skeletal homeostasis. If uncorrected this gastro intestinal loss of calcium and Vitamin D results in the development of metabolic bone disease. Osteomalacia can occur in such circumstances in the absence of tetany probably because secondary hyperparathyroidism maintains the serum ionized calcium at the expense of the skeleton.

If a malabsorption syndrome is suspected proper diagnosis and treatment requires an understanding of the altered physiology of fat digestion and absorption. We now recognize that the major fraction of dietary fat is in the form of triglycerides which are emulsified by conjugated bile salts and subsequently hydrolyzed by pancreatic lipase to monoglycerides and free fatty acids. Fat is absorbed in this latter form and reconstituted to triglyceride within the intestinal mucosal cell. It is then secreted into the mesenteric lacteals and transported in the form of chylomicrons to the thoracic duct. Recent evidence suggests that synthetic median chain triglycerides are in part absorbed directly into the portal venous system without prior hydrolysis. It is now obvious that altered fat absorption will occur if there is defective secretion of bile salts, inadequate amounts of pancreatic lipase, or a gross anatomical lesion of the intestine or morphological abnormality of the mucosa.

There are numerous causes of the malabsorption syndrome but for practical purposes those associated with metabolic bone disease are the following: -

Gluten enteropathy (celiac disease)
Chronic biliary obstruction
Surgical derangement of the gastro intestinal tract
Pancreatic disease.

Gluten enteropathy occurs in both childhood and adult life, 60 per cent of cases in adults having a history of diarrhoea in childhood. It is probably a genetically determined disease in which the afflicted individual has a hypersensitivity to the gluten fraction in wheat. The diagnosis is established by demonstrating total or sub-total villous atrophy in intestinal biopsy material and confirmed by improvement of the patient on a gluten free diet (see Table I). Dietary exclusion of gluten must be as strict as possible. Failure to respond in established cases is usually due to inadequate gluten restriction but occasional cases have an associated milk allergy and improvement only occurs when milk is eliminated from the diet. In celiac disease with osteomalacia calcium absorption is resistant to massive doses of Vitamin D but is restored toward normal with a gluten free diet. It is therefore important to recognize such cases because treatment with calcium and Vitamin D alone will not halt the progress of bone demineralization.

Biliary obstruction if prolonged as is the situation in biliary cirrhosis will result in a negative calcium balance and subsequent bone disease. Such patients are easily recognized clinically by the presence of jaundice and pruritis. The serum alkaline phosphatase is invariably elevated to very high levels because of the combination of increased bone osteoblastic activity and decreased biliary excretion of the enzyme. All patients with chronic biliary obstruction require an abdominal laparotomy and

From the Department of Medicine, Halifax Infirmary.

TABLE I

GENERAL PRINCIPLES IN DIETARY GLUTEN RESTRICTION

Ref: Massachusetts General Hospital, *New Eng. J. Med.* 271: 1156, (1964)

FOODS EXCLUDED

Any food containing wheat, wheat flour, rye, barley or malt. Careful efforts to avoid foods that frequently contain "concealed" gluten, including meat loaves, canned-meat dishes, frankfurters and cold cuts, beer, and ale, commercial salad dressings, most commercial ice creams and foods prepared with bread and cracker crumbs.

FOODS ALLOWED

Beverages - milk, tea, coffee, carbonated beverages, cocoa (read labels on cocoa and instant coffee) and whiskies.

Bread - only those made from rice, corn, arrowroot, soybean, potato and gluten-free wheat-starch flours.

Cereals - cornmeal, oatmeal, rice and puffed rice.

Cheese - all types of pure cheese.

Desserts - gelatin, ices, homemade ice cream and custard, rice pudding and cornstarch pudding, cakes, cookies and pastries prepared with low-gluten flours.

Eggs - permitted.

Fats - butter, margarine, salad oil, vegetable shortening, lard, bacon, pure mayonnaise, nuts and peanut butter.

Fruits - as desired.

Meat-fish-poultry - as desired (rice, soy, potato or cornmeal flours may be used in "breading" or in preparation of stuffing or gravies).

Soups - all clear soups, most vegetable soups made with clear stock and homemade cream soups and chowders thickened with cream, cornstarch or potato flour.

Sweets - sugar, molasses, honey, jam, jelly, corn syrup and maple syrup.

Vegetables - potatoes and all other vegetables as desired (sauces may be thickened with cream or cornstarch or allowed special flour).

cholangiogram to exclude a surgically correctable bile duct obstruction. If such a lesion is excluded treatment is entirely symptomatic and consists of a low fat diet (less than 40 grams), calcium gluconate 6 grams daily and Vitamin D 100,000 units by intramuscular injection once monthly. Vitamin A 100,000 units and Vitamin K 10 mgms. should be given with the Vitamin D injection because of the associated deficiency of these vitamins in biliary obstruction. Bile salt replacement would seem to be the logical therapy in biliary obstruction but a satisfactory preparation is not presently available: current preparations frequently cause diarrhoea and in the presence of bile duct obstruction their absorption will intensify the pruritis which is invariably present. As mentioned earlier synthetic median chain triglycerides with a carbon chain length of less than 10, bypass the usual pathway of fat obstruction and enter the portal venous system rather than the mesenteric lacteals. They also undergo more rapid intraluminal hydrolysis and absorption has been demonstrated in situations where bile and pancreatic secretions are decreased. A diet containing median chain triglycerides may therefore have value in future management of patients with biliary obstruction or pancreatic insufficiency.

Steatorrhea of varying severity often follows a subtotal gastrectomy with gastrojejunostomy but is an uncommon complication of more conservative operations such as a Bilroth I procedure or pyloroplasty with vagotomy. The steatorrhea which occurrs in this situation is a result of a combination of factors. Impaired mixing of pancreatic and bile secretions with ingested food and rapid intestinal transit time account for the majority of cases but an occasional patient will have steatorrhea resulting from afferent loop stasis with bacterial overgrowth in the upper small bowel.

Osteomalacia developing in post-gastrectomy patients is very resistant to therapy. Oral calcium and parenteral Vitamin D may relieve bone pain but it is unlikely that bone structure will return to normal. Although the exact mechanism of calcium absorption is unknown it is reasonable to postulate that a significant loss occurs from chelation with unabsorbed fat. Pancreatic enzymes will improve fat absorption and replacement with a preparation such as Cotazyme, three capsules with each meal may be effective in halting the progress of bone demineralization. If the clinical situation indicates bacterial contamination of the afferent loop a trial period on a broad spectrum antibiotic (tetracycline, 1 gram daily for 10 days) may produce a dramatic decrease in the amount of steatorrhea. Rarely an occasional post-gastrectomy patient will have progressive weight loss, malnutrition and bone disease in spite of intensive conservative management. In this situation it may be necessary to consider reconstruction of a Bilroth II to a Bilroth I procedure in order to establish normal bowel continuity.

In clinical practice malabsorption secondary to pancreatic disfunction is seen in fibrocystic disease of childhood, chronic relapsing pancreatitis and carcinoma of the pancreas. If untreated the defect in lipolysis and protein digestion causes marked weight loss and cachexia but Vitamin D absorption is not altered to the same degree that it is in other malabsorption syndromes. Consequently bone disease is rarely a predominant clinical feature. Treatment consists of the frequent administration of a pancreatic enzyme preparation. Because pancreatic lipase is only effective in an alkaline medium it is often beneficial to simultaneously administer an antacid. It is also noteworthy that pancreatitis both acute and chronic can occur as a complication of primary hyperparathyroidism. The finding of an elevated serum calcium level in the presence of clinical pancreatitis should alert the physician to the possibility of an underlying hyperparathyroid state. Treatment directed toward the parathyroid glands will often result in relief of the symptoms of pancreatitis.

The physician should be alerted to the possibility of malabsorption in all patients with a history of excessive weight loss associated with a normal or increased food intake. The following diagnostic procedures are of value in the practical assessment of such a patient: -

1. *24 hr. urine calcium* - On a normal diet a value of less than 100 mgms. indicates Vitamin D deficiency. If osteomalacia is secondary to a malabsorption syndrome urine calcium excretion is always decreased.
2. *Stool examination* - The passage of stools which are pale, bulky, greasy and of offensive odor is pathognomonic of steatorrhea. Microscopic examination of the specimen stained with Sudan III will confirm the presence of excess fat. Undigested meat fibers in the stool indicate a pancreatic lesion in the absence of rapid intestinal passage.
3. *Fat balance studies* - In borderline cases steatorrhea is confirmed by placing the patient on a 100 gram fat diet for three days and during this interval all stool specimens are collected. If the fecal fat content exceeds a mean of 7 grams in 24 hr. stearorrhea is present. Because of bacterial metabolism in the bowel it is unnecessary and unreliable to make separate estimations for "split," "unsplit" and neutral fat.
4. *Serum carotene* - This is mueh less sensitive than a fat balance study but is a useful screening test. Low values are obtained in greater than 60% of cases with steatorrhea.
5. *Oral glucose tolerance test* - This test is frequently helpful in distinguishing patients with pancreatic lesions from those with a primary intestinal defect. In celiac disease the glucose curve is often flat in contrast to pancreatic disease where a diabetic curve may be obtained.
6. *D-Xylose test* - Following the oral administration of 25 grams of the sugar, urine is collected for the subsequent five hours. Normal individuals excrete 5 grams of D-xylose in the urine while persons with intestinal malabsorption excrete less than this amount. In malabsorption states due to pancreatic disease D-xylose absorption and excretion is usually within normal limits. Results obtained with this test are not valid in the presence of hepatic, thyroid or renal disease.
7. *Radiological examination* - A flat film of the abdomen should be obtained before barium is ingested. The presence of pancreatic calcification indicates disease in this organ. In the absence of alcoholism such calcification may be a helpful clue to the presence of an underlying hyperparathyroidism. Flocculation, clumping and dilution of barium within the small intestine is common in malabsorption syndromes but is not helpful in distinguishing primary intestinal, from pancreatic, steatorrhea.
8. *Small bowel biopsy* - Intestinal specimens are obtained by per oral intubation with a special biopsy capsule. The technique is simple, safe and painless. Microscopic examination of biopsy material obtained by this method is the most helpful single procedure in the etiological diagnosis of a malabsorption syndrome.

In conclusion a malabsorption syndrome may be the primary cause of metabolic bone disease. An adequate history and physical examination in conjunction with the laboratory tests described above will often establish the correct diagnosis and permit institution of specific therapy. □

Appreciation

Pierre E. Belliveau

Doctor Pierre E. Belliveau, an able and devoted general practitioner, died on September 20th, 1966, after a prolonged and disabling illness. It was a heart-felt loss to Meteghan, his parish; to the Municipality of Clare, where he practiced efficiently for more than forty years; and to the medical profession to which he always gave honourable support.

Pierre Belliveau was born in Belliveau Cove on May 18th, 1894. Following his classical course and Bachelor of Arts Degree at St. Anne's College, Church Point, in 1917, he entered Dalhousie University in the Faculty of Medicine, where he received his Doctorate in 1922. In the same year he started his medical practice in Meteghan, Digby County, N. S., where he remained till his death.

He married Angèle Robichaud in October 1924, and they had six children: Mrs. Alyre Comeau (Mariette) in Yarmouth; Robert, medical practitioner in Meteghan; Mrs. Louis Comeau (Lorraine) in Comeauville; Gerald, medical practitioner in Yarmouth; Pierre, with the Extension Movement at St. Francis Xavier University and residing in Belliveau Cove; and Paul, Priest with the Eudist Order in Buffalo.

Besides his active medical practice, Dr. P. Belliveau had the qualities of a historian. Especially interested in the History of the Acadian People, he gave much time and effort to that field, and in 1954, St. Anne's College awarded him an Honorary Doctorate.

In Politics, he found time to fulfill adequately the post of Secretary of the Clare Liberal Association for several years. His efforts for the betterment of Clare, his willingness to help his fellow citizens, made him an appropriate candidate for Member of the Legislative Assembly. Elected in 1953, he was M.L.A. until 1963. Had it not been for his failing health, it was indeed felt that he had the qualifications for a Senatorship.

The Concelebrated Requiem Mass, presided by his son, Rev. J. Paul Belliveau, was surely a tribute of respect and high esteem for this man who manifested such charity and good will towards his patients. His many colleagues were honorary pallbearers at this impressive ceremony, Knights of Columbus were pallbearers to a true and faithful Knight, members of the local branch of the Canadian Legion gave a last salute to their honorary President, veteran of the first World War.

It is heart-breaking to realize that, at the age of seventy, such a charitable and devoted doctor who, in the entire municipality, was known never to have refused a call, is now unable - and will never be able - to answer the call, a duty which he so willingly and pleasantly performed. □

P. H. LeB.

Appreciation

Roberta Bond Nichols

It was with surprise and sadness that we learned of the death of Doctor Roberta Bond Nichols on October 27th last. Many of us knew that of late she had been gradually relinquishing some of her customary activities and was about to retire as anaesthetist from the Staff of The Halifax Children's Hospital after twenty-six years of service but few knew that these were prompted by impairment in health. We felt that she was systematically preparing for a long and richly deserved retirement. How wrong we were! But a few days of illness and a life full of meaning was brought to a close.

Roberta, as she was known to her friends, was a Halifax girl, a daughter of the Parsonage. Her father, Rev. George J. Bond was the Minister at Grafton Street Methodist Church, since 1925 St. David's Presbyterian Church. As a girl she attended Tower Road School, as her children were to do in later years, Halifax Ladies' College, and Mount Allison Academy. Coming to Dalhousie she received her B.A. degree in 1920, and in the Autumn of the same year began the study of medicine. She joined the largest class that Dalhousie had in its history of medical teaching up to that time. It contained a large number of veterans of World War I back from service overseas. It was a mature and serious minded group, but none more stable and reliable than Roberta Bond. She entered medicine with a sense of dedication and determination to meet every demand of its study and practice.

After graduation in 1925 she went to her mother's home, Newfoundland, to practice. In the course of time she returned to Halifax and married Edward W. Nichols, Ph.D., later Professor and Head of the Department of Classics at Dalhousie University. Professor Nichols was a scholar, a man of the finest character, who was a splendid teacher and a writer of elegant prose. Their home life was one of culture in its very best sense.

In 1939 Professor Nichols died after a short illness, and Roberta was left to face the world with four young children, three boys and a girl. Without hesitation she turned again to the practice of medicine for a livelihood. The beginning of World War II meant more and more work for every available civilian doctor and she did her full share. The Department of Anatomy needed help, hospitals needed anaesthetists, Roberta was on the job. How she managed these and many other activities as well as caring for the demands of a growing family, commanded the wonder and admiration of us all.

By the time the War was over she had an established place as a teaching and practicing anaesthetist, and still assisted in the Anatomy Department. Soon her family one by one would enter college, and it was a source of great happiness to her that they did well.

We shall always remember Roberta. She taught us many things. She showed us above all both the virtue and rewards of struggle; how even adversity to the brink of defeat can be turned into triumph. Through it all she "pursued the even tenor of her way", steady, reliable and cheerful.

Do you recall the words Bunyan put into the farewell of Mr. Valiant-for-Truth? I think Roberta would have repeated them:

> "'I am going to my Father's; and though with great difficulty I have got hither, yet now I do not regret me of all the trouble I have been at to arrive where I am. My sword I give to him that shall succeed me in my pilgrimage, and my courage and skill to him that can get it. My marks and scars I carry with me, to be a witness for me that I have fought His battle who will now be my rewarder!. . . . . So he passed over, and all the trumpets sounded for him on the other side." □

H.L.S.

Personal Interest Notes

As a tribute to the late **Dr. Roberta B. Nichols,** who so cheerfully edited this section for many years The Editorial Staff has decided that these notes should be omitted from this month's issue. It is hoped to renew publication of this section in the near future, when The Editorial Staff is able to appoint a new Personal Interest Notes Editor.

THE GRACE MATERNITY HIGH RISK FLOOR

A special floor of the Grace Maternity Hospital has been set aside for the long term care of high risk obstetric patients, such as those with uncontrolled diabetes, hyperemesis gravidarum, toxaemia and cardiac disease. A specialised team of consultant specialists and nurses has been made available for these patients. Any doctor in Nova Scotia may make arrangements to have a high risk obstetric patient admitted to this floor by contacting a member of the staff of the Grace Maternity Hospital. A full review of this new facility will appear in a later issue of the Bulletin.

INDEX TO VOLUME XLV

The Index of this present volume will be printed as a detachable centre page in the first issue (January) of the next volume.

ADVERTISER'S INDEX

NOVA SCOTIA GUIDE FOR PHYSICIANS IN DETERMINING FITNESS TO DRIVE A MOTOR - VEHICLE

Prepared by

The Committee on Traffic Accidents

The Medical Society of Nova Scotia

(N. S. Division C. M. A.)

1965

INDEX

The following report is based on the report of a Committee on "The Medical Aspects of Automobile Injuries and Deaths" of the American Medical Association. Liberal use has also been made of the format used in British Columbia. Many changes have been made in rewriting these standards in order to suit the local needs in Nova Scotia.

INTRODUCTION

Traffic accidents killed about 48,000 individuals in 1964 and injured about 1,500,000 according to the National Safety Council. In Nova Scotia fatalities are usually around 190 per year or more. It is the purpose of this guide to call attention to the areas in which the medical profession may be of help in combating this serious health problem. The guide has been prepared to help physicians in examining patients and to advise them on their ability to operate a motor-vehicle safely. Physical, mental and emotional states or disabilities likely to impair driving ability, are specifically enumerated along with the problems of temporary incapacities from medicaments and alcohol. The physical and mental demands of the task of driving vary greatly with the type of vehicle and the type of driving. For this report, 2 types of vehicles have been considered - a private motor vehicle and a commercial motor vehicle. The driver of a commercial or passenger transport vehicle, cannot usually choose the vehicle he will drive nor the route he will take. The driver of the private vehicle may in many cases select both the time of his travel and the route. The driver of the commercial vehicle will spend more hours daily in driving than most drivers of private vehicles. There is little similarity between the demands on the individual who drives half a mile to the store on a rural road with no intersections and the individual who drives in rush-hour traffic in a metropolitan area. The standards demanded must be very different. Human failure overshadows all other factors in the production of highway accidents. The human mechanism must be in good condition to cope with the split-second timing needed to manoeuver responsive high speed motor-vehicles. There is no doubt that poor judgment, impaired reaction time, faulty attitudes, emotional disturbances and physical disabilities including the impairment of the sensory organs, are basically responsible for most accidents. The physician is qualified by training to ascertain the physical, mental, emotional and physiological impairments of the individual. He is in a good position to evaluate these impairments in relation to safe driving abilities. Obviously it is not always easy to arrive at a decision in these cases. Variations in the severity of illness or impairments, variations in individual reactions and adjustments, and variations in individual response to drugs are significant factors in evaluating these cases. However, even in the realm of behaviour patterns the physician is in an ideal position to do effective counselling by translating scientific data into terms of lay understanding. Few patients realize the small amount of alcohol required to impair dangerously their driving ability, or that alcohol is involved in about 50 percent or more of automobile accidents with fatalities. Nor does the slightly inebriated driver realize that he materially increases the likelihood of a serious accident. A conscientious individual may inquire, "Doctor, is it all right for me to drive an automobile in my condition?" This situation usually is conducive to a receptive patient attitude if the advice may be bad news.

Frequently it may be necessary for an equally conscientious physician recognizing his responsibility for the safety of his patient and the public, to caution the patient against driving for a certain period of time, or even permanently, unhappy as this may make the patient.

Many of these conditions have been graphically described for the lay public in various publications of the Committee on Safe Driving of the American Medical Association entitled "Are you fit to drive?" When physicians discover any condition in their patients which might make it inadvisable for them to drive a motor vehicle, they should advise the patient to report the condition on the next application for renewal of the driver's license. This is for the patient's own protection. If this information is not reported on the application, and the applicant subsequently becomes involved in an accident, to which the condition, either directly or indirectly, might be a contributing cause, in some cases the insurance company may legally refuse a liability. Although it is the role of the physician to advise the patient as to the patient's fitness to drive, his actual ability to drive must always be tested by the Motor Vehicle Department, and the responsibility for issuing or not issuing a license must always remain that of the Superintendent of Motor Vehicles. The enclosed form has been designed by the Committee on Medical Aspects of Traffic Accidents of the Nova Scotia Division of the Canadian Medical Association for the operator of a private motor vehicle. It has been simplified as much as possible, but more stringent and detailed forms will still have to be completed in some cases where the applicant is applying for a chauffeur license. **If the Physician or the Superintendent of Motor Vehicles feels that further opinion is necessary we have a Medical Review Board which has the final say.**

MEDICINE

Rheumatic Fever

Active rheumatic fever calls for restriction of activity; therefore, such patients should be advised not to operate a motor vehicle.

Rheumatic Heart Disease

Appraisal must be made of the extent of damage of the valves and to the derangements in rhythm, particularly if likely to alter suddenly, and to the efficiency of the myocardium.

Aortic Stenosis

When marked is prone to lead to angina, syncope and sudden death. Patients with severe aortic stenosis should be advised not to operate a motor vehicle. Patients with lesser degrees of aortic stenosis without symptoms should be advised not to operate a passenger transport vehicle but may operate a commercial or private motor vehicle.

Patients with aortic insufficiency and left ventricular failure and dyspnoea or paroxysmal nocturnal dyspnoea should be advised not to operate a motor vehicle. Patients with a lesser degree of aortic insufficiency without dyspnoea should be advised not to operate passenger transport vehicles but may operate a commercial or private motor vehicle.

Patients with myocardial insufficiency due to mitral stenosis or insufficiency should be advised not to operate a commercial or passenger transport vehicle but may continue to operate a private vehicle when the myocardial failure, if any, is well controlled.

Subacute bacterial endocarditis necessitates immediate treatment and abstinence from operating any motor vehicle.

Uncontrolled auricular tachycardia, flutter or fibrillation and a rapid ventricular rate, unless controlled, cause severe incapacity and should be regarded as contraindications to operating any motor vehicle. However, when these disorders of rhythm are well controlled, such patients may operate a private motor vehicle. Because of the workload and the stress on the heart, and because of the danger of embolization, patients with auricular fibrillation should be advised not to operate a commercial or passenger transport vehicle.

Hypertensive Cardiovascular Disease

Hypertension in itself is not disabling for safe operation of a motor vehicle, but the complications arising from hypertension - i.e. damage to brain, eyes, heart or kidneys - may well prove to be disabling. When complications of hypertension are limited to the optic fundi, the degreee of impairment of driving ability should be evaluated solely on the basis of loss of vision. If there is evidence of cardiac damage resulting in congestive heart failure or angina pectoris, such patients should be advised not to operate any motor vehicle, unless the congestive failure or angina is well controlled by therapy, then the patient may only operate a private motor vehicle. The level of the blood pressure must obviously be interpreted in the light of its constancy. The patient's sex and the patient's age, as well as the degree of complications, must be assessed, before any decision is made to restrict driving ability on blood pressure figures alone. Fixed hypertension without complications is not a contraindication to operating a private or commercial motor vehicle.

Arteriosclerotic Heart Disease

If sclerosis of the coronary arteries has resulted in angina pectoris and these attacks are mild, infrequent and controlled with therapy, such patients may still drive private motor vehicles and light commercial but not heavy commercial and passenger transport vehicles.

More severe angina, even if accompanied by varying degrees of heart block, ventricular premature contractions or arrhythmias, if well controlled by therapy, need not prevent the operation of private motor vehicles. These patients should not operate commercial or passenger transport vehicles.

Severe angina brought on with little effort should be considered a contraindication to the operation of a motor vehicle.

The patient with acute coronary thrombosis should be advised that he should not operate any transport vehicle for at least two months following the attack. Assessment thereafter would be dependant upon the presence or absence of congestive circulatory failure or severe angina pectoria.

Syphilitic Heart Disease

Patient with syphilitic aortitis having syncope, congestive heart failure, or angina pectoris, should be advised not to operate a motor vehicle, unless these symptoms are well controlled, by therapy, in which case it is permissible that they should drive a private vehicle but not a commercial or passenger transport vehicle.

Metabolic Heart Disease

If signs of myocardial insufficiency are present such patients should be advised not to operate a commercial or passenger transport vehicle but may operate a private vehicle.

Congenital Heart Disease

Asymptomatic congenital heart lesions are not contra-indications to safe driving. Patients developing signs of myocardial insufficiency should be advised not to operate a commercial or passenger transport, but may operate a private vehicle as long as their signs remained controlled by therapy.

Cor Pulmonale

Patients with right sided heart failures secondary to diseases of the lungs should be advised not to operate a commercial or passenger vehicle. However, if the symptoms of right ventricular failure, are mild and well controlled by therapy such patients may operate a private motor vehicle.

Myocarditis

Evidence of active myocarditis is a contra-indication to the operation of a motor vehicle.

Arterial Aneurysms and Arteriovenous Fistulas

Any patient with an arterial aneurysm, because of the danger of rupture, should be advised not to operate a motor vehicle. Arteriovenous fistulas resulting in severe heart failure constitute a contra-indication to the operation of any motor vehicle. Both conditions may be amenable to surgical treatment.

Diseases of the Pericardium

In the acute phases of the pericarditis, patients under therapy are unable to operate any motor vehicle. Viral pericarditis does not constitute a contraindication to the operation of a motor vehicle, once the acute phase has subsided. Patients with chronic constrictive pericarditis may operate a private motor vehicle if the condition is well controlled, but should be advised not to operate commercial or passenger transport vehicles.

Cerebral Vascular Disease

Patients with inadequate blood flow to the brain, having attacks of syncope or dizziness should be advised not to operate a motor vehicle. If there has been any cerebral vascular episode causing changes in personality, alertness, ability to make decisions, or if there has been actual loss of motor or sensory power or co-ordination, such patients should be advised not to operate a motor vehicle. However, if such changes in function are minimal, with little or no disability, it may be possible for these individuals to drive private motor vehicles.

Disease of the Veins

Patients with thrombophlebitis or previous thrombophlebitis resulting in edema of the extremities and impairment of use should be advised not to operate a commercial or passenger transport vehicle, and if sufficient disability exists they should be advised not to drive a private motor vehicle. Patients with active phlebothrombosis should be advised not to drive a motor vehicle because of danger of embolism.

Cardiac Enlargement

Enlargement of the heart should not in itself be considered a contraindication to the operation of a motor vehicle but such enlargement points to the presence of organic heart disease, which deserves appropriate evaluation.

Diminished Cardiac Reserve

Minor degrees of impairment of function of the myocardium should not be considered a contra-indication to the operation of a private or light commercial motor vehicle, but may not operate a heavy commercial or passenger motor vehicle. If myocardial insufficiency is more marked, the patient should be cautioned against the operation of a light commercial motor vehicle as it is likely to prevent proper performance under emergency conditions. If congestive heart failure is well controlled, such patients may operate private motor vehicles only.

Arrhythmias and Conduction Disturbances

Auricular premature beats are of little consequence and do not preclude the operation of motor vehicles. Patients with paroxysmal auricular tachycardia, flutter or fibrillation with myocardial insufficiency should be advised not to operate commercial or passenger transport vehicles. If, however, such attacks are well controlled by therapy, then the patient may operate a light commercial or private motor vehicle only. Auricular fibrillation of the chronic nature provides the risk of embolization from the auricles. Such patients should therefore, not operate a commercial or passenger transport vehicle but may operate a private motor vehicle if well controlled by therapy. Ventricular arrhythmias other than occasional ventricular extra systoles are usually associated with heart disease. Such patients should be advised not to drive commercial or passenger transport vehicles but may drive private vehicles if well controlled by therapy. Auriculoventricular block in a minor degree is of no significance. The patient with prolonged degrees of arterioventricular block or complete arterioventricular block, if associated with syncope (Stokes-Adams syndrome) should be advised not to operate any motor vehicle. If these attacks have been well controlled by therapy for one year or longer such patients may operate a private motor vehicle.

Hypotension

Hypotension in itself is not a contraindication to the operation of a motor vehicle. If, however, it results in attacks of syncope, such patients may operate a private motor vehicle. If hypotension is present and is considered to be related to symptoms of dizziness and/or syncopal attacks, it should

be assessed in the light of the seriousness of the underlying disease process.

Carotid Sinus Sensitivity

Individuals with carotid sinus sensitivity who experience attacks of syncope should be advised not to drive a motor vehicle. If after therapy the patient is cured this decision should be revised.

METABOLIC DISEASES

Disturbances in function of the endocrine glands causing severe generalized asthenia muscle weakness, spasm or tetany, sudden episodes of vertigo or unconsciousness, should be advised not to operate a motor vehicle until these symptoms have been controlled by appropriate therapy.

Diabetes Mellitus

Patients with diabetes mellitus of a degree that can be controlled adequately on diet alone may drive any type of motor vehicle. Other individuals with mild diabetes controlled by diet and a sulfonylurea type of drug who experience no reactions from this drug may drive any type of motor vehicle. If insulin is required and the diet is well controlled and the insulin requirements are well stabilized, the individual may drive a private motor vehicle. However, he should be advised not to drive a commercial or a passenger transport vehicle, because of the possibility of sudden attacks of hypoglycemia. The uncontrolled diabetic should be advised not to drive any type of motor vehicle. Individuals with true renal glycosuria may safely drive any type of motor vehicle.

Hyperinsulin States

Individuals suffering from recurring spontaneous attacks of hypoglycemia should not drive any type of motor vehicle. Mild cases who never develop unconsciousness or disabling symptoms may drive a private motor vehicle but should be advised not to operate a commercial or passenger transport vehicle.

Thyroid Diseases

Individuals with thyrotoxicosis, because of the co-existance of cardiac and emotional disturbances, should be advised not to operate any type of motor vehicle until the disabling symptoms are well controlled. Patients with an enlarged thyroid gland with pressure symptoms should be advised not to drive a commercial or passenger transport vehicle, although they may usually drive a private motor vehicle. Marked myxedemia or cretinism is a contraindication to driving a motor vehicle.

Parathyroid Diseases

Individuals with hyperparathyroidism with muscular weakness and hypotonia should be advised not to drive a motor vehicle unless symptoms are mild and controlled by therapy, in which case they may drive a private motor vehicle only.

Acute hypoparathyroidism with increased neuromuscular excitability is a contraindication to the operation of a motor vehicle.

Chronic hypoparathyroidism must be assessed on the basis of severity of symptoms. Mild cases without tetany may drive private motor vehicles but not commercial or passenger transport motor vehicles. The development of tetany, muscular weakness or easily provoked fatigue, should be regarded as symptoms which contraindicate the operation of a motor vehicle.

Pituitary Disease

Diabets, insipidus. These individuals should not drive a commercial or passenger transport vehicle, but provided there are no visual disturbances or other disabling central nervous system symptoms, they may operate a private motor vehicle.

Anterior Pituitary Deficiency

Hypopituitarism resulting in spontaneous hypoglycemic episodes should be regarded as a contraindication to the operation of a motor vehicle. Under close medical supervision, those people making a good recovery and without hypoglycemic episodes may drive a private motor vehicle but should be advised not to operate a commercial or passenger transport vehicle.

Acromegaly

With the development of marked muscular weakness, pain and easy fatigue, cardiac enlargement and intractable pain from headaches, patients with acromegaly, or gigantism should be advised not to operate a motor vehicle. Moreover, about fifty percent of these patients develop visual disturbances which make it unsafe for them to drive.

ADRENAL DISEASES

Adrenal Cortical Hyperfunction

Patients with Cushing's disease with marked muscular weakness and osteoporosis should be advised not to drive. Good recovery can enable these patients to operate a private motor vehicle.

Adrenal Cortical Hypofunction

Patients with Addison's disease, particularly those with asthenia or a persistently low blood pressure should be advised not to drive a motor vehicle unless the symptoms are mild and well controlled by therapy, in which case they may operate a private motor vehicle only.

Hyperfunction of Adrenal Medulla

Pheochromocytoma leading to attacks of headache, dizziness, weakness and blurring vision contraindicate the operation of any motor vehicle until these symptoms are relieved by treatment.

DRUGS

Central Nervous System Depressants

The drowsiness induced by analgesic drugs sufficiently impairs driving ability so that patients with such drowsiness should be advised not to drive a motor vehicle. In addition, morphine, its derivatives, and the synthetic narcotics, such as demerol (pethidine), cause varying amounts of euphoria, inability to concentrate, apathy, dimness of vision, and rapid flow of uncontrolled thought. Patients under the influence of these drugs should be advised not to operate a motor vehicle.

Hypnotics, Sedatives and Anaesthetics

Patients receiving mild sedation who experience no drowsiness may drive. Small doses of these drugs quieting highly excited or jittery patients may actually temporarily improve driving ability. However, this is not the usual circumstance. Therefore, it is best to advise patients taking hypnotic doses of these drugs not to drive a motor vehicle until eight hours later. Barbiturate addicts are incapable of driving a motor vehicle safely. Patients receiving barbiturates or local anaesthetics for minor surgery should be advised not to drive until the following day.

Tranquillizing Drugs

These frequently cause drowsiness and with larger doses may produce hypotension, productive of episodes of faintness or dizziness. Therefore, during the initial phase of dosage adjustment, patients should be advised not to drive a motor vehicle. At all times, patients must be carefully observed for symptoms of drowsiness or faintness. Patients stabilized on maintenance dosage of these drugs who are without symptoms may drive a private motor vehicle, but should be advised not to drive a commercial or passenger transport vehicle.

Central Nervous System Stimulants

These drugs may produce headache, dizziness, agitation, irritability and decreased ability to concentrate and are often followed by a period of fatigue and depression. In rare instances the individual may take one of these drugs to prolong the period of alert driving. The dosage, however, should not be more than the equivalent of 5 to 10 milligrams of amphetamine and without repetition that day. Drugs of this type should be used only on the advice of a physician.

Antihistamine Drugs and Those Preventing Motion Sickness

There is great individual difference in the degree of dizziness and drowsiness caused by these medications and this is unpredictable. Patients under these medications should be advised not to drive until it is established by prior trial that they experience no disability.

Anti-infective Agents

Such agents causing nausea, vertigo, dizziness, ringing in the ears, and deafness should prevent any person from being allowed to drive a motor vehicle.

Sulphonamides

Patients receiving sulphonamides should be warned that if they develop drowsiness or dizziness they should at once cease to drive a motor vehicle.

Cyclopegics and Mydriatics

These drugs should be used with the caution that normal distant binocular vision should return before safe driving can be achieved.

Hallucinogens

Hallucinogens such as marijuana have singular abilities for changing normal emotional reactions, even causing individuals to become oblivious or indifferent to their surroundings. Individuals under the influence of these drugs should be advised not to drive a motor vehicle.

Fatigue

Fatigued and drowsy or sleepy drivers are responsible for many accidents. They are unable to make split-second decisions, reaction time is slowed, and there is loss of peripheral vision and attention. In addition, individuals may become victims of highway hypnosis. Among commercial and passenger transport drivers, most accidents occur within a few hours after the start of the trip. This is due in part to off-duty activities. Thus all drivers should be cautioned against driving after inadequate rest. Moreover, persons who periodically develop highway fatigue should be advised against driving a commercial or passenger transport vehicle. Patients should be advised not to drive over eight hours a day. The vehicle should not be kept too warm, and on continuous drives the operator should be advised to stop and get out and walk around every two or three hours.

Fever and Infectious Diseases

Patients with fever should be advised not to drive because of decreased attention and impaired judgment, and reaction time.

Carbon Monoxide

Carbon monoxide from exhausts and from smoking decreases visual sensitivity in areas of low illumination. The relative oxygen deficiency from smoking three cigarettes is equal to that of travelling at an altitude of 7,500 feet. Since adequate ventilation does not take care of chain smokers who inhale, drivers should be advised to refrain from frequent smoking prior to and during periods of night driving or at high altitudes.

EYES

	Vision	Field	Endorsement
Heavy Transport	20/30, 20/30	120° each eye	—
Light Transport	20/30, 20/60	120° each eye	—
Private Cars	20/40, better than 20/200	120° each eye	—
	20/50, better than 20/200	120° each eye	Drive daylight only
	20/40, 20/200 or worse	120° in better eye	Drive with rear view mirror on defective side.
	20/40 monocular vision only	less than 120° other eye	

Note: - If glasses are required to obtain these visual standards, a note to this effect should be made in the office record or to any request for information from the Medical Review Board.

Visual Acuity

In Nova Scotia the visual acuity requirements are 20/40 with corrected vision. The Motor Vehicle Branch tests for visual acuity, and if they require further opinion, they will refer the patient to an ophthalmologist. However, should the family doctor note some striking abnormality with the eyes, he should record his comments and advise the patient to report to the Motor Vehicle Branch. Patients with less than 20/50 corrected vision in the better eye should be advised not to drive. Patients with corrected vision between 20/40 and 20/50 should be advised not to drive in congested traffic, in hazardous road conditions, at high speed or at night. Regulations for licensing drivers of commercial and passenger transport vehicles, require higher visual acuity than for drivers of private vehicles.

Visual Fields

Patients with visual form fields of 140 degrees or more may operate a motor vehicle safely. Patients with form fields less than 110 degrees should be advised not to drive. The patient with visual fields between these limits may be evaluated on the basis of the conditions under which he must drive and the amount of lateral vision he possesses. Homonomous hemianopsia and bi-temporal field defects, if complete or confined to inferior quadrant are not compatible with safe driving.

Ocular Muscle Imbalance

The accident potential of ocular muscle imbalance is indirect through causing driver fatigue. However, when diplopia is present, accidents are directly attributable to the diplopia Therefore, all patients with diplopia should be advised not to drive a motor vehicle unless it is controlled with treatment.

Colour Blindness

Colour blindness has in the past been considered to be a potential cause of highway accidents. At the present time, however, traffic lights have been standardized and, except in cases of those engaged in long-distance driving or heavy transport work, it is doubtful if colour blindness or deficiency is of any great consequence.

Dark Adaptation

Dark Adaptation or susceptibility to glare are of importance in night driving, but at the present time no valid data is available as to their importance in the causation of highway accidents. Dark glasses must never be worn for night driving, and windshield tinting should be limited to the upper third.

Depth Perception

Tests for depth perception are inadequate at this time. The road test is still the best and most practical.

OTOLARYNGOLOGY

Deafness

Although deafness is a handicap in the safe operation of a motor vehicle, usually it is compensated for quite well. The deaf driver, being fully conscious of his disability, tends to be cautious and fully alert at all times. Moreover, the use of an outside rearview mirror is of great value. The wearing of a hearing aid does not prove practical in noisy commercial or passenger transport vehicles; however, in the operation of private vehicles, patients who successfully wear hearing aids frequently find them useful. Deafness is disqualifying in the operation of passenger transport vehicles but not necessarily for the operation of commercial or private vehicles, except in such people with bilateral total nerve deafness such as deaf-mutes.

Dizziness

Patients subject to attacks of Meniere's syndrome dizziness no matter what the cause, should be advised not to operate a motor vehicle until proper treatment has controlled such attacks. The patients may then resume driving a private motor vehicle but not a commercial or a passenger transport vehicle. The character of these attacks may change so that sufficient warning of the impending attack is given, enabling the patient to drive his vehicle over to the curb or off the highway. In such an event the patient may resume driving a private motor vehicle. Patients seriously handicapped with speech disorders or irremedial deformities and disease of the mouth, nose and throat interfering with feeding, speech or breathing should not operate a passenger transport or commercial vehicle. Patients with a tracheotomy should not drive a motor vehicle because of the possibility of sudden obstruction of the tracheotomy - Post laryngectomies would be an exception.

SURGERY AND ORTHOPAEDICS

All patients with orthopaedic disorders must show that they have sufficient strength to control the vehicle, to turn the steering wheel, and to apply the breaks sufficient to slide the wheels. They must be able to reach all controls required in the operation of the vehicle, whether by mechanical means on the vehicle or by suitable prosthetic devices, and those so handicapped must demonstrate that they can successfully operate their vehicles in this manner. These people are more liable to fatigue and should be advised not to drive without adequate rest periods.

Head and Neck Movements

Relationships and mobility should be normal for operators of commercial and passenger transport vehicles. Variable degrees of restricted movements of the head and neck are compatible with safe operation of private vehicles. Any limitations of rotation of the head by fifty percent or more by disease or splinting necessitates compensatory aids to enable these individuals to drive safely. Outside mirrors and swivel-type bucket seats can sufficiently aid some of these people. Patients wearing neck casts or braces should be advised not to drive until recovery has progressed to the point where pain and restrictions of cervical movement are minimal. Patients with spastic torticollis should be advised not to drive a motor vehicle. Patients with mild congenital torticollis without pain or significant disability may be advised that it is safe for them to drive.

Thoracic Spine

Patients with disabling diseases of the thoracic spine should be advised against driving a commercial or passenger transport vehicle. Patients with marked deformity, painful restriction of motion, or difficulty in respiratory expulsion should be advised not to drive a motor vehicle. However, proper bucketswivel seating may provide sufficient compensation for some of these individuals. Patients with interscapular pain which causes restriction of the shoulder joints and increases vulnerability to fatigue and irritability should be advised not to drive until they recover. Mild to moderate degrees of scoliosis do not interfere with safe driving. Severe degrees of scoliosis when accompanied with painful arthritis may constitute a contraindication to driving. Those wearing braces should be carefully evaluated by the physician as to their ability to manipulate a motor vehicle safely. Patients with osteoporosis are vulnerable to vertical fractures from abrupt deceleration and should be so warned. Such patients can be safe-guarded by bucket-type seating and proper safety restraints, particularly seat belts. Patients with myeloma of the ribs and spine should be advised similarly.

Lumbar Spine

The lumbar spine is particularly vulnerable to compression fractures during abrupt forward deceleration, especially if the hips are stiff. Such patients should be advised to seek seating revisions or shoulder-type harness and safety belts. The lumbar region of the spine is vulnerable to fatigue and should be normal for drivers of passenger transport and commercial vehicles. Painless lumbar abnormality may not prevent a patient from safely operating a private vehicle. Patients with herniated lumbar discs with pain should be advised against driving during attacks of disabling pain.

Upper Extremities

Normal functions are required for drivers of passenger, transport and commercial motor vehicles. A one-armed person, provided the single form is a good one, can operate a private motor vehicle safely especially when equipped with power steering and a wheel button. The same is true of the loss of one arm one leg, which requires the balancing effects of proper prosthesis. The hands should be able to grasp to provide bracing against rapid deceleration, and an adequate number of opposing digits to the thumb are therefore essential for proper gripping and driving. Power steering is of great assistance, but each individual with hand-function impairment must demonstrate sufficiently his ability to properly operate a motor vehicle, and if power steering is used this should include control of steering when there is a power failure.

Lower Extremities

Lower extremities of normal function are essential for drivers of passenger transport and commercial vehicles. However, with good arms,

even the absence of both legs may not prevent the safe driving of private motor vehicles with proper prosthesis and special hand controls. During prolonged driving, fatigue will accrue from improper buttock and thigh support. Ankylosis of the hips needs special seating arrangements and warning against sudden deceleration.

NEURO-PSYCHIATRIC DISORDERS

The patient with an emotional, neurological or psychiatric disorder, presents the conscientious physician with a difficult problem.

NEUROLOGICAL DISORDERS

Convulsive Disorders

Epileptic patients not receiving medicaments who have been seizure free for a minimum of two years are considered good risks for the operation of private vehicles, but should be advised not to drive a commercial or passenger transport vehicle. Epileptic patients under medication should be advised not to drive a motor vehicle until they have been seizure free for a minimum of two years. Then they should only drive private motor vehicles. All epileptic patients should be advised not to consume alcoholic beverages in any form for at least twenty-four hours prior to driving. Moreover, fatigue should be avoided and six hours should be the maximum number of hours behind the wheel in one day. Epileptic patients should be advised that night driving may be particularly dangerous because photic stimuli from opposing head-lights may precipitate a seizure. Emotional stress should be minimized by avoiding driving in peak traffic hours. Epileptic patients may not drive if they have undesirable side effects from medication, which might impair their ability to drive. These patients are advised to secure a review of their progress at least once annually by a physician.

Faintness, Syncope and Episodic Weakness

It would be impossible to cover all the syndromes, diseases and physiological disorders which may produce these symptoms. It is important, therefore, that the pattern of these symptoms be evaluated carefully in terms of impairment of driving ability. The isolated occurrence probably is of little concern. Patients having had a history of multiple occurrences should be advised not to drive a motor vehicle until a diagnosis can be established and satisfactory corrective measures have been instituted. In all instances in which no diagnosis can be made and symptomatic therapy is not effective, the patient should be advised not to drive a motor vehicle.

Narcolepsy

Narcolepsy is rare and infrequently encountered. Patients under treatment should be advised not to drive a motor vehicle until they have been sympton-free for two years and are experiencing no adverse side effects from medications.

Disorders affecting Muscular Control or Co-ordination

Obviously this includes a wide variety of diseases affecting the nervous system. Each of these will pose special problems with respect to the driving abilities of the individual patient. The more serious disorders generally cause either rapid deterioration or sufficient functional disability of such degree that the patient will of his own volition cease driving when his ability reaches a point that makes driving unsafe.

All individuals suffering from acute or chronic brain syndromes, such as cerebral vascular disease, degenerative brain disease, and so on, that may impair consciousness, memory, behaviour, judgment, co-ordination or motor power to sufficient degree as to render them incapable of handling a motor vehicle competently ought not to drive either a private or commercial motor vehicle.

Mental Deficiency

Patients with an intelligence quotient below 80 are not fit to operate a motor vehicle. Any patient who has not reached grade V should have proof of an I.Q. of 80 or better from a qualified psychologist.

Emotional Disturbances

Many varied stresses and situations may create in normal individuals a temporary emotional upset impairing their driving ability. The manifestations occur in three abnormal behaviour patterns. Firstly, the individual is so absorbed in this problem that he is indifferent and inattentive to traffic and the world about him. Secondly, some individuals become despondent with actual depression and psycho-motor retardation. Such individuals have sufficiently slow reflexes to make driving a real hazard. Thirdly, many such individuals become antagonistic, impulsive or openly aggressive with loss of judgment and loss of some sense of caution. Patients exhibiting any of these three symptoms should be advised not to drive a motor vehicle until they have recovered.

Individuals exhibiting aggressiveness, anti-social trends and social irresponsibility frequently have high accident rates. They are impulsive and show poor judgment.

PSYCHIATRIC DISTURBANCES

In Nova Scotia, when a patient is discharged from the Provincial Mental Hospital, the attending

physician from the hospital should be the one to determine the patient's driving competence. If he is on probation, the Medical Syperintendent should note on his probation paper whether or not the patient is fit or unfit to drive during the probation period.

A psychiatric patient on discharge from a general hospital or a private hospital should be advised by his attending physician if he believes that the patient cannot safely operate a motor vehicle. The attending physician should advise a responsible member of the patient's family of this also.

Similarly when a psychiatric patient is being treated on an Out Patient basis, in a Provincial clinic, general hospital, private clinic or by a physician in his office, it is the responsibility of that physician to advise both the patient and a responsible member of the patient's family if he believes that the patient is not fit to operate a motor vehicle.

Patients who have returned to the community from hospital care and patients who are receiving Out Patient psychiatric care are frequently on maintenance therapy with medication. The physician attending should advise the patient and a responsible member of his family that the patient ought not to drive if there is drowsiness, syncopy or lack of co-ordination present. These effects are particularly noted with certain of the tranquillizers.

Psychoneurosis

Anxiety reactions, disassociation reactions, conversation reactions, phobic reactions, obsessive compulsive reactions and depressive reactions are among the conditions observed in the psychoneurotic patient. The patient with a psychoneurosis represents an unknown quantity with respect to highway safety. Each case requires separate evaluation regarding alertness, social behaviour and possibly psychomotor retardation. If no significant behavioural problem or drug therapy side reactions exists, the psychoneurotic patient may be advised by his physician that it is safe for him to drive. Some particularly compulsive patients are safer drivers than "normals".

Personality Disorders

This group represents the biggest problem with regard to safe driving. They include many people who are accident prone and who drink to excess. Different studies have shown them to be "difficult, aggressive people with an aversion to authority and restrictions generally". They frequently get along poorly, not only in their driving record but also in other respects in their daily living; for example, poor work record, a history of being involved with the law etc. These are the people that particularly concern licensing bodies and about whom they are apt to consult the physician.

ALCOHOL

In Nova Scotia alcohol is the responsible factor in a large percentage of motor vehicle accidents attributed to speed, reckless driving and driving on the wrong side of the road. It is estimated that in Nova Scotia approximately 50 per cent of accidents involve an impaired driver. The effect of alcohol on human beings is in all phases and stages a depressant. Studies reveal that test subjects after consumption of alcohol almost unanimously believe that their performance is unimpaired or even better than usual, whereas objective testing demonstrates that performance is impaired.

Alcohol and Driving Ability

Holcomb concluded from his studies that driving performance began to be impaired at a blood alcohol level of 0.05 per cent or 50 mg. per 100 ml. of blood. Other studies indicate an even lower level of impairment beginning at a blood alcohol level of 0.035 per cent. In Nova Scotia the Medical Society has approved a level of 0.1 as indicative of impairment.

Alcohol Consumption and Blood Alcohol Level

Numerous studies using individuals accustomed to drinking, all are in full agreement that six 12-ounce bottles of 3.2 per cent beer or 6 ounces of 90 proof whisky consumed within one hour will put the average moderate drinker in the zone of impaired driving ability; that is, with over 0.1 per cent alcohol in the blood.

Rate of Oxidation and Elimination

The average person of 150 pounds can oxidize and eliminate about one-third fluid ounce of alcohol per hour, decreasing the concentration of alcohol in his blood by approximately 0.015 per cent per hour. Two hours will reduce the blood alcohol concentration about 0.03 per cent if the individual does not consume more alcohol. This offers a clue to the length of the necessary waiting period after drinking before driving.

The average individual with a blood alcohol level of 0.05 per cent or higher suffers significant deterioration of his driving skill and is a potential menace on the highway. Thus one drink may be tolerated. Two drinks would put him in the level of impairment for about two hours. Three drinks are too many.

Chronic Alcoholics

Habitual alcohol intoxication is a contraindication to safe driving because of the inability to predict the periods when safe driving is possible.

SUMMARY

These standards are offered as a guide in determining the fitness of a driver to operate a motor vehicle. Many specialist groups in Nova Scotia have participated in gathering the up-to-date material. It is hoped that the Committee on the Medical Aspects of Traffic Accidents of the Nova Scotia Division of the Canadian Medical Association will keep this guide under constant review as additional information comes to light when the many and varied organizations dealing with Traffic Accidents make their reports. It is also hoped that the physicians throughout the province will feel free to make suggestions regarding changes for the consideration of this Committee. The traffic accident problem is becoming rapidly one of the major public health problems. It is anticipated in five years time, with the additional mileage driven by the additional cars that will then be on the highways, that the death toll will be double that of 1964 (380).

It is our earnest desire to aid the physicians in the province in performing their duty and moral obligation and that they will feel obliged to recommend to the patients not qualified under these standards, that they should not drive.

In the future, we hope that a much closer liaison will be established between the Committee and the Motor Vehicle Branch in this province. With this form of closer co-operation it would become feasible to establish medical standards that could be considered as law. It is not our intention now, nor in the future, to make compulsory reporting of medical disabilities law. We feel that this would put an unnecessary burden upon the practitioner of medicine in the province. The task of determining whether or not a person is a fit or unfit driver, must remain the responsibility of the government and the only practical part that a practitioner can play is in an advisory role. Along these lines, it would be most helpful to have a medical form provided by the provincial government which could be used for new drivers, as well as those coming up for review because of questionable medical problems. □

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ABBREVIATIONS USED: Abst. - Abstracts; Corr. - Correspondence; CR - case reports; Edit. - Editorials; Rev. - Review; 1000 - Thousand Word Series.