INDEX OF MEDICAL GUIDELINES

Definitions

As with other substance abuse problems, many widely ranging definitions have been proposed to designate problems of abuse of alcohol. Despite a multitude of scientific medical, genetic, sociological, economic and other studies world-wide, there remains no clear consensus. This shows the complexity of the related problems. The definitions tend to focus on (a) cause - genetics, race, family, cultural attitudes, socially accepted norms, etc; (b) behaviours - frequency, intensity of intoxication, social and economic concomitants; (c) physical and mental diseases resulting from prolonged excessive use.

The World Health Organization (W.H.O.) provided the following definition in 1952:

> "Alcoholics are those excessive drinkers whose dependence upon alcohol has attained such a degree that it results in noticeable mental disturbance, or in an interference with their mental and bodily health, their interpersonal relations, their smooth social and economic functioning; or those who show prodromal signs of such development."

In the 1970's the W.H.O. modified its stand by referring to an "alcohol-dependence syndrome with or without alcohol related disabilities".

The American Psychiatric Association in 1983 (D.S.M. III) defined Alcohol Abuse and Dependency (Alcoholism) as "a pattern of pathological alcohol use that causes impairment in social or occupational functioning, and either tolerance or withdrawal reactions".

Whether or not "alcoholism" is defined as a disease is dependent on the definition of what constitutes "disease". Since neither term has precise boundaries the classifications remain open to argument.

In common usage, alcoholism, alcohol abuse or alcohol dependency is a behaviour disorder defined by the problems it creates rather than by the chemistry of the substance per se.

Etiology

For practical purposes alcohol dependency arises when, for a variety of reasons, the dependent individual has chosen to use alcohol, has learned to enjoy its mood altering effects, has lost control of this acquired taste and has become dependant upon it on either a regular or intermittent basis. While there has been much dispute in the past, the current medical consensus no longer accepts that alcohol dependency is a disease process, and it is reasonable to state that, until such time as the secondary organic disorders make their appearance, any associated disability is of a transient nature, dependent entirely upon the ingestion of alcohol and is the result of a voluntary decision and act on the part of the dependent individual.

Essential Features

In all cases, the essential features are:

1. **Exposure to Alcohol**

   The availability of alcohol is and has been for many generations so widespread that, for practical purposes, it can be considered a universal phenomenon.

2. **The Decision to Indulge**

   Many factors (childhood up-bringing, religious beliefs, cultural and social customs, peer pressures and the like) enter into the decision to ingest alcohol. There is no medical evidence that alcohol is necessary to satisfy any normal physiological or metabolic requirement of the body. It is clear, therefore, that the decision to use or not to use alcohol is a personal decision of the individual.

. **Types of Alcohol Dependence**

   (a) a learned behavioral response (40% - 60%)
   (b) associated with a personality disorder (20% - 30%)
   (c) associated with psychiatric disease - usually a neurosis but sometimes a psychosis (20% - 30%)

The first group comprises those individuals who, for one reason or another, (e.g. the social or cultural practices of their environment) have learned to like the effects of alcohol and have become dependent upon it.

In groups (b) and (c) there must, of course, have been the initial personal decision to partake of alcohol, and an element of learned behavioral response has developed in these more susceptible individuals.

Pension Considerations

1. Alcohol Dependency is the preferred term to be used in the submission of claims and in the ruling given, rather than the previously used terms of Acute, Episodic, or Chronic Alcoholism, etc.

2. Alcohol Dependency, without associated organic physical disease, is not considered a disease within the meaning of the Pension Act.

3. Since Alcohol Dependency can, and does, lead to organic physical and mental changes, these organic disorders constitute diseases associated with continuing disability (e.g. Cirrhosis of the liver) and should be ruled on in the usual way, upon application by the veteran or his/her representative.

4. Where entitlement is awarded for psychiatric disease, and there is evidence of alcohol dependency as a manifestation of that disease, any resulting organic physical disease will be considered on a consequential basis, depending on the individual merits of the case, and provided that application has been made by the veteran or his/her representative.

Alzheimer's disease is classified as one of the dementias, that is, one of the conditions characterized by failing memory, loss of intellectual function, personality change and abnormal behaviours. It is a degenerative condition in which there is a severe, diffuse loss of nerve cells in the cortex and some of the deeper nuclei of the brain, resulting in a shrinkage of the cortical gyri and an enlargement of the ventricles. Originally, the term was applied only to those cases in which dementia developed between ages 45 and 60. Clinically and pathologically, however, Alzheimer's disease is indistinguishable from the senile dementia of later onset. For this reason, it is common practice to diagnose both pre-senile and senile dementias as Alzheimer's disease.

The underlying cause of the nerve cell loss in Alzheimer's disease is unknown. Application of the term in a given case implies that the dementias of known cause, such as those resulting from multiple brain infarct, alcoholism, trauma, tumour, and drug intoxication, have been ruled out.

Alzheimer's disease may begin in the late forties, in the fifties, the sixties, or even later. At first it is characterized by the forgetting of names and words, then of appointments, and location of personal belongings. As the condition advances, the lack of appropriate words slows speech and writing and eventually leads to loss of comprehension and an inability to speak full sentences. Social behaviour deteriorates. Personality changes, paranoid or bizarre behaviours may be seen.

Familiar streets are no longer recognized, and even the location of the bathroom at home is not known. Eventually there is loss of coordination which causes difficulty walking, dressing, and using everyday utensils such as knife, fork, and spoon. Finally, the ability to walk or even stand is lost and the patient becomes bedridden, requiring total care.

Initially, assessment is based primarily on the effect of memory disturbance on present employment (if under retirement age) and ability to function in society. Later, the degree of dependence on others for safety within the home and activities of daily living should be determined. Unless another disease process intervenes, the condition will progress over an average range of 5 to 10 years, with total disability existing and institutional care required for a variable period of time before death.

General Information

Arteriosclerosis is a general term for the degenerative changes that occur in arteries. Medically, degeneration is defined as:

> "a process by which tissue deteriorates, loses functional capacity, and may be converted or replaced by other kinds of tissues."

There are three types of arteriosclerotic degenerative changes commonly recognized:

1. **Atherosclerosis** - described below.

2. **Monckeberg's Sclerosis** - a change in the musculo-elastic layer (middle coat) of the large and medium sized arteries.

3. **Arteriolar Sclerosis** - generalizing thickening of the wall of the arterioles which are not affected by (1) and (2) above. Clinically when found this is most evident in the kidney.

1. ATHEROSCLEROSIS

Definition

Atherosclerosis is a variable combination of changes of the inner lining of arteries consisting of focal accumulation of lipids, complex carbohydrates, blood products, fibrous tissue and calcium deposits with associated changes in the middle coat of the wall of the artery.

Clinical Manifestations

The disease is insidious in onset and may exist for many years without revealing its presence. The arterial changes are usually widespread affecting particularly the arteries of the heart, brain, kidneys and lower limbs. Symptoms when they occur, are invariably related to the particular site or sites where the condition has progressed to the greatest degree.

Symptoms are caused by deficiency in the blood supply of the part affected; and appear gradually when the deficiency is due to narrowing of the arteries (ischaemia), suddenly when the blood supply of a part is completely cut off (infarction). A wide variety of symptoms may thus present depending on the severity of the degenerative process and the part of the body affected.

The following are some typical examples:

1. Heart - attacks of central chest pain radiating to the upper extremities (angina), sudden collapse with possible fatal outcome;

2. Brain - mental changes, stroke;

3. Kidneys - renal failure;

4. Lower Extremities - cramp-like pains on exercise, gangrene of toes.

Clinical Manifestations ..CONTINUED

As can be seen from above, this type of arteriosclerosis produces almost all the clinical conditions which cause disability from arteriosclerosis.

Etiology

Atherosclerosis is the commonest disease of the artery wall and is of worldwide distribution. While it is much more in evidence in the older age groups, no age is immune. The question which arises is not who has atherosclerosis, but rather who has more and who has less, (and particularly who has symptoms from this condition). It affects the arterial tree from the largest vessel (the aorta) to the thinner walled arteries of the heart, brain and gut, the smallest arteries (arterioles) alone escaping. Its effect on the coronary arteries make it the most common single cause of death.

The Atheromatous Plaque

Many agents have been investigated in an endeavour to incriminate those which might play a part in causing, or advancing the atheromatous process, but with little success.

One of the most obvious features of the atheromatous lesion is the presence of lipids and experimental work has shown that they are derived from the cholesterol in the blood plasma. The disease process begins by the deposition of lipids from the blood plasma on the inner lining (intima) of the artery wall as tiny translucent elevations. These lipids are transported into the intima where a series of changes occur resulting in the formation of fatty streaks which coalesce to form plaques (atheroma) projecting into the lumen of the artery. At the same time, minute thrombi occur in the intima with the formation of fibrous tissue concurrent with degeneration of the elastic tissue of the artery walls. The site of election of the plaques, which may eventually calcify or ulcerate, is at the bifurcation or branching of arteries where the pressure of blood is greatest. As the disease advances, (1) the artery wall loses its elasticity, (2) narrowing of the lumen of the artery occurs which may progress to occlusion (blockage), (3) sudden occlusion may be brought about by the formation of a blood clot (thrombus) or by a detached portion of atheromatous material blocking a vessel (embolism), (4) degeneration of the atheromatous plaque may lead to rupture of the affected artery and haemorrhage.

The Role of Lipids

It is known that sustained high levels of cholesterol in the blood (hyperlipemia) favours the occurrence of atherosclerosis as is seen in diabetes, myxodema and xanthomatosis. Lesions resembling atherosclerosis have also been produced in animals by artificially inducing hyperlipemia. Much attention has, therefore, been given to the possible relationship of diet to blood cholesterol and in turn to atherosclerosis. The lipid content of the blood is raised by a diet rich in animal fats and races who have a low intake of fat are not so prone to the development of atherosclerosis. It has also been noted that the wholesale starvation of people in time of war was associated with a marked decline in its incidence. On the other hand the majority who develop atherosclerosis have normal cholesterol levels, and the general conclusion is that increased level of blood lipids is not the primary cause of atherosclerosis, but is an important factor in the development of the disease.

Metabolic Disorders

Although atherosclerosis is not ordinarily considered a metabolic disorder per se, it is apparent that in certain metabolic states its development is accelerated. Physicians have observed a relationship between gout and atherosclerotic changes in the heart and epidemiological studies have established the validity of this relationship and have been able to quantity it. Diabetes representing a disturbed glucose metabolism, as already stated, is also associated with an increased risk of atherosclerosis.

Hypertension

Hypertension increases the incidence of atherosclerosis. As already stated, the sites of election of atheroma are at points where the pressure of blood is greatest. It is believed that the lipids gain access to the intima as a result of increased permeability due to the stretching-effect of the raised blood pressure.

Heredity

A number of investigations have been carried out on groups with symptoms of atherosclerosis generally showing a family history of similar problems. Medical authorities in general agree that a genetic factor operates in determining the rate of progress and severity of the disease.

Age and Sex

Atherosclerosis increases steadily with age, but not at the same rate in all people. That sex hormones may be a factor is suggested from the prevalence of the condition in the male and the relative immunity of the female up to the menopause. Thereafter the incidents in females rises and by the age of 70 there is no difference between the two sexes.

Obesity

Evaluation of the role of obesity in the development of atherosclerosis is complicated by the high incidence of accompanying hypertension and by the fact that both obesity and atherosclerosis tend to increase with age. The increased incidence of atherosclerosis in obese persons indicated by various statistical studies is, therefore, difficult to evaluate and the exact etiological role, if any, of obesity in relation to atherosclerosis has not been established.

Physical and Mental Stress, Physical Exercise

The most widely studied and useful information about the effects of physical and mental stressors on atherosclerosis comes from studies of its complications in coronary heart disease. This is readily understandable because generalized atherosclerotic changes are difficult to study in the living whereas those in the heart, having specific, sudden and dramatic effects, and amenable to study by measuring changes in the electrical activity of the heart muscle.

Numerous and extensive studies of atheromatous coronary disease have failed to reach a consensus that any specific physical, mental, social or economic stressors affect the development or rate of advance of atheroma.

There is a considerable body of evidence that persons with Type A personality are more prone to coronary occlusions in their middle years than those with Type B personality.

Type A types are described as:

(a) ambitious, aggressive, competitive, impatient, and pushed for time;

(b) alert and rapidly reacting, muscularly tense, with emphatic speech and rapid pace in activities;

(c) tending toward quick emotional responses of irritation, anger or hostility.

Type B types are those displaying contrasting types of behaviours. It follows that Type A's have a higher level of energy expenditure than Type B's.

While it has been found that Type A's have more coronary troubles than Type B's, it has not been demonstrated that they have more atherosclerosis. The reason for increased incidence may well be that their personality traits and reactions worsen what would be mild problems in a Type B personality.

Cigarette Smoking

Experimental studies have shown that smoking affects the heart rate, cardiac output, blood pressure, coronary blood flow, blood coagulation and the level of blood lipids. This accounts for the association between cigarette smoking and the morbidity, and mortality of atherosclerotic heart disease as shown in extensive population studies. Whether smoking actually influences the progression of atherosclerosis, or whether its effects are solely to accentuate the manifestations of the atherosclerosis already present, has not yet been finally settled.

Sepsis

A careful review of the vast literature on the subject of atherosclerosis contains no hypothesis or support of a relationship between it and sepsis. The absence of such a hypothesis from the literature is significant when it is borne in mind that sepsis and atherosclerosis are universal and yet clinicians engaged in the practical study of the disease have never suggested a connection between the two.

Other Factors

Careful studies of the effects of varied conditions such as fatigue, change of climate, respiratory disease, or psychiatric illness indicates that such conditions have no influence on the course of atherosclerosis.

Conclusion

Atherosclerosis is a degenerative process, the onset and rate of progress of which is determined by inborn constitutional factors. The maximum incidence of manifestations is seen in the older age groups and in those suffering from chronic hypertension and disease characterized by hyperlipemia. Much evidence has been accumulate, clinical, pathological, epidemiological and experimental, and many theories as to causation have been put forward, carefully investigated and evaluated by constantly improving methods of research. The sum of investigations, to date, which have covered all external factors

to which a patient may be exposed shows that with the exception of smoking and diet insofar as it can influence the level of blood lipids this disease is not caused or adversely affected by such factors.

Pension Considerations

Veterans Affairs Canada has established that the term "arteriosclerosis" shall be used to identify the following major areas of clinical arterial disease -

(a) Arteriosclerotic heart disease.

(b) Arteriosclerotic cerebrovascular disease.

(c) Arteriosclerotic peripheral vascular disease.

It is realized that some clinicians might prefer to use the term "artherosclerosis" but the above terminology is understood by all lay people involved in the pension adjudication process and so should not be altered or confusion could result.

Arteriosclerosis (atherosclerosis) is recognized as a natural degenerative process present in the population at large. It is accepted that the first pre-clinical arteriosclerotic aging changes begin early in life with progressive vessel changes common to all individuals varying only in the rate of progression. It is generally agreed, therefore, that when medical evidence of clinical disease of arteriosclerotic origin appears, the affected vessels have been involved in the progressive process for a prolonged, but variable period of time.

Arteriosclerosis is undoubtedly a part of the aging process and the Pension Act does not provide for pathophysiological change from age. Veterans Affairs Canada has adopted the practice, for pension purposes, that up until such time as there is acceptable clinical evidence indicating the onset of disability from arteriosclerotic change, the arteriosclerotic process is neither a disability, nor a disabling condition. It follows, therefore, the onset of a disability on which an award can be based is the time at which clinical disease resulting in disability is demonstrated.

This practice allows for consideration of an award for one or more areas of arteriosclerosis (cardiac, cerebral, peripheral) where clinical evidence of disability in the involved area(s) has been first demonstrated during the period of service under consideration, and of the other area(s) if involved at a much later date. Example: A veteran might be granted entitlement for arteriosclerotic heart disease as incurred during his W.W. II service. In addition he might wish to claim for arteriosclerosis of cerebral or peripheral involvement many years later as being related to the same period of service; and the decision as to these entitlements would be based on the evidence available to Veterans Affairs Canada at the time claimed.

Veterans Affairs Canada recognizes that many skilful and highly respected clinicians accept reasonable current theories which have not yet been accepted in authoritative up-to-date textbooks. Veterans Affairs Canada considers that before a theory can be considered acceptable to the majority of the profession, such a theory must be outlined and accepted in authoritative and up-to-date textbooks. When such acceptance has been so demonstrated, it can then be considered to be a consensus of medical opinion.

DEGREE OF RELATIONSHIP OF ARTERIOSCLEROSIS (ATHEROSCLEROSIS) TO CONDITIONS SUCH AS HYPERTENSION AND DIABETES MELLITUS

Medically it is unquestioned that hypertension and diabetes hasten the progress of arteriosclerosis in all arteries. As all arteries are affected, it is reasonable to expect that over a period of years clinical arteriosclerotic disease may produce disability in all three areas. Accepting this premise, Veterans Affairs Canada has decided to consider all claims for consequential arteriosclerotic manifestations under the diagnosis of "Arteriosclerosis".

Disability in all areas of clinically recognized disease will be assessed under the diagnosis "Arteriosclerosis" based on the degree of consequentiality established by ruling after the first appearance of disability in any of the major areas affected.

The question of estimating the degree of consequentiality in the individual cases of arteriosclerosis to pensioned hypertension and/or diabetes mellitus is most difficult. The description of what is known of the so-called risk factors appear in the body of the Guidelines.

There is so much individual variation in the progression of arteriosclerosis that the single factor, acceptable in all cases where a relationship to hypertension and diabetes mellitus is being considered, is the age of clinical onset. As a general rule, the appearance of secondary clinical arteriosclerosis in a veteran in his thirties or earlier would allow for a three-fifths relationship, in his forties a two-fifths relationship, and in his fifties and older a one-fifth relationship.

The result will be that there will be one ruling of arteriosclerosis consequential upon hypertension or diabetes mellitus with the degree of consequentiality established the first time entitlement is awarded. Future manifestations in other areas would not need to be ruled upon for entitlement purposes - they would be covered by assessment of the overall condition of arteriosclerosis.

It is stressed that the above only applies when the arteriosclerotic disease is consequential upon conditions such as hypertension or diabetes mellitus.

Amputation and Arteriosclerotic Coronary Artery Disease

Statistical studies carried out in 1954 and 1965 failed to show an increase in cardiovascular disorders in amputees compared with the general population.

All surveys confirm the fact that there is no relationship between amputations and the subsequent development of hypertension.

In 1977 the Medical Follow-Up National Research Council of the United States undertook a survey to determine if there was a relationship between amputation of an extremity and the subsequent development of Cardiovascular disorders. This study concluded:

1. A comprehensive review and professional analysis of the literature and,

2. An analysis of statistically valid samples of disability claims of veterans having service-connected extremity amputations, matched by age, sex, and war period with non-amputee veterans.

The following facts were statistically significant:

1. The risk factor for coronary artery disease in upper limb amputees and single below knee amputees was the same as for the control group.

2. There was an increased risk factor for coronary artery disease in bilateral lower limb amputees and single lower limb amputees if the amputation was at the knee or higher.

3. No specific factors could be found to account for increased risk factors in the second group.

4. This slight increase in risk factors is evident only in the later years.

Despite the fact that no specific reason for the increased risk can be ascertained cognizance should be given to this recent survey. For pension purposes it is apparent that:

1. There is no relationship between amputations and the subsequent development of hypertension.

2. Upper limb amputations play no part in the development of coronary artery disease.

3. Arteriosclerotic heart disease (syn. coronary artery disease) developing in an amputee who has bilateral lower limb amputations, or a single limb amputation with a stump measuring 7 inches or less (measured from the joint line to the tip of the stump) is considered to have a minimal (1/5) consequential relationship to the amputation(s).

4. This statistical relationship applies to the traumatic amputations and is unrelated to amputations which have arisen as a result of a disease process.

2. MONCKEBERG'S SCLEROSIS

This condition is more accurately termed medical calcification of muscular arteries. It is a degenerative process related to aging which involves the media (muscular cost of the artery) and the internal elastic lamina.

The arteries affected are those of the head, neck and extremities. The affected arteries become palpably hardened due to the calcification of the muscular layer, and exhibit pipe-stem rigidity with often a lumpy sensation on palpating the arteries which is termed beading. This beading is due to irregular calcification of the affected layers of the arteries.

This condition does not produce the clinical signs and symptoms normally associated with arteriosclerotic disease and is not considered to be related to hypertension.

The affected arteries may have concurrently atheromatous change which does produce the symptoms and signs of clinical arteriosclerotic disease. The presence of the medical calcification change in the arteries does not necessarily mean that the affected arteries will show atheromatous change and vice versa, despite the fact that both of these changes are related to the natural aging process.

3. ARTERIOLAR SCLEROSIS

In this condition there is a thickening of the wall of the small arteries which results in a narrowing of the vessel lumen. It affects the arteries of a smaller order than the "small" arteries affected by atheroma such as the coronary and cerebral vessels.

The most frequent occurrence of this type of arteriosclerosis is in the kidneys, spleen, pancreas, and adrenal glands. The two major etiological factors in its development are -

1. The aging process.

2. Hypertension.

Hypertension may produce the lesions, particularly in the older age group, but the findings may be present without any evidence of hypertension.

Any disability resulting from this type of arteriosclerosis would not normally be recognized as arising from arterial disease. Usually the disability would present itself as being a disease of the organ affected.

The organ affected would be diagnosed on an organic basis and any ruling made, and disability assessed would be related to the diagnosis of the condition affecting the organ involved. In almost all cases the diagnosis is made only on microscopic examination of the affected tissues and can not be recognized clinically as due to the arteriosclerotic process.

This disease results from the inhalation of significant quantities of asbestos fibres and can present decades later several ways.

Pulmonary Fibrosis

A diffuse type of interstitial fibrosis which is quite non-specific and if sufficiently advanced may cause a restrictive type of breathing defect.

Benign Pleural Disease

Transient and often asymptomatic pleural effusions can occur. More commonly pleural fibrosis which may rarely be diffuse, or more commonly localized as pleural plaques. These plaques have a distinctive radiological appearance and are presently regarded as markers of previous asbestos inhalation. The latency period is about 20 years.

Malignant Pleural Disease

Mesotheliomas occur after a latency period of 30-40 years and the original exposure to asbestos inhalation can be quite brief as compared with other manifestations of the disease, e.g. as little as a month working in the asbestos industry.

Other Lung Cancers

All types of bronchogenic carcinoma are more common where there has been previous significant exposure. The risk increases directly with the duration and intensity of exposure. The risk is greatly intensified if the person is also a smoker.

PENSION CONSIDERATIONS

1. The question often arises as to whether the claimed exposure was sufficient to cause the manifestation of asbestosis under discussion:

    a) In the case of mesothelioma; any exposure is probably sufficient.

    b) In the case of pleural plaques; their very existence is proof of exposure.

    c) In the case of diffuse pulmonary fibrosis; casual exposure to possible asbestos fibres in the air such as might be experienced by workers in a building insulated with asbestos, or sailors on a ship, is thought to be insufficient. By contrast, sailors employed as pipefitters or in the engine room or shipyard workers might achieve sufficient exposure.

2. It is important to realize that the finding of diffuse pulmonary fibrosis by itself is non-specific and has many possible causes.

3. Pulmonary fibrosis causes only shortness of breath on exertion, and PFT show a restrictive pattern. It COPD co-exists, it usually reflects the person's smoking habits.

4. Pleural plaques are asymptomatic and not a pre-malignant condition. They are not a cause of disability.

Trauma to the head can be divided into:

1) injuries to the scalp only, e.g. lacerations, with no involvement of the skull or brain;

2) trauma severe enough to cause damage to the skull, but with no evidence of concussion or injury to the surface of the brain;

3) trauma severe enough to cause damage to the skull, with evidence of concussion and/or damage to the brain coverings or to the brain tissue itself.

A head concussion is defined as a usually reversible traumatic paralysis of nervous function which is always immediate; its effects on brain function may last for several seconds up to several hours. A concussion is manifested by loss of consciousness, suppression of reflexes, transient arrest of respiration and a brief period of bradycardia and hypotension.

In general, the more severe the initial head injury, the greater is the likelihood of sequelae therefrom; for example, injuries to the scalp only do not lead to neurological sequelae; simple concussion almost never leads to sequelae; skull fracture may or may not be associated with brain injury, whereas massive head injury will virtually always result in permanent sequelae.

It must be noted that signs of brain injury tend to improve as the months pass; it is only when the situation has stabilized and deficit has become "fixed", i.e. permanent, that one may speak of sequelae and assess the residual disability.

Sequelae of severe head injury include:

1. Impairment of motor function;

2. Disturbance of speech;

3. Post traumatic epilepsy, which occurs in 20% to 40% of patients with severe head injury. The interval between the head injury and the first seizure is usually between three and nine months.

4. Organic brain syndrome, with intellectual impairment and behavioral abnormalities.

5. Post traumatic syndrome: this is also referred to as "post-traumatic nervous instability" and "post-concussional syndrome" amongst other terms. Although it is not clearly defined, nor is it uniform in its manifestations, nor is there a recognized pathophysiological basis for it, the existence of the syndrome cannot be denied and it is often the most troublesome sequela of head injury in terms of treatment; it is manifested primarily by headache, which is often localized to the site of the original trauma, along with dizziness, nervousness, fatigue and inability to concentrate, in the absence of demonstrable intellectual impairment. This syndrome may persist for months or years, and its intensity and duration are augmented by compensation problems and litigation. Psychological disturbances are inevitably present.

6. Post-traumatic psychiatric disorders: these are very rare, and include organic personality disorder and organic affective disorder.

Definition:

1. Carcinoma is the term applied to malignant new growths arising from an epithelial tissue. Such growths harm the host through their invasive qualities and their tendency to metastasize throughout the body.

2. Carcinoma of the larynx arises in the epithelial lining of the larynx. It is of world-wide distribution affecting all races. It commences insidiously as a change in character in a cell or group of cells of microscopic proportions and quite unrecognisable by the naked eye. This cell or group of cells proliferates into a mass which in time gives rise to symptoms. According to its site, it is classified as supraglottic, glottic and subglottic, the vocal cord being the commonest site of origin. Subglottic carcinoma is rare. Papilloma, hyperkeratosis and leukoplakia are regarded as precancerous conditions.

Clinical Manifestations:

The dominant symptom is hoarseness of the voice and this occurs at an early stage where the tumour arises from the vocal cords. In the supraglottic and subglottic varieties, the early symptoms are slight. There may be discomfort in the throat increased by swallowing and sometimes a swelling in the neck due to metastasis may be the presenting symptom. At times, subglottic lesions remain symptomless until involvement of the vocal cords causes hoarseness of blocking of the airway causes respiratory distress. As the disease progresses there may be complete loss of voice, difficulty in swallowing, airway obstruction and referred pain to the ear accompanied by general cachexia. In most instances the diagnosis can be made by clinical examination, including laryngoscopy and biopsy examination.

Aetiology

1. Carcinoma of the larynx constitutes about 2% of all reported cases of malignant disease. The disease affects men about 7 times more often than women, though there is some evidence that the incidence in women is increasing. It is essentially a disease of the elderly, affecting mainly the sixth and seventh decades of life. The highest incidence is among Asian races, particularly Indians.

2. No definite causes are yet known, but it is likely that chronic mucosal irritation by heavy smoking, excessive alcohol (especially spirits), and the chewing of tobacco or aromatic nuts play a significant part in its aetiology. It has been shown that metaplasia and malignant changes in human laryngeal epithelium develop in proportion to tobacco smoke exposure and that the changes may be reversed by abstention. Very prolonged vocal strain with severe chronic laryngitis and also previous irradiation of the neck may be additional contributory causes, though chronic respiratory infection alone is not thought to play any part. A highly significant association has been noted between the disease and asbestos dust exposure. Authoritative opinion is agreed that the disease is not caused or influenced by such factors as trauma, climatic exposure, hardship, stress or diet.

Conclusion:

The disease is of unknown aetiology. Possible aetiological factors are mentioned above. Environmental factors do not otherwise influence the cause or progress of the disease.

References:

Shaw H. Tumours of the Larynx. In: Ballantyne J and Groves J, eds. Scott-Brown's Disease of the Ear, Nose and Throat; Vol 4, 4th ed. London; Butterworths: 1979; 421-508

Definition

1. Malignant or neoplastic change (synonym cancer) can affect many tissues of the body. It occurs in all countries, affects all races and is one of the commonest fatal diseases of man. It commences insidiously as a change in character of a cell or group of cells which proliferates and eventually forms a recognisable mass. It may invade neighbouring structures, and cells may be carried via lymphatics or the blood stream to form remote metastases. In its early stages its presence may be compatible with apparently perfect health, and an advanced stage may be reached before symptoms are noticed.

2. Malignant mesothelioma is a type of neoplasm which arises from the serous membranes of the thoracic cavity (the pleura) or of the abdominal cavity (the peritoneum).

Clinical Manifestations

1. The onset of pleural mesothelioma is usually insidious, with the disease well advanced before it causes symptoms. Pain or discomfort in the chest is the most frequent presenting symptom. In advanced disease breathlessness, dry cough, wasting and pleural effusion occur.

2. The onset of peritoneal mesothelioma is even less clearly defined. Vague discomfort may be followed by anorexia and constipation. Eventually ascites causes abdominal distension, and intestinal obstruction is likely to occur.

Aetiology

1. Mesothelioma of the pleura and peritoneum is known to be related to previous inhalation of asbestos fibres. Evidence that asbestos has been inhaled at some time is afforded by the presence in the sputum of characteristic asbestos bodies. These bodies may be detected for many years after exposure to asbestos has ceased, and have no other significance than providing evidence of past inhalation. Such bodies, or bodies of similar appearance, have been found in the lungs of a high proportion of urban dwellers with no known industrial exposure to asbestos and with no asbestos related disease. No other aetiological agent for malignant mesothelioma has been demonstrated, but occasional spontaneous cases are believed to occur.

2. Crocidolite is the most dangerous type of asbestos and chrysotile the least, but the use in industry of a single fibre type is rare. Occupational exposure may occur in asbestos mining, in the asbestos processing industry, and in the installation or removal of asbestos insulation material. Para occupational exposure may affect workers in the vicinity of an asbestos hazard. Mesothelioma has occurred in housewives apparently in relation to washing the asbestos contaminated overalls of members of their households. Asbestos fibres, derived from natural or man made sources, are widespread in the atmosphere. There is however, no evidence that the concentration of air borne asbestos to which the general public may be exposed causes mesothelioma, though it is probably responsible for the frequency with which asbestos bodies are found in town dwellers.

3. Mesothelioma may follow exposure to asbestos, but may also follow periods as short as a month of more intense exposure. The latent period before the tumour develops is very long, generally more than 20 years. Cigarette smoking does not appear to affect its occurrence. The course of the disease is not influenced by external factors, and at the present time no effective treatment is known.

Conclusion

1. Malignant mesothelioma is a rare disease affecting the pleura or peritoneum. It is known to be associated with exposure to asbestos dust. The course of the established disease is not affected by external factors.

References

Wagner JC. Diseases Associated with Asbestos Dusts. Practitioner 1979; 223: 28-33.

Gloag D. Asbestos - Can It Be Used Safely? Asbestos Fibres and the Environment. Br. Med. J. 1981; 282: 551-553, 623-626

Parkes W.R. Asbestos-related Disorders. Br. J Dis Chest: 1973: 67: 261-300.

MEDICAL GUIDELINES **CEREBROVASCULAR DISEASE**

This term designates any abnormality of the brain resulting from a pathologic process of the blood vessels; the commonest of these processes are cerebral thrombosis, cerebral embolism, and intracranial hemorrhage.

Cerebral Thrombosis refers to abnormal deposition of platelet aggregates (thrombus) within an arterial wall, with subsequent organization into a significant plaque which narrows or occludes the vessel, resulting in ischemia or infarction in the territory supplied by the artery, in this case a specific area of the brain.

Cerebral Embolism refers to the transportation of a foreign body (embolus), from one part of the circulatory system to another where it becomes impacted; most commonly the embolus is a thrombus which has formed either in the heart or in a blood vessel wall and has subsequently become detached; thus, a thrombus may dislodge and become impacted in a cerebral artery, resulting in cerebral embolism, with ischemia or infarction in the territory supplied.

Intracranial hemorrhage refers to massive bleeding within the skull. It may cerebral, i.e., into the brain itself, or within the coverings of the brain, in which case it is referred to as subarachnoid, subdural or epidural hemorrhage causes damage by way of direct or indirect pressure of extravasated blood on adjacent or underlying brain structures.

The most frequent mode of presentation in cerebrovascular disease is the stroke, defined as a sudden, non convulsive focal neurologic deficit; this deficit, in turn, reflects the size and location of the cerebral infarct or hemorrhage. Commonly encountered manifestations include hemiplegia, mental confusion, sensory loss, visual field defects, diplopia, aphasia and ataxia.

Stroke may or may not be preceded by warning attacks with transient neurological manifestations lasting from a few minutes to an hour (transient ischemic attacks). These warning signs are more common in the case of thrombotic strokes than in embolic or hemorrhagic strokes.

Most thrombotic strokes are due to arteriosclerosis, and the arteriosclerotic heart disease and peripheral vascular disease, in that it has the same underlying mechanism and the same risk factors.

Embolic strokes most commonly arise when a fragment has broken away from a thrombus in the heart; less often, the source of the embolus is an arteriosclerotic plaque in a major artery, usually the common carotid or internal carotid artery. Embolic strokes occur somewhat more suddenly than thrombotic strokes and are not usually preceded by warning attacks.

Intracranial hemorrhage is the third most frequent cause of stroke, and it can have several causes, the commonest of which are:

1. primary cerebral hemorrhage, usually in association with hypertension;

2. ruptured berry aneurysm, which results from developmental defects in the arterial wall.

Once the cerebrovascular accident (stroke) is complete, the assessment is based on the extent and severity of the permanent neurological deficit; this is so variable as to preclude any form of tabular guideline, and every case must be assessed individually.

BACKGROUND INFORMATION

At or shortly after enlistment in the Active Force during World War II the recruit received an examination by a member of the Canadian Dental Corps. This dentist recorded:

1. The number of teeth missing and the evidence of past treatment.

2. The treatment required for the remaining teeth.

3. The state of oral hygiene and periodontal tissue.

DENTAL TREATMENT WAS AVAILABLE THROUGHOUT THE VETERAN'S SERVICE. Other than the first division that went over seas almost immediately, many units were dentally fit on overseas posting. Through lack of appreciation of the need of dental care or, from fear, a man could occasionally avoid treatment. This was rarely the case as the dentist could have him paraded for necessary care and his dental documentation would show his refusal of treatment.

CONDITIONS DURING SERVICE

Living conditions and diet in Canada and England during World War II were as a rule excellent and cannot be considered to have contributed to dental disease. Medical and dental services were readily available. When actively engaged with the enemy in the European Theatre of War, personal hygiene often became difficult. However, these periods were not so prolonged that major damage could be done to the dental tissues. Service in the Far East and confinement to POW Camps, especially in the Far East, created situations wherein personal care was not always possible, dental treatment was not available and, in some instances, dietary deficiencies and tropical diseases took their toll of the dental structures. As a consequence of the known vitamin and nutritional deficiencies, all pensioners who were prisoners of the Japanese are allowed full dental care by the Treatment Services of the Department of Veterans Affairs for the balance of their lives.

AT DISCHARGE

1. In the early years of the War, when a veteran was released from service, he/she received complete dental care and any necessary dental work, including the provision of prothesis by the Canadian Dental Corps.

2. In the later years of the War and at demobilization the amount of work was far too great for the Canadian Dental Corps to handle, and the Treatment Services Branch of the Department of Veterans Affairs supervised the provision of dental care after discharge which was termed Ex-Defence Dental Treatment. The veteran received notification of his treatment rights for dental care for one year. He was told to contact a civilian practising dentist who would recommend the necessary treatment and forward his recommendations to the Department of Veterans Affairs Treatment Services Branch. On approval by the Department, the dentist would proceed to provide the necessary treatment, including provision of dental prostheses. This brought the veteran's dental state into a condition at least as good as, and frequently superior to, that when he enlisted.

3. Some veterans, through lack of interest or appreciation of the need for treatment, did not take advantage of this rehabilitation program.

POST-DISCHARGE

In the post war years the dental condition could deteriorate depending on the amount of care the veteran took of his teeth, declining health from age and medical conditions which affect the population as a whole. It is most difficult, many years after wartime service, to relate presently found dental conditions to dental disease well treated during World War II service.

DISEASES WHICH COMMONLY AFFECT THE DENTAL TISSUES

Dental Caries

This is a hard tooth tissue destructive disease of civilization which has a high incidence between the ages of sixteen and twenty-five, a common military age. It is a slow, progressive, widespread disease which if untreated may result in loss of teeth. The process is characterized by decalcification, softening, decay and loss of tooth substance. The Dental Profession agrees that caries is a generalized disease of teeth being related to diet, heredity and personal oral hygiene. Dentists also agree that early caries in teeth was frequently missed in World War II where routine x-rays were not taken, as many early caries are not visible or detectable by a dental probe. Dental caries were almost always present, to some degree, and frequently extensive, on enlistment in World War II as dental care had been widely neglected during the Nineteen Thirties depression years. When pre-enlistment caries was severe, extractions were required when the teeth could not be saved and bridges and dentures were supplied when necessary during his service. In effect, during service all necessary dental care to maintain dental health was supplied. The individual could not be forced to accept the treatment offered except when acute conditions prevailed, but treatment was rarely refused.

Gingivitis

This is an inflammatory condition of the gums, or soft tissues, caused by neglected oral hygiene and aggravated by the excessive use of tobacco.

Periodontitis Simplex (Pyorrhoea)

This condition follows a neglected gingivitis and produces an infective condition of the gingivae (or soft tissues around teeth) with pockets of pus forming between the gingivae and the tooth in many cases. It is caused mainly from lack of good oral hygiene and otherwise occurs under conditions of privation and inadequate diet.

Necrotic Gingivitis, Vincent's Infection, Acute Ulcerative Periodontitis, Trench Mouth, Etc.

These are all synonyms for an acute inflammatory disease of the oral mucous membranes. The teeth become painful, with haemorrhages in the gingivae, saliva has a metallic taste and is excessive; there is a necrotic odour, temperature ranges from 100 to 102 degrees and malaise is quite evident. Most such patients were hospitalized, and rarely treated as outpatient, but some did receive outpatient care only, and were relieved of duty while treated. It is the opinion of the

Veterans Affairs Canada Dental Consultant that the use of "V: treatment in the Canadian Dental Corps reports during wartime was not necessarily treatment for Vincent's Infection, but was an anti-bacterial treatment for any inflammatory condition involving the gingivae or periodontal tissues. His opinion is the "V" treatment record, in which the man was not hospitalized, or taken off duty, does not justify a diagnosis of necrotic gingivitis.

RECORDS

1. Examination on enlistment showed the number of teeth missing, the number of teeth affected by caries and the state of periodontal tissues.

2. The record of dental treatment during service showed not only the loss of teeth during service, but the man's own desire to improve or maintain his oral health.

3. The examination at discharge, usually a cursory one, showed the dental and oral health. The dental state at discharge from service can be readily compared with his state at enlistment in most cases.

4. The Ex-Defence Dental Treatment provided by the Department of Veterans Affairs Treatment Services, for a period of one year following discharge, was extended under certain circumstances (e.g., in those pursuing veteran's education). The treatment, provided by a civilian dentist, was the dental treatment necessary to maintain, or restore the oral health to a condition as good, if not superior to that on enlistment.

5. When there is a request for ruling in respect of loss of teeth or a dental condition, an effort should be made to obtain evidence that there was continuing treatment necessary and that dental care had not been neglected during the postwar years subsequent to the Ex-Defence Dental Care. Recent examination is required to determine the present state of the veteran's dental health.

PENSION CONSIDERATIONS

It is generally accepted by the Dental Profession replacement of missing teeth by an adequate professional dental prosthesis (fixed or removable, partial or complete) does not represent an assessable disability.

Dental Caries and Gingivitis

These two conditions are very common in our modern society and are considered to result from diet, heredity and personal dental hygiene. Most teeth lost during service is World War II were carious on enlistment and despite treatment could not be saved. Routine x-rays of teeth were not done during service in World War II so that early caries was not detectable and on visual and probe examination was not always identified. Most of these areas of caries would develop into visible caries during service and would be treated adequately. Certainly as caries is a generalized disease of the teeth new caries could develop in any veteran and the veteran might lose the affected tooth (or teeth) either during service or subsequent to service.

Protheses were supplied both by the Canadian Dental Corps during service and by the Ex-Defence Dental Care by the Department of Veterans Affairs Treatment Services following discharge. Each serviceman was notified of the dental treatment provided by Treatment Services, but not all veterans took advantage of the service.

The veteran was, therefore, supplied with a prosthesis(es), adequate and serviceable by professional standards, so that on completion of his treatment it was not considered that he had a disability following his discharge. The decision by a veteran that he/she did not wish to wear such a professional prosthesis is not deemed to be a bonafide disability.

Loss of Teeth Resulting from Injury Without Loss of Bone

A prosthesis will restore an individual's ability to masticate. This would leave the veteran without an assessable degree of disability. It is considered entitlement should be granted for the purposes of any necessary dental treatment for the loss of teeth from injury.

Loss of Teeth Resulting from Injury With Loss of Bone or Deformity of Bone

Depending on the extent of such loss of bone, or deformity, and the degree of malocclusion, if present, an assessable degree of disability can usually be identified. Entitlement should be granted for continuing dental care even though there may be a minimal or no assessable disability.

Necrotic Gingivitis (Vincent's Infection)

Vincent's Infection, in most cases, was treated successfully during the service period. In most cases, at discharge there was no loss of teeth resulting from the infection and the teeth were considered to be in a normal state and there would be no reason to consider that Vincent's Infection had resulted in any disability or later loss of teeth. When the claims are made at the present time for the results of Vincent's Infection it would have to be shown that teeth have been lost due to regression of alveolar bone more than one would expect from age alone. Only if the record shows infection persisted in the post discharge period, with the necessity of extended dental care at that time, should entitlement be recommended.

European Prisoners of War

Confinement of veterans in European Prisoner of War Camps limited their ability to maintain oral health and it could be expected that with inadequate dental care, caries might progress depending to a great degree on the length of the incarceration. Loss of teeth could well result, which under ordinary service conditions would have been treated and preserved by the Canadian Dental Corps Treatment. In large part nutrition was adequate though limited in European Prisoner of War Camps, but rarely was vitamin deficiency encountered.

As the susceptibility to dental caries varies widely from individual to individual the records would show the evidence of unexpected loss of teeth. Veterans Affairs Canada will accept applications for "Loss of Teeth Due to Dental Caries" from European Prisoners of War, and will rule on an individual basis and, when justified from the record, grant full treatment rights for dental care.

Prisoners of the Japanese

As noted in "CONDITIONS DURING SERVICE" on Page 1.

Due to recognized nutritional vitamin deficiencies, all prisoners of war of the Japanese who receive the benefits of an award under Section 71.2 of the Pension Act are provided full dental care by Treatment Services of the Department of Veterans Affairs.

REGULAR FORCE SERVICE

1. **Dental Caries With Loss of Teeth**

   Progression of dental caries during Regular Force service is also dependent upon heredity, diet and personal dental hygiene. The Armed Forces provide an adequate diet, routine professional dental care and provision of professional prosthesis, when required, during service and at discharge. No disability is considered to exist when teeth lost from caries are adequately replaced by a professional prosthesis.

   There is no justification for considering that loss of teeth from caries arose out of or was directly related to Peacetime service duties.

2. **Gingivitis and Necrotic Gingivitis**

   As these are inflammations, they cannot be considered to have arisen out of or have been directly related to Peacetime service duties.

3. **Loss of Teeth Due to Injury**

   When the loss of teeth due to injury is considered to have occurred on duty, this would allow for entitlement for treatment purposes for the upper and/or lower jaw.

Assessments, except the first one, should never be made as the result of one examination. The file should be reviewed over a two year period if possible, to obtain the overall trend of improvement or worsening before raising or lowering an assessment.

In some types of skin conditions where there is known to be seasonal variation, review dates for examinations should be arranged to show the skin condition at its best and at its worst, e.g., psoriasis.

Skin conditions, where they are unsightly on exposed parts of the body, for cosmetic as well as prohibitive reasons, e.g. in certain trades, the hands for prohibitive and cosmetic reasons, and the face for cosmetic and social reasons, rate a comparatively higher rate of pension, than would the same type of skin lesion covered up by clothing.

Pruritis is another factor to consider and would be pensioned according severity and area involved. Weeping or excessive scaling and soiling of clothes is a further factor.

Ordinarily, assessments for skin conditions vary from 1% to 20%.

Any skin condition of the face is worth 5% as a minimum if it interferes with the veteran's appearance at all in the social and labour world. The same amount anywhere else in the body might rate 1-3% covered up. Very seldom is a face rated more than 20%, however.

Skin conditions on genitals, particularly weeping or scaling ones, would rate more than elsewhere for obvious reasons. 5-10% for small lesions; 15-20% for more or less total involvement.

Pruritis ani and moist conditions about the anus mostly rate 5% on the average with very severe ones 10 - 20%.

Multiple lesions all over the covered areas of the body, 5-20% as an average, depending on number of exacerbations, weeping and scaling, or continued chronicity and size of lesions and amount of associated irritation.

Ordinary athlete's foot or hyperhidrosis entitles an award of 1% to 10%; very bad cases, 15%. Involvement of the hands in minor degree with contact dermatitis, 5%, and this would hold for psoriasis, or any dry or weeping eczema of mild degree, not subject to severe exacerbations. Severe exacerbations and frequently recurrent exacerbations of dermatitis of the hands up to 20%.

Seldom does any skin condition rate more than 20%, but severe weeping pruritic lesions over large areas of the body, including genitals and anus or face, can go as high as 60-70% depending on recurrency rate or chronicity as well as area.

Diabetes mellitus is characterized by disturbed glucose metabolism resulting in inappropriate hyperglycemia (elevated blood sugar). It is due to an absolute or relative deficiency of the insulin hormone produced by the pancreas.

Diabetes is generally considered in two categories. Type I diabetes or insulin dependent diabetes mellitus (I.D.D.M.) depends on insulin for the prevention of ketoacidosis. Type II diabetes or non-insulin dependent diabetes mellitus (N.I.D.D.M.) does not depend on insulin to ward off ketoacidosis. Type I diabetics therefore require insulin in their treatment while Type II diabetics may be successfully treated with dietary control, weight control and possibly oral medications.

The familial nature of diabetes has been known for centuries. Current medical studies seem to indicate that in Type I diabetes an inherited susceptibility to the disease is genetically determined. Some medical studies seem to indicate that when susceptible persons are exposed to certain viruses latent diabetes may become manifest. Data, however, are thus far inconclusive and there are many other factors involved.

Type II diabetes shows a stronger familial inheritance. Identical twins are nearly 100% concordant for the disease. However, environmental factors such as obesity and diet seem to play a role in the expression of this disease.

Pension Considerations:

1. Diabetes mellitus is a constitutional disease with an inherited predisposition.

2. There is no reason to relate primary diabetes mellitus to any factors encountered during periods of service.

3. Secondary diabetes mellitus may result from pancreatitis, pancreatic resection, severe trauma, an infiltrative disease (e.g. carcinoma, hemochromatosis, or cystic fibrosis) or a disease that alters hormone levels (e.g. Cushing's syndrome, pheochromocytoma, Conn's syndrome). In such cases, diabetes could be the result of a specific factor encountered during service, e.g. trauma, or may be considered consequential to another disease process or pensioned conditions.

4. Certain medications, e.g.; pentamidine, cortisone, birth control pills, and other may in certain instances contribute to a diabetic state but are not considered causative of diabetes mellitus. However, they may be aggravating factors and if such medications are taken on a chronic basis, for pensioned conditions, then diabetes mellitus might be considered as minimally consequential to such.

These guidelines are divided into four sections as follows:

1. DISC DISEASE (GENERAL)

2. LUMBAR DISC DISEASE

3. CERVICAL DISC DISEASE

4. DORSAL DISC DISEASE

1. DISC DISEASE (GENERAL)

Intervertebral Discs

The vertebral bodies throughout the spine are separated from one another by intervertebral discs except in the case of the first and second cervical vertebrae, sacrum and coccyx. The disc is a highly resilient structure which allows greater motion between vertebral bodies than if the bony surfaces were directly in contact. Because of its elastic nature, the disc also functions as a shock absorber when the spine is subjected to vertical compression force.

There are two components, the annulus fibrosus and the nucleus pulposus. The annulus forms the outer boundary and is composed of fibrocartilaginous (mainly fibrous) tissue, arranged in concentric rings in which the fibres run obliquely from one vertebra to the next. The fibres in successive layers slant in alternate directions. The peripheral fibres pass over the cartilaginous end plates to unite with the bone of the vertebral bodies. The annulus essentially is thicker anteriorly.

The nucleus pulposus, situated between the middle and posterior thirds of a disc is highly elastic, consisting of collagen fibres in a muco-protein gel. The fluid behaviour of the nucleus pulposus converts a vertical pressure to a horizontal thrust and hence the energy is extended to the annulus fibrosus.

Intervertebral discs account for about 25% of the length of the vertebral column above the sacrum. This proportion decreases with the degeneration of aging.

Manifestation of Disc Disease

Degenerative changes occur in the intervertebral disc during a life-time, but these natural changes, in the absence of clinical symptoms, are not evidence of disability.

Disc disease manifests itself in three ways:

1. Symptoms from changes in the disc itself resulting in restricted mobility.

2. Symptoms from complications of disc disease due to protrusion of the disc causing nerve root pressure or cord pressure.

3. Bony changes with the formation of osteophytes which may completely bridge the disc space and limit movement, or impinge on spinal nerve foramina to cause root entrapment.

Clinical Terminology

The following terms apply to disc disease:

Disc Degeneration

The pathophysiological changes which occur within the disc components involve both the nucleus pulposus and the annulus fibrosus.

Disc Protrusion of Herniation

The nucleus pulposus may bulge through, protrude through, or actually be extruded through the annulus. In the latter case, one refers to sequestration of disc material.

Osteoarthritis

This term is applied when hypertrophic osteophytes are seen radiologically in relation to the margins of the vertebral bodies or apophyseal joints, and from Veterans Affairs Canada's point of view the term is synonymous with moderately advanced degenerative disc disease.

Spondylosis

The term spondylosis is often used synonymously with late stage disc disease. The term is usually applied in reference to the cervical spine. It denotes disc degeneration with radiological findings as outlined under the term osteoarthritis.

2. **LUMBAR DISC DISEASE**

Lumbar Disc Disease, like osteoarthritis and arteriosclerosis, is fundamentally a natural degenerative condition associated with the ageing process, commencing early in life and progressing steadily thereafter. In any individual, the rate of this progression is determined mainly by constitutional factors. Trauma may alter this natural process.

The absence of x-ray evidence of disc disease does not exclude the presence of degenerative changes of sufficient degree to cause disability. On the other hand, x-ray evidence of disc degeneration may be present without any clinical symptoms of disability or actual disc disease.

The following pathological facts must be clearly understood:

(1) Disc degeneration starts at a very early age. At birth, the nucleus of the disc is a well-defined structure clearly demarcated from the annulus of the disc. By the second and third decades, the border between the nucleus and annulus is less well-defined. Fibrous elements are more prominent. There is a progressive cavitation and desiccation of the nucleus in the fourth and fifth decades and the condition is more frequently symptomatic. At the same time, radial cracks occur in the annulus most frequently at the postero-lateral margins of the disc space.

2. **LUMBAR DISC DISEASE**...CONTINUED

(2) A healthy normally functioning disc can withstand vertical stresses of approximately 600 kgs. Severe vertical pressure alone would cause disruption of the vertebral plate or a compression fracture of a vertebral body before it would cause rupture of a healthy disc.

When the force is applied in flexion, it is estimated that there is an increase in the stress on the disc of approximately two and a half times that which occurs on vertical compression alone. A rotational element in the movement further increases the risk of injury to the disc.

When advanced degeneration is present, rupture of the disc may occur from a vertical load of approximately 200 kgs.

(3) The relative importance of degenerative change and injury causing clinical disability varies with age and with individual factors. In a small percentage of cases, perhaps 5% of persons under 55 years of age, a severe injury could be held totally responsible for the disability (regardless of the presence of pre-existing degenerative changes). It has been estimated that 75% of people in the older age group have some low back disability due to disc instability resulting from normal degenerative changes.

De Palma and Rothman, in their book "The Intravertebral Disc", outline the relationship between degenerative changes and trauma in following manner:

> "Disc degeneration is not usually due to one insult, but rather to the combined ravages of the biochemical and mechanical changes of ageing, associated with longstanding mechanical stress. A history of injury which may have precipitated a low back syndrome may often be elicited, but this injury has played an incidental role in what is truly a chronic degenerative process."

It is thus apparent that the natural history of the progressive degenerative changes in the discs must be taken into account in determining what fraction of the disability can reasonably be attributed directly to service. Service factors may cause aggravation (permanent worsening) of the degenerative process. The degree of aggravation is expressed in fifths.

(1) Some degree of disc degeneration is already present at the time of enlistment although the condition would usually be asymptomatic.

(2) Disc Disease is practically never obvious on enlistment as this term is defined in the Pension Act.

2. **LUMBAR DISC DISEASE**...CONTINUED

(3) Information given on enlistment or subsequently during service concerning previous low back or sciatic pain can beyond reasonable doubt be considered evidence of disc disease, if corroborated by the finding of clinical or radiological evidence of disc degeneration or disease within ten years, provided only that there is no objective evidence of any other cause of the same low back or sciatic pain.

(4) The degree of aggravation which can be assessed under subsection 21(1) or the degree of service relationship under subsection 21(2) of the Pension Act varies with:

(a) **The Presence or Absence of Symptoms or Signs Prior to Injury**

If, prior to an incident, disc disease has been symptomatic and/or there is x-ray evidence of degeneration, the degree of aggravation recommended will be usually less than when the incident initiates the first manifestation of the condition.

(b) **The Age at Which the Claimed Injury Occurred**

A given injury precipitating symptoms in a 20 year old person warrants a higher degree of aggravation than a comparable event initiating symptoms at 40 years of age. This is because in a younger person the discs and supporting soft tissues have not been affected by the degenerative changes to the same degree and, hence, are more resistant to injury.

(c) **The Severity of the Precipitating Incident or Injury in Relation to the Onset of Symptoms at Any Age**

(i) Normal service activities precipitating symptoms, are not aggravating factors beyond those accepted as part of the normal wear and tear of living and aging.

(ii) A severe unexpected injury involving flexion and rotational components would cause greater aggravation than injury under controlled conditions such as a deliberate lift.

(iii) Direct trauma, in the absence of flexion compression and rotational components, rarely is a causative or aggravating factor in disc disease.

(d) **The Time Interval Between the Injury and the Onset of Continuing Disability**

(i) An isolated episode of back strain which causes an acute disability limited to the back, for 3 weeks or less, and is followed by an asymptomatic period of 5 years, is considered to be a self-limiting soft tissue injury. Soft tissue injuries do heal and, if followed by a 5 year asymptomatic interval, are not considered to aggravate the normal degenerative process.

2. **LUMBAR DISC DISEASE**...CONTINUED

(ii) An asymptomatic interval of less than 5 years before the diagnosis of disc disease probably indicates some degree of aggravation, and the degree will be determined by the level of disability found and the time interval before diagnosis.

(iii) Recurring or continuous symptoms which lead to continuous disability and surgical intervention probably indicate a greater degree of aggravation.

DEVELOPMENTAL AND TRAUMATIC CONDITIONS OF THE LUMBOSACRAL SPINE AND THEIR EFFECT ON LUMBAR DISC DISEASE.

1) SPINA BIFIDA PER SE does not contribute nor predispose to the development of lumbar disc disease.

2) Sacralization of the lumbar spine and lumbarization of the sacral spine may accelerate lumbar disc degeneration to a mild degree.

3) Spondylolysis and spondylolisthesis by their nature cause instability of the lumbosacral spine. This instability has an adverse effect on the disc immediately below the displaced vertebrae and influences the development of degenerative changes to a moderate to severe degree.

4) A fixed scoliosis in the lumbar spine may influence the development of degenerative changes to a minimal degree.

5) Fracture of the vertebral body is considered to severely affect development of degenerative changes. Such changes occurring in the area of a fractured vertebra should be considered part and parcel of the entitlement and included in the assessment. A fractured transverse process, on the other hand, is caused by a different type of injury than an injury that would cause disc damage. A transverse process fracture is caused by a lateral flexion injury and not the flexion-rotation injury that normally damages a disc. Lumbar disc disease therefore, cannot be considered consequential to an injury that caused a fractured transverse process by itself.

LOWER LIMB DISABILITY RELATED TO LUMBAR DISC DISEASE

Certain conditions of the lower extremities adversely affect the mechanics of the lumbar spine.

i) **Leg Length Discrepancy**

(a) It is now recognized that a difference in leg length of even 1.5 cm may alter the mechanics of the lumbar spine and aggravate the normal degenerative changes. This may be a true shortening as a result of a fracture, or a functional shortening as may be due to a fixed flexion deformity of the hip or knee joint.

(b) Below knee amputations may create a functional shortening and hence an aggravation of lumbar disc disease. An above knee amputation causes further strain because in addition to the lateral strain from shortening, there is also a rotation component and, hence, these cases merit a higher degree of aggravation.

2. **LUMBAR DISC DISEASE**...CONTINUED

ii) **Other Conditions of the Lower Limb**

(a) Fusion of the hip, knee or ankle, mainly on the basis of functional shortening, may aggravate lumbar disc disease.

(b) Conditions which alter gait (with the rare exception of a severe chronic gait abnormality) without an actual shortening or associated functional shortening are not in general considered to aggravate lumber disc disease.

(c) Foot conditions such as pes planus, pes cavus, hallux valgus and the like are not considered to aggravate lumbar disc disease.

Conditions listed above that are considered to possibly influence disc disease, are only considered to influence degenerative changes in the lumbar spine. Dorsal and cervical disc disease are not considered aggravated by any conditions of the lower limbs.

MECHANICAL LOW BACK PAIN

Low back pain is a symptom and not a diagnosis, but has been used by consultants as a non-specific term referring to various conditions where no definite etiology has been established to indicate that the origin of the pain is musculo-skeletal, and not arising in, nor referred from other organ systems. Mechanical low back strain is a preferred term to indicate such a disabling condition (Lumbo Sacral Strain). Physicians should not use the term in cases of back pain that is believed due to early degenerative changes of the spine, particularly of the facet joints. In such cases the diagnosis may be difficult to confirm radiologically, but eventually almost all such complainants show progressive degenerative changes and the diagnosis becomes evident. In cases of claims for "mechanical low back pain" or "strain", the disability should be clarified, and a more specific alternate diagnosis sought before ruling and assessment. Because of the non-specific nature of the term "mechanical low back pain" it constitutes a symptom rather than a diagnosis. In cases of claims for such a non-specific diagnosis, a specific diagnosis and clarification of impairment are required for a ruling assessment.

LUMBAR STRAIN (CHRONIC LOW BACK STRAIN)

(i) Lumbosacral strain is considered to be a soft tissue injury involving only the ligaments and muscles of the lumbosacral region and not affecting the discs. Symptoms are confined to the lumbar region with no radiation beyond the buttocks. If there is radiation beyond the buttocks, then disc lesion is definitely suspected in addition to soft tissue injury.

(ii) Although most acute low back strains heal without any residual disabilities some cases persist with symptoms of chronic or recurring pain which is confined to the back and does not radiate beyond the buttocks. These cases would be ruled on under the diagnosis of chronic lumbosacral strain, (chronic low back strain).

2. **LUMBAR DISC DISEASE**...CONTINUED

(iii) A severe strain of the lower lumbar area, may in addition, involve the fibres of the annulus of the disc, as well as other soft tissues. The annular fibres may heal without residual disability but in some cases, the healing process may not be complete and a prolapsed disc may occur. In these cases, if clinical disc disease occurs within 5 years, the acute episode is simply an aggravating factor in accelerating the clinical condition of lumbar disc disease as described under lumbar disc disease.

(iv) When there is evidence of recurring low back strain, but no evidence of disc disease for 10 years, then it is considered that the initial injury damaged only the soft tissue (Lumbosacral Strain) and did not produce any injury to the disc. In these cases, there is no consequential relationship between the lumbosacral strain and the development of disc disease in later years.

3. **CERVICAL DISC DISEASE**

While the pathology of cervical disc disease is similar to that in the lumbar and dorsal regions, there are certain features which to a degree are specifically applicable to the cervical spine.

The prime function of the cervical spine is one of movement and the stress associated with movement is the major factor in causing degenerative change, in contrast to the stress of weight bearing which is the major factor in lumbar disc disease.

Cervical disc disease usually becomes apparent later than lumbar disc disease.

The osteophytes which arise as a result of disc degeneration are the main cause of root pressure and limited motion of the cervical spine. This is in contrast to the lumbar region where protrusion of the disc is the prime cause of minor root pressure even in the absence of hypertrophic changes.

4. **DORSAL DISC DISEASE**

The pathophysiology of dorsal disc disease is the same as in the lumbar and cervical regions. Because of the presence of the rib cage, there is limited motion in the thoracic spine. Symptoms due to dorsal disc disease are minor and only rarely is the disc degeneration complicated by protrusion and nerve root pressure.

DEFINITION

This is a condition of unknown etiology, which is characterized by progressive contracture of the palmar fascia of one or both hands. Rarely, it can also affect the plantar fascia of the feet, and very rarely the fascia between the structural portions of the penis.

The essential pathological change in the palmar fascia is that of areas of richly cellular connective tissue developing into scar-like tissue with contracture. This produces a thickening of the palmar fascia, usually restricted to certain areas; and this thickening progresses to a hardening and finally a shortening of the areas of the palmar fascia which are involved. There is an accompanying loss of subcutaneous fat beneath the skin and over the fascia involved, associated with the development of dense adhesions between the fascia and the skin. This eventually produces a dimpling, and fixation of the skin over the contracture, and flexion deformity of small and ring fingers.

ETIOLOGY

As in medical condition of unknown etiology, there are numerous theories as to the cause.

The following statements are generally accepted by medical authorities:

1. There is a hereditary predisposing factor in 25% to 50% of cases.

2. Several other etiological factors have been suggested, but the significance of these factors has not been resolved.

3. There is controversy regarding the effects of trauma in the etiology of Dupuytren's Contracture which has not been resolved. In 1974 the bulk of the evidence in the standard text books is that Dupuytren's Contracture is more common amongst people in occupations where there is not excessive manual activity.

4. It is not considered to be the result of infection or inflammation.

INCIDENCE

1. The condition occurs more frequently in males than in females in the ratio of approximately 5 to 1.

2. Bilateral involvement occurs in over 50% of cases, but not necessarily at the same time, and frequently at an interval of several years.

3. Where both hands are involved, the condition affects the right hand first in 64% to 75% of cases; the left hand first in 30% to 35% of cases.

4. It rarely occurs before thirty-five years of age, increasing in incidence to fifty years of age, and then the incidence levels off.

INCIDENCE...CONTINUED

5. It occurs most commonly in the palmar fascia at the base of the ring (fourth) finger over the metacarpophalangeal joint.

6. It may rarely affect the palm of the hand only, or the fingers only; but usually affects both areas.

7. The plantar fascia of the feet is so rarely involved that figures of incidence are not quoted by the authorities.

8. When deformities of fingers are present, it should be considered a late stage of the disease.

PROGRESSION OF DISEASE

The usual chain of events is that a nodule appears underneath the skin of the palm over the ring (fourth) metacarpophalangeal joint at the distal skin crease of the palm. This nodule is non-tender. Slowly, the process extends from the nodule distally to the fourth finger as well as proximally, and eventually shortening takes place, so that this finger is gradually flexed at both the metacarpophalangeal joint and the proximal interphalangeal joint. By this time, there is gross evidence of thickening in the palmar fascia, usually accompanied by a puckering of the skin at the point of the original nodule. The whole process from initial discovery of the nodule in the palm of the hand, up to the flexion of both joints of the finger usually takes up to five to seven years; but there is considerable variation in the rate of progression. By this time, commonly both the fourth and fifth fingers are affected. When the disease process is more extensive, it affects the other fingers, the middle, the index, and lastly the thumb, in that order, with accompanying more intensive involvement in the palm. Very rarely, the condition has been recorded as progressing far more rapidly and the contracture becomes fully developed in a matter of six months from the time it was first observed. However, it is also recorded that condition may become permanently arrested at any stage in its development.

PENSION CONSIDERATIONS

1. Obvious on enlistment - Dupuytren's Contracture would be obvious on enlistment only in its moderately advanced and advanced stages. If gross deformity in at least one digit is described within six months after enlistment, it should be considered that the condition was present on enlistment. Early stages would never be obvious and would be recognized only if the examiner's attention was especially drawn to the involved hand, for some other reason.

2. If Dupuytren's Contracture is found at enlistment and is worse on discharge, it should be considered aggravated during service.

3. If not described on enlistment, but found after one year or more service, it should be considered pre-enlistment, not obvious, and either aggravated or not aggravated during service.

PENSION CONSIDERATIONS ...CONTINUED

4. If not described on enlistment and if service is of short duration, the condition could be considered pre-enlistment, not obvious, and either aggravated or not aggravated during service.

5. When Dupuytren's Contracture is diagnosed as affecting one hand at discharge, it can be assumed that the surgeon would have looked carefully at the uninvolved hand. If he describes no evidence of the disease in the uninvolved hand, it should be assumed this hand was clinically normal.

6. If Dupuytren's Contracture is diagnosed as affecting one hand on discharge, with no involvement of the other hand, and if the unaffected hand develops diagnostic evidence of contracture within six months, it should be assumed that the process probably commenced during service.

   However, if the contracture develops in the unaffected hand after six months and before three years post-discharge, careful consideration should be given to the veteran's statement, the nature and length of his service and expert medical opinion.

7. With etiology unknown and with no definite evidence that trauma either causes or aggravates the condition, there appears to be no medical basis to consider that Dupuytren's Contracture could be directly related to or aggravated by Peacetime Military Service.

Preamble

Du Vries in his text Surgery of the Foot states " between 40% and 50% of the civilized society has, or will have, some foot disorder".

Anatomical and Physiological Factors

The foot serves two separate functions:

(a) It transmits the entire body weight to the ground.

(b) It provides the propulsion force when walking or running.

On weight bearing there is normally some depression of the longitudinal arch which increases the weight bearing area. During propulsion, the weight and force is transmitted from a small area behind the toes. For efficient foot function, the foot should flatten somewhat on standing and arch when pushing off during locomotion. The function of the longitudinal arch is to provide a resilient spring for weight bearing and forward propulsion in walking. A "fixed" arch cannot repeatedly perform this function without a strain.

There are no medical guidelines as to what is to be considered a "normal arch". Orthopaedists agree that the recorded height of the arch is dependent both upon the observer and his concept of what is a "normal arch", and on the stance of the person when he/she is examined. In the "at ease" position, the arch is much flatter than when standing with the toes pointed forward.

If in the opinion of the observer, the longitudinal arch is flatter or lower than his interpretation of the "normal arch," then the descriptive term flat feet or pes planus is applied. This term has been frequently used in the absence of symptoms based only on the appearance of the foot both by laity and the profession. The term is used indiscriminately, particularly by non-specialists, to indicate flattening of the transverse metatarsal arches as well as of the longitudinal arches and does not identify whether one or both arches are affected by flattening. Flattening of one of these arches does not imply that there is flattening of the other.

When examination of the foot reveals that the arch is higher than the observer's concept of a "normal arch", then the term pes cavus may be applied. Feet with exceptionally high longitudinal arches are often accompanied by "clawing" of the toes.

A) PES PLANUS (Flat feet, Valgus Foot)

The term pes planus is a relative term used, when in the observer's opinion, the person's longitudinal arch is somewhat lower when compared to the norm. The longitudinal arch is reduced so that on standing, the medial border is close to, or in contact with the ground. It is usually associated with some degree of pronation or twisting outward of the foot (valgus deformity or eversion).

A) PES PLANUS (Flat feet, Valgus Foot)...CONTINUED

Trauma resulting in muscle weakness or paralysis from nerve or muscle injury may cause flat feet. Most cases, however, are idiopathic and due to a hyper mobile foot. In such feet, the supporting ligaments are somewhat lax and unable to maintain the normal curvatures of the foot with weight bearing.

Although flat feet may be evident by the time growth is completed, such may not necessarily be the case. Flat feet may or may not result in symptomatology. Indeed most people with flat feet are asymptomatic but the flattening does predispose people with flat feet to foot strain when compared to normal.

In feet previously normal, foot strain is caused by excessive standing by a person unaccustomed to it. In feet whose intrinsic structure is already impaired (e.g. flat feet), chronic or recurrent strain often arises from the ordinary amount of standing demanded in everyday life.

Pension Considerations:

1) Pes planus may be pre-enlistment, develop during service or develop in post discharge period.

2) Increasing complaints during service not relieved when placed in a protective category would indicate a degree of worsening or aggravation.

3) Flat feet becoming increasingly symptomatic during service and remaining so post discharge would indicate a degree of permanent worsening.

4) Development of callosities, plantar fascitis or calcaneal spurs would indicate a degree of permanent worsening.

5) Calcaneal spurs, osteoarthritis tarsal-metatarsal joints, metatarsalgia and plantar fasciitis are considered in the assessment for flat feet.

6) Osteoarthritis at metatarsal phalangeal joint, hallux valgus and hallux rigidus should be ruled on as partially consequential to flat feet.

7) Normally flat feet are assessed nil to 15%, extraordinarily severe cases 15-20% range. This assessment refers to bilateral flat feet.

B) PES CAVUS (Claw Foot, Hollow Foot)

Pes cavus is characterized by exaggeration of the height of the longitudinal arch, slight shortening of the foot, prominence of the metatarsal heads in the sole, associated clawing of the toes and loss of flexibility of all joints of the foot.

In most cases, the deformity has a congenital basis. It is sometimes familial. In other cases, there is an underlying neurological disorder causing muscle imbalance. For instance, it may follow poliomyelitis.

The degree of deformity is variable and the foot tends to tire easily and develop callosities under the flattened transverse arch and on any or all of the toes. The deformity often becomes evident in childhood. It may affect one foot or both feet. In some cases, the symptoms are negligible. When symptoms do arise, they may result from 1) painful callosities beneath the metatarsal heads, 2) tenderness over deformed toes from pressure against the shoe, 3) osteoarthritis of the tarsal joints.

Pension Considerations:

1) With the rare exception, pes cavus is a developmental condition, evident by the time growth is completed and pre-enlistment in origin.

2) May give rise to metatarsalgia that is included in the assessment for pes cavus.

3) Claw toes, callosities are included in assessment for pes cavus.

4) Development of metatarsalgia, corns or callosities in a previously asymptomatic pes cavus would indicate a degree of aggravation.

5) Symptoms that disappear when placed in a protective category would not indicate any permanent aggravation.

6) Assessment for pes cavus is generally from nil to 15%. In exceptional cases, 15-20%. Again this assessment refers to a bilateral pes cavus condition.

C) METATARSALGIA (Pain in the Forefoot)

Metatarsalgia is not a diagnosis, but a term that refers to the symptom of pain in the forefoot, i.e. pain and discomfort in the region of the transverse arch. There are four main causes: 1) anterior flat foot (dropped transverse arch) as may be seen in pes planus or pes cavus, 2) stress fracture of a metatarsal bone (march fracture), 3) plantar digital neuritis (Morton's metatarsalgia), 4) Freiberg's Disease.

C) METATARSALGIA (Pain in the Forefoot)...CONTINUED

Pension Considerations:

1) Metatarsalgia should not be ruled on separately. Where possible, the primary condition should be sought and the metatarsalgia included in the entitlement or assessment.

2) If metatarsalgia is ruled on separately, the assessment should range from 0-5% each foot. In extreme cases, an assessment of 10% per foot may be justified.

Anterior Flat Foot (dropped transverse arch)

This is perhaps the most common cause of metatarsalgia. It is due to a permanent flattening of the transverse arch of the foot. The primary cause in most cases, would seem to be an inherent inefficiency of the intrinsic muscle of the foot. An anterior flat foot may be associated with either pes planus or pes cavus and should be included in the assessment for such: A separate ruling is not indicated. The arch may also be flattened by direct trauma, with stretching or disruption of the supporting soft tissues.

HALLUX VALGUS

Hallux valgus refers to a lateral deviation of the great toe at the metatarsal phalangeal joint. It is present in 5% of persons in early childhood and by the age of 15 years, 15% of males and 54% of females have hallux valgus without any evidence of disability. As a corollary, bunion is much more common in middle-aged females.

There are two recognized causes of primary hallux valgus: genetic or congenital factors, and the wearing of tight stockings or shoes with narrow pointed toes. High heels favour the development of hallux valgus because the forefoot is forced into the pointed part of the shoe. Army boots are broad in the forefoot and do not cause lateral deviation of the toes and therefore do not contribute to the development of hallux valgus.

Hallux valgus may also develop secondarily to: neuro-muscular diseases of the foot, e.g. polio; trauma; degenerative changes related to ageing, affecting the first metatarsal-phalangeal joint; or following amputation of the second toe.

Pension Considerations:

1) Hallux valgus may be a pre-enlistment condition or may develop during the service period or post discharge.

2) Pes planus may contribute to the development of hallux valgus and therefore hallux valgus may be considered consequential to pes planus to a moderate degree.

3) Service footwear is broad in the forefoot and therefore not considered to contribute to a hallux valgus condition.

4) Hallux valgus may accelerate or initiate the development of a hammer toe of the 2nd toe and such may be considered mild to moderately consequential to hallux valgus.

FRACTURE

Pension Considerations:

1) In general, healed fractures of themselves do not represent a disability. If solidly united, good alignment and no evidence of osteomyelitis or other problems at the fracture site, then assessment would be nil.

2) Fractures into a joint surface are considered to eventually cause degenerative changes within that joint and therefore such degenerative changes may be included in the assessment.

3) Degenerative changes in a joint near a fracture, although not necessarily involved by the fracture, may be considered in the assessment of a fracture. One must consider the time interval from the time of the fracture and development of degenerative changes, the extent of degenerative changes when compared to the corresponding joint in the other limb and the extent of degenerative changes when compared to the accepted normal for age.

I. PEPTIC ULCER DISEASE

Peptic ulcer is a benign, non-specific ulcer which occurs only in those portions of the digestive tract exposed to the action of acid gastric juice. In order of decreasing frequency, ulcer occurs in the duodenum, stomach, post-operative stoma, lower part of the esophagus, and rarely in Meckel's diverticulum. For practical purposes, the term peptic ulcer applies to a benign ulcer of the stomach and/or duodenum. The ulcer begins in the mucosa and usually invades the deeper tissues of the gastric and/or duodenal wall. Ulcers may heal completely but in an established chronic ulcer there is a continual struggle between the process of ulceration and healing.

Ulcers may be single or multiple. Different pathological and physiological factors are probably involved in the development of gastric and duodenal ulcer. If the specific location of the ulcer has been identified, then the ulcer is labelled duodenal or gastric. Where there are conflicting reports as to site, the term peptic ulcer is used.

Peptic ulcer is extremely common and it is estimated that about 12% of all persons at some time suffer from either chronic gastric or duodenal ulcer.

For pension purposes, entitlement for duodenal ulcer, gastric ulcer or peptic ulcer indicates entitlement for any benign ulcer of the stomach and or duodenum.

CLINICAL MANIFESTATIONS

There are three classical symptoms associated with peptic ulcer disease:

1. Epigastric pain, usually burning or gnawing in character, occurring between meals.

2. Relief of this discomfort by either food or alkali.

3. Periodicity of symptoms with the remission of these symptoms for weeks or months.

Not all cases display the classical symptoms but nearly all ulcer patients have periods of remission of discomfort.

The initial clinical evidence of ulcer may arise from complications of the disease without any prodromal symptoms. Perforation or bleeding (as manifested by vomiting of blood or melena) may be the first indications of the peptic ulcer.

Symptoms such as nausea, discomfort immediately after food, vomiting, loss of appetite, gas, abdominal cramps or diarrhea unaccompanied by any of the classical features are not suggestive of an ulcer diathesis.

DEFINITIVE DIAGNOSIS

Under ideal conditions, probably 85% to 95% of ulcers can be demonstrated by x-ray. The absence of a positive x-ray diagnosis does not exclude the condition but, with repeated x-ray examinations, a positive diagnosis can usually be established when classical clinical symptoms are described. The earliest x-ray finding may simply be

deformity of the duodenal cap, without actual demonstration of an ulcer crater. With classical symptoms and in the absence of other G.I. pathology, this reflex spasm is probably due to duodenal ulcer, providing the diagnosis is confirmed at a later date. Diagnosis may be established or confirmed by gastroscopic examination.

ETIOLOGY

The accepted medical opinion is that duodenal ulcer is a constitutional disease which is commoner in people with GROUP O blood type.

It is a well known fact that gastric secretion may be increased by emotional factors but there is no evidence to conclude emotional factors can cause peptic ulcer. There has been no certain demonstration that actual ulcer formation is indeed proceeded by a significant increase in gastric secretion. Most patients with peptic ulcer have excess gastric acidity and increased night volume of acid. These findings may also be present in people who do not develop the condition.

Much work has been done on the mucosal barrier, the parietal cell mass, the effect of gastrin on gastric secretion, but despite this no specific cause for peptic ulcer has been identified. Peptic ulcer occurs when there is a breakdown of the mechanism which protects the mucosa.

PENSION CONSIDERATIONS

A. Pre-Enlistment

Rarely is there a positive clinical diagnosis of ulcer recorded on the enlistment documents. There may be a "yes" entered opposite "gastrointestinal" but only occasionally do the qualifying remarks suggest peptic ulcer. Review of the first history subsequent to enlistment, supported by other histories recorded during service, should establish beyond reasonable medical doubt whether the condition is pre-enlistment or incurred during service.

There are three common situations which establish whether the peptic ulcer disease is pre-enlistment beyond reasonable medical doubt:

1) Enlistment entry referring to previous gastrointestinal disturbance. The service history confirms the fact that the pre-enlistment symptoms are essentially identical to those which are complained of during service. The diagnosis is supported by a positive or suggestive x-ray report, (duodenal spasm without a demonstrable ulcer niche).

A. Pre-Enlistment ...CONTINUED

2) No enlistment entry of previous gastrointestinal disturbance. Chronic peptic ulcer disease is characterized by periods of remission and activity. The first history following enlistment and subsequent medical histories support the conclusion that the symptoms, first recorded during service, are identical to those stated in the histories to have been present prior to enlistment. If these symptoms are compatible with peptic ulcer then the condition can be considered pre-enlistment beyond reasonable medical doubt if the clinical findings are confirmed by positive or suggestive x-ray findings during service.

PENSION CONSIDERATIONS... CONTINUED

3) Clinical situations which correspond to 1 or 2 but the positive diagnosis is not established until post-discharge. In these circumstances the whole clinical picture must be evaluated. The repeated history of pre-enlistment distress is as important as the service symptoms and if neither can be explained on any other basis, the condition diagnosed post-discharge is pre-enlistment beyond reasonable medical doubt.

B. **Incurred During Service**

Classical symptoms which arise during service, along with some supporting evidence of continuity, indicate the onset of the condition although confirmation may not be established until some years later. An enlistment record of some gastro-intestinal upset pre-enlistment is not considered to be due to ulcer unless the service histories confirm that the pre-enlistment symptoms are indeed due to ulcer disease.

C. **Generals**

1) X-ray or Gastroscopic Findings

If scarring or deformity are evident on either of these examinations it is considered that the peptic ulcer disease has been present for at least 3-4 years.

2) Assessments of Peptic Ulcer Disease

Foremost in determining assessment of peptic ulcer disease are symptomatology and long term "clinical picture of the disease process". Such factors as frequency of exacerbation, time loss from work, medication, diet and compliance are all considered in the assessment of peptic ulcer disease: bleed (hemorrhage), perforation, dumping syndrome, bile reflux, anemia, and post vagotomy diarrhea. A discussion on Dumping Syndrome and bile (alkaline) gastritis appears later in the Guidelines.

Post gastrectomy syndrome should not be accepted as a diagnosis and ruled on separately. It may refer to any one or a combination of complications previously mentioned (dumping syndrome, bile reflux, post vagotomy diarrhea) and already included in the assessment.

Post-operatively, several months may be required for development of optimal function. Thus early complications should not considered to constitute long term disability until at least 6 months have elapsed. When appropriate these complications are reflected in the assessment for duodenal ulcer, by increasing the latter above the minimum 15% range. This 15% minimum for post operative ulcer patients is considered to include the average complaints and expected problems of disturbed physiological function in these patients. The so-called "small stomach syndrome" where the patient is unable to eat a large meal because of fullness and/or bloating with gastrointestinal discomfort is considered to be included in the 15% assessment. Generally such symptoms can be managed by dietary

C. **Generals**...CONTINUED

manipulation with smaller and more frequent meals. Complaints seemingly due to the "small stomach syndrome" do not warrant an increase in the 15% minimum post operative assessments.

However, dysfunctions above the norm that are considered due to genuine post operative complications (e.g. Dumping, Bile Gastritis, Post Vagotomy Diarrhea) may warrant an increase in the minimal assessment. A maximum total assessment of 35% for duodenal ulcer should, however, rarely if ever be exceeded.

Separate rulings should be arranged for:

i) Intestinal obstructions, stomal obstruction.
ii) Carcinoma of stomach.
iii) Hiatus hernia, Petersen's hernia.

These conditions should not routinely be considered in the assessment for peptic ulcer disease.

STRESS, PERSONALITY AND NEUROTIC DISORDERS

The ulcer diathesis is essentially a constitutional disease with or without a specific family history. Stressors may temporarily increase the flow of gastric juice but rarely can stress be considered an aggravation factor. In rare cases, service records may reveal an identifiable specific stressor which could cause minor aggravation. Duodenal ulcer may occur in people who have a chronic anxiety state but there is convincing proof that the incidence is no higher in these people than in the general population and there is suggestive evidence the incidence may be lower.

The above statement is expressed very clearly by Kessel and Munro in their article on "Epidemiological Studies in Psychosomatic Medicine", in the Journal of Psychosomatic Research - "but it has not been satisfactorily demonstrated that personality factors, environmental 'stresses', or psychological illnesses are etiologically associated with peptic ulceration".

MEDICATIONS

Many claims are submitted for ulcers on the basis that the peptic ulcer disease has developed as a result of medications taken for other pensioned conditions.

Certainly many drugs are known to be gastric irritants. The list of medications listing gastrointestinal side effects is increasing all the time. Among the most commonly mentioned are aspirins and other anti-inflammatories (Non-Steroidal Anti-Inflammatory Drugs, Phenylbutazones, Indocid derivatives) and corticosteroids. Not everybody placed on these medications develops peptic ulcer disease and it is felt that there is a certain group of individuals predisposed to develop peptic ulcer disease. When such an individual is placed on one of these medications on a regular basis it is felt that such may contribute to the development of and/or aggravation of the peptic ulcer condition to a mild to moderate degree. In determining the degree of aggravation one must consider the

amount and frequency of medications taken and whether these medications are taken entirely for a pensioned condition or for both pensioned and non-pensioned conditions.

There are occasional claims submitted on the premise that the claimed conditions are consequential to peptic ulcer disease when because of the peptic ulcer disease these individuals are unable to take medications for the claimed condition. An example would be a person with a duodenal ulcer who is unable to take arthritic medication for his osteoarthritis because such medication irritates his ulcer, but by not taking these medications his arthritis is more symptomatic and difficult to control. In such instances, it is recognized that the peptic ulcer disease influences symptomatology of the claimed condition and a minimal to moderate consequential relationship is considered.

II. DUMPING SYNDROME

This is a relatively rare complication of gastrectomy (total or partial) for peptic ulcer disease. The term tends to be over used in referring to multiple vague complaints which may occur after ulcer surgery; in fact, dumping syndrome has two forms, early and late, with very specific clinical manifestations.

Early dumping syndrome, the commoner form and usually called simply dumping syndrome, is characterized by epigastric fullness, cramps, nausea, palpitation, extreme weakness, hot sensations and cold sweats occurring immediately after eating. The exact mechanism is uncertain, but is probably related to the too-rapid emptying of partly-digested or undigested food into the small bowel. The symptoms usually respond to dietary manipulations and are rarely disabling.

The late dumping syndrome is very rare and consists of a rapid rise in blood sugar after eating, followed by an equally precipitous fall in blood sugar which results in symptoms of hypoglycemia, i.e. dizziness, fainting, sweating and mental confusion. These symptoms occur 1½ to 3 hours after eating. This syndrome is also readily controlled by dietary manipulation and is rarely disabling.

Additional assessment for dumping syndrome, above and beyond the assessment for gastrectomy, should be reserved for those who present definite and persistent evidence of dumping syndrome (early or late) not readily corrected by dietary manipulation. Assessment should then be in the 5-10% range and the assessment for duodenal ulcer increased accordingly.

III. BILE GASTRITIS

This a complication of gastrectomy for peptic ulcer surgery whereby the reflux of bile from small intestine into the stomach remnant results in nausea, bilious vomiting and epigastric pain.

The mere presence of bile in the gastric remnant is almost universal and, in and of itself, is not evidence of bile gastritis. In order for the diagnosis to be acceptable, there must be objective evidence of bile reflux and of gastric inflammation, proven by endoscopy,along with significant symptoms.

III. **BILE GASTRITIS**...CONTINUED

Bile gastritis is usually treated medically, but with limited success. Rarely, surgical treatment is required.

Once the diagnosis is well established, the assessment of bile gastritis would be of the order of 5-10% and the assessment for duodenal ulcer increased accordingly. However, the importance of accurate diagnosis cannot be overemphasized, since many patients present post-gastrectomy symptoms which are vague, transient, and already included in the basic assessment.

IV. **HIATUS HERNIA**

A hiatus hernia is basically a protrusion of abdominal contents (usually stomach) through the esophageal hiatus of the diaphragm and into the thoracic cavity.

A hiatus-hernia can be demonstrated in nearly 50% of adults after the age of 50 years. In most individuals it is asymptomatic but may give rise to symptoms because of esophageal reflux of gastric contents or because of its large size, by interfering with cardio-pulmonary function. Rarely a hiatus hernia may develop following extensive trauma to the diaphragm.

There is no direct relationship between peptic ulcer disease and hiatus hernia. However, in the past it has been accepted, following subtotal gastrectomy of the Billroth II type, that since the pancreato-gastric ligament has been interfered with, the gastric remnant would be more subject to herniation through the diaphragm. Unfortunately, there are no sound medical facts to substantiate this hypothesis. It is an attractive theory but standard medical literature fails to substantiate any relationship.

Today, the commonest surgical procedure with regard to duodenal ulcer is vagotomy (of whatever type) and pyloroplasty. This procedure may be combined with antrectomy or partial gastrectomy but the essential feature is that the esophageal hiatus has to be dissected free in order to expose the vagus nerves. The interference with the esophageal hiatus may well play a part in the development of hiatus hernia. How great a part will depend on the time interval following surgery and the development of the hernia. The patient's age must also be considered. The latter fact is important because the increased incidence of hiatus hernia is considered consequential to a moderate degree to these surgical procedures.

A hiatus hernia without reflux esophagitis should in general be assessed at nil. Assessment of esophageal reflux is considered in the next section.

V. **ESOPHAGEAL REFLUX (REFLUX ESOPHAGITIS, PEPTIC ESOPHAGITIS)**

Reflux esophagitis results from regurgitation of gastric contents into the esophagus. The pathophysiology includes a permanently or intermittently incompetent lower esophageal sphincter. Severity depends on frequency and duration of reflux and the ability of the esophagus to generate peristaltic waves that would normally prevent prolonged contact of the mucosa with acid and pepsin. A hiatus hernia may or may not be present. The presence of a hiatus hernia is of no consequence unless it is associated with reflux (an

V. ESOPHAGEAL REFLUX (REFLUX ESOPHAGITIS, PEPTIC ESOPHAGITIS) ...CONT'D

exception is the paraesophageal type which has a tendency to incarcerate, ulcerate and develop other complications).

The following symptoms are considered suggestive of reflux esophagitis:

° Substernal burning, cramping, severe pain or pressure.
° Symptoms aggravated by recumbency or increase of abdominal pressure and relieved by upright position.
° Nocturnal regurgitation, cough, dyspnea, possible aspiration.

Symptoms are the result of reflux of acid or alkaline gastric contents into the esophagus because of an incompetent lower esophageal sphincter. The conditions associated with an incompetent esophageal sphincter are hiatus hernia, short esophagus, pregnancy, obesity, pernicious vomiting and nasogastric tubes.

Complications of reflux esophagitis include dysphagia, odynophagia, erosions, stricture, ulceration and anemia (iron deficiency from occult bleeding). Aspiration may cause cough, dyspnea or pneumonitis.

As mentioned in hiatus hernia, interference with the esophageal hiatus during surgical procedures such as vagotomy may predispose to development of hiatus hernia and an incompetent lower esophageal sphincter. The latter would predispose to the development of reflux esophagitis. The time interval between operation and development of esophageal reflux is an important point in determining the degree of consequentiality. Usually a moderate degree of consequentiality is considered.

Gastroesophageal reflux is generally assessed between nil and 10%. Complications as mentioned above would be included in the assessment with a general assessment from 5% to 10% for esophageal erosions, 10% to 20% esophageal ulcerations and 15% to 30% for stricture formation.

VI. PANCREATITIS

Acute pancreatitis is a severe abdominal disease produced by acute inflammation in the pancreas and associated release of pancreatic enzymes into surrounding tissues. The pathogenesis is not known although a large percentage of cases occur with biliary tract disease and/or alcoholism. Trauma may play a role and vascular or allergic causes have been postulated. Heredity may play a role particularly in chronic pancreatitis where there seems to be an association with hyperlipoproteinemia; hyperparathyroidism and hypercalcemia have also been implicated. Pancreatitis may also occur in association with certain drug therapies including prednisone or thiazide diuretics.

As a general rule, there is no relationship between peptic ulcer and pancreatitis. There is no medical evidence to suggest that idiopathic pancreatitis is more common in post surgical patients than in the general population. There are, however, three circumstances in which there is a relationship between peptic ulcer disease and pancreatitis.

1. A penetrating duodenal ulcer may invade the pancreas and cause inflammation. This inflammation is localized and self-limiting and unlikely to lead to chronic pancreatitis.

2. During surgery for duodenal ulcer, the pancreas may be interfered with and in the immediate post-operative period an acute self-limiting pancreatitis is not unusual.

3. There is only one late complication of surgery which may be implicated in the development of pancreatitis. It is believed that pancreatitis probably arises on a reflux basis and in those cases where there is demonstrable evidence of an afferent loop syndrome then conceivably late development of chronic pancreatitis may well be related to the surgery for peptic ulcer.

VII. CHOLELITHIASIS

Chronic cholecystitis with cholelithiasis is a very common condition. There is no direct relationship between peptic ulcer and gallbladder disease. Vagotomy and pyloroplasty is a common surgical procedure for duodenal ulcer. It is a well recognized medical fact that following truncal vagotomy there is dilatation of the gallbladder with delayed emptying. Statistical reports are conflicting but there is a sufficient element of doubt to lead one to the conclusion that the physiological changes which occur in the gallbladder following truncal vagotomy may in a minor way contribute to the formation of gallstones.

VIII. CARCINOMA OF THE STOMACH

Gastric carcinoma may appear in association with, but independent of gastric or duodenal ulcer. There is no evidence to suggest that there is an increased incidence of gastric carcinoma in people with peptic ulcer.

In considering the possible relationship between carcinoma and peptic ulcer, it is essential to know whether the benign ulcer was duodenal or gastric, or if surgery had been carried out for the benign ulcer.

1. Gastric Ulcer

Today practically all authorities agree that malignant gastric ulcers are malignant from the start. It is not proper to use the term "never", but a benign gastric ulcer rarely, if ever, undergoes malignant change. If a gastric carcinoma arises at the site of a persisting benign gastric ulcer, the possibility of a relationship cannot be denied absolutely, in the present state of medical knowledge.

2. Duodenal Ulcer

Duodenal ulcer never undergoes malignant change.

3. Surgery for Peptic Ulcer

The cause of gastric carcinoma is unknown but a statistical report from Oslo indicates there may well be a greater incidence of carcinoma of the stomach in patients who have had previous surgery, either Billroth II gastrectomy or

gastroenterostomy, for benign gastric conditions. This increased incidence only occurs if the surgery was carried out twenty or more years prior to the onset of the carcinoma. Statistically the incidence of gastric carcinoma is lower than average in the first fifteen years post-surgery. These facts have not been confirmed or denied by other investigations.

Multiple factors are involved in the development of any carcinoma. On the basis of the current statistical evidence there appears to be a moderate consequential relationship between the gastric surgery and the gastric carcinoma which develops twenty or more years thereafter.

Headache is one of the most common afflictions of mankind; it happens to virtually everyone at some point in life; it is considered by most as nothing more than an occasional annoyance, and does not represent a disability; in a small minority of individuals, the severity and/or frequency of headaches is such as to interfere with the ability to function, and may constitute a disability, albeit a relatively minor one, when compared to that which occurs from other neurological disorders.

Obviously, headache is a symptom and not a diagnosis; it is only when the characteristics of the headache, such as its location, time of occurrence, frequency, duration, associated symptoms, qualities (e.g. "throbbing",etc.), and relieving or worsening factors are carefully analyzed that one may arrive at a clinical diagnosis.

Unfortunately, headache may not be classified as neatly as some other medical disorders; some individuals suffer from headache of multiple causes, which by force, may be referred to as "mixed-tension-vascular headache". In some cases, headache persists where there is no cause identified nor mechanism invoked, and one is reduced to using the term "chronic headache".

The commonest types of headache encountered will be briefly described:

1. **Tension headaches**: This is the so-called "ordinary" headache which may affect anyone, especially when one is harassed or fatigued. It is usually a diffuse head pain often described as a sensation of tightness, and is due to sustained tension of the scalp and neck muscles; it may be triggered by psychological factors. Tension headache usually lasts up to a few hours, is not severe, and is relieved by anxiolytics or simple analgesics.

2. **Migraine headache**: This is a type of vascular headache produced by dilation and increased pulsation of the arteries of the scalp and face. The headache is often severe, at times preceded by an aura or prodrome, i.e., mood disturbances, lethargy, or visual phenomena; it may be unilateral or bilateral, and is characterized by its "throbbing" quality, often with accompanying nausea, vomiting and photophobia.

   Rarely, migraine may be associated with transient neurological phenomena such as hemiplegia or ophthalmoplegia.

3. **Cluster headache:** This is a type of vascular headache which derives its name from the fact that the headaches cluster in time, i.e., one or more headaches will occur every day for several weeks or a few months at a time, and then the headaches stop and the patient will remain free of headache for months or years, until the next "cluster".

   The condition is much commoner in men than in women; the pain begins rather suddenly and rapidly increases in intensity; it is extremely severe and centers around one eye and into the cheek, temple and forehead; it is boring or piercing rather than throbbing, and is accompanied by reddening of the eye, drooping of the eyelid, and nasal congestion. The headache lasts for up to an hour.

4. **Post-traumatic headache:** This type of headache follows head trauma, beginning immediately or a few hours after the event, lasting several days, and gradually decreases over the ensuing weeks or months. It is localized to the site of the original injury.

5. **Mixed (tension-vascular) headache**: This term is used when the type and pattern of headache incorporates components of tension and migraine headaches, and no clear distinction can be established.

6. **"Chronic headache"**: This refers to headache with no known organic cause which occurs almost every day, or all day every day, which does not correspond to the usual headache patterns, which is unresponsive to treatment and is associated with psychological disturbances. This headache is usually present upon first awakening in the morning, persists throughout the day, may be described in a variety of ways, and has no particular location. It is frequently accompanied by other functional symptoms, and concomitant psychological factors are the norm.

ASSESSMENT OF HEADACHE

In most instances, headache is more an annoyance than a true disability; this would pertain, for example, in most cases of tension headache.

In some cases, when headache actually interferes with ability to function, some minimal degree of disability is considered to exist, usually of the order of less than 5%. Exceptionally, the assessment would be 5% or more in cases of complicated migraine, i.e. where a continuing deficit has occurred.

In dealing with headache and other episodic neurological disorders such as epilepsy, one usually does not have a fixed deficit to assess; rather, the assessment is based on the overall impact which the disorder has on the individual's activities of daily living.

There are 3 classes of hearing loss:

1. Sensorineural (formerly called perceptive);

2. Conductive;

3. Mixed -a combination of the first two.

Anatomy of Hearing Loss

Sounds are collected by the external ear (auricle) and transmitted down the external ear canal to set the eardrum in motion. The eardrum separates the canal from the middle ear with its 3 ossicle - malleus (hammer), incus (anvil), and stapes (stirrup). The eardrum's vibrations are picked up and amplified by the ossicle and conducted to the cochlea (organ of hearing). The whole system, from the auricle to the stapes, is the conducting apparatus of the ear. Any abnormality in the system, from wax in the canal to fixation of the stapes by otosclerosis, can cause a conductive hearing loss.

The ossicle's vibrations are transmitted indirectly to the fluid in the cochlea. Movement of the fluid stimulates the cochlea's hair cells. They transmit electrical impulses along the auditory nerve (of hearing) to the brain. The whole system, from the cochlea to the auditory cortex in the brain, is the sensorineural apparatus. Any abnormality in the system - cochlear damage, acoustic neuroma - can cause a sensorineural hearing loss.

Types of Hearing Loss

Hearing loss is a symptom, not a diagnosis. In any claim for hearing loss an attempt has to be made to determine the cause of the problem.

As a general rule, sensorineural hearing loss affects mainly the high tones, conductive hearing loss the low. The rule is not invariable. Early Meniere's disease classically causes a low tone sensorineural hearing loss; otosclerosis causes a conductive hearing loss that may affect the high tones as well as the low.

Audiometry gives the answer. A complete audiogram shows hearing by both air and bone conduction. In a conductive hearing loss, hearing by bone conduction is normal, hearing by air conduction is diminished. The air conduction graph is therefore at a lower level in the audiogram than the bone conduction graph. The gap between the two graphs is known as the "air-bone gap".

In a sensorineural hearing loss, air and bone conduction are affected equally; the two graphs are at approximately the same level; there is no air-bone gap.

In a mixed hearing loss, air and bone conduction are both affected, but the loss by air is more severe than the loss by bone. The air conduction graph is lower than the bone conduction graph. There is an air-bone gap.

Sensorineural Hearing Loss

Of chief concern are hearing loss due to age (presbycusis) and hearing loss caused by noise (noise induced hearing loss).

Age, not noise, is the commonest cause of sensorineural hearing loss and probably of all hearing loss. Both initially cause a high tone hearing loss which gradually spreads to other frequencies, and it is sometimes difficult to tell whether age or noise is to blame. Actuarial tables and graphs show the average hearing levels at different ages for people who have not been exposed to excessive noise, and it is possible to say with some certainty how much of a particular hearing loss is due to age, and how much to other causes - noise, for example.

To cause a hearing loss, the noise has to be loud enough and act for a sufficiently long time. The louder the noise, the shorter the time in which it will damage hearing. Characteristically, the first sign appears as a dip or notch at one of the higher frequencies, usually 4000 or 6000 Hz. This may be temporary (temporary threshold shift), with complete recovery of the hearing in a few hours to a few weeks. With continued exposure, the hearing loss becomes permanent because of irreparable damage to the cochlea's hair cells, As exposure continues, more hair cells are damaged, and the hearing loss spreads to adjacent frequencies (2000, 1000 Hz). Once exposure to the noise ceases, no further loss of hair cells and therefore no further loss of hearing occurs. Any further deterioration means that other causes of hearing loss are in action - age or other noise for example.

Above a certain intensity, noise acts in a different way. It becomes explosive and causes blast injuries. It may rupture the eardrum, causing a conductive hearing loss. If no further damage has been done, the hearing loss may be temporary: if the eardrum heals, complete restoration to normal is possible. The blast may damage or dislocate the ossicles of the middle ear, causing a conductive hearing loss that may be permanent unless the ear is successfully operated upon. The blast may tear the sensitive part of the cochlea (the organ of Corti) from its moorings, causing a sensorineural hearing loss that is permanent and irreparable. Any combination of these injuries may occur, so that the hearing loss may be conductive, sensorineural, or mixed.

Obviously, military service is not the only source of noise. Modern society is exposed to noise from many sources: jet aircraft, drilling and blasting in mines, tractors and other agricultural machinery, air conditioning, music, traffic, chainsaws, snowmobiles - the list is long. Hunting and shooting are known to be causes of noise induced hearing loss. All these possibilities have to be taken into consideration in evaluating hearing loss in Regular Force personnel. (No problem arises with Active Force personnel: a permanent hearing loss is pensionable if it begins in Active Force Service.)

Age and noise are only two among many causes of sensorineural hearing loss. Severe head injuries and skull fractures are known to be causes. Barotrauma, caused by pressure changes in the middle ear as in flying or deep-sea diving, is a rare cause: more usually it causes a temporary conductive hearing loss.

Sensorineural Hearing Loss...continued

Ototoxic drugs are a cause. The best known are the amino-glycoside antibiotics. One of them, streptomycin, more commonly affects the vestibular apparatus of the inner ear, with vertigo as a result: another dihydrostreptomycin, was a notorious cause of deafness, and for this reason is no longer used. Quinine and the salicylates (aspirin) can cause a hearing loss that is usually temporary, with restoration to normal when the drug is stopped.

Meniere's disease and acoustic neuroma (tumor of the auditory nerve) both cause a triad of symptoms: sensorineural hearing loss, tinnitus, and vertigo. Any of the three may be the first symptom. A battery of tests is required to make the diagnosis, and without them it cannot be considered that the diagnosis has been established. They include standard pure tone audiometry, site-of-lesion audiometric investigations, brainstem evoked response audiometry, electronystagmography, x-rays, and CAT scan. Neither of these conditions are caused by noise or environmental factors, and no relationship exists with any circumstance of Regular Force service.

Psychogenic deafness may be conscious (feigned) or unconscious. The diagnosis can usually be confirmed by sophisticated audiometric testing including brainstem evoked response audiometry.

Virus, notably the mumps virus, can cause a sensorineural hearing loss. It may be unilateral or bilateral.

Conductive Hearing Loss

Blast injury and barotrauma have already been mentioned as possible causes of conductive hearing loss. Among the more common causes are otitis externa, otitis media, and otosclerosis.

Otitis externa is an inflammation of the skin of the external ear canal. Swelling, or collection of discharge in the canal, or both may cause a conductive hearing loss. It is temporary - with one exception. A form of malignant otitis externa occurs in elderly diabetics. It is resistant to treatment, and could cause a permanent conductive hearing loss.

Otitis media is inflammation of the middle ear caused by infection, and may be acute or chronic.

In acute otitis media, the main complaint is pain. Pus accumulates in the middle ear (hence the name acute suppurative otitis media) and bulges the eardrum. The eardrum may rupture, pus will be discharged, and the pain will stop at once. A conductive hearing loss is present from the onset. With proper treatment the infection clears, the perforated eardrum usually heals leaving a small scar, and the hearing returns completely to normal.

With or without treatment, the perforation may persist. The hearing may or may not be normal, depending on the size and situation of the perforation. The ear is prone to recurrent infections and purulent discharge. The otitis media has become chronic. This type of chronic otitis media is seldom,if ever, a threat to life, and can often be treated successfully by surgery (tympanoplasty).

Sensorineural Hearing Loss...continued

A more dangerous type is chronic otitis media with cholesteatoma. This doesn't begin with acute otitis media, but is chronic for the start. (Some authorities believe it may occur as a result of repeated attacks of acute otitis media in childhood which were unrecognized, untreated, or inadequately treated. Some have even postulated attacks of acute otitis media in utero.)

Cholesteatoma is an ingrowth of squamous epithelium (skin) which expand like a tumor. If untreated, it may invade the mastoid bone, causing chronic mastoiditis; the facial canal, causing facial paralysis; and the labyrinth (inner ear) causing labyrinthitis, with vertigo and sensorineural hearing loss. These can lead to meningitis or brain abscess, and possible death. This is the type of chronic otitis media that was at one time treated by radical mastoidectomy. The usual treatment now is a form of tympanoplasty.

A non-suppurative form of otitis media has been called catarrhal otitis media, serous otitis media, otitis media with effusion, and other names. In this variety, the middle ear contains serous or mucoid fluid, and there is a conductive hearing loss that can be reversed by treatment. If untreated, the condition may progress to chronic adhesive otitis media - in effect, scarring of the middle ear - with a permanent conductive hearing loss.

A history of earache prior to enlistment or even a history of previous discharge from the ear is not evidence of previous otitis media. There are many causes of both symptoms. An earache may even be caused by a condition unconnected with the ear - referred pain from a tooth, for example, Real evidence of a pre-enlistment otitis media would be such facts as: a documented history; a conductive hearing loss detected at enlistment by the methods of testing then in use (conversation or whispered voice tests, audiometry); a scarred, fixed, or perforated eardrum (proof of previous middle ear disease); and the scar of a previous mastoidectomy.

Otitis media can be related to Regular Force service in only one uncommon circumstance: rupture of the eardrum from blast or injury, with subsequent infection of the middle ear.

Otosclerosis is a hereditary disease. An outgrowth of spongy bone near the oval window blocks movement of stapes, causing a conductive hearing loss. The hearing loss commonly first appears in the late teens or early twenties. With time, the process may spread to the inner ear, causing a mixed and ultimately a profound sensorineural hearing loss.

Otosclerosis diagnosed during Active Force service would be considered of pre-enlistment origin. The disease is not caused by noise, infection, or environmental factors, and could not be considered as related to any circumstance of Regular Force service.

Tinnitus

The word refers to a subjective symptom of noise in the ears. Most commonly the sufferer complains of a ringing in the ears, but others may complain of hissing, buzzing, whistling, or roaring. The last is particularly common in Meniere's disease.

Most people have at some time experienced tinnitus, and there is no doubt that it can be annoying or even distressing, but because it is subjective, its presence cannot be proved (or disproved) and its intensity cannot be measured. It may exist on its own, but frequently accompanies ear disease.

It may, for example, be present with noise induced hearing loss, in otosclerosis, and nearly always in Meniere's disease. It is said that 50 per cent of persons with presbycusis complain of tinnitus.

As with hearing loss, tinnitus is a symptom, not a diagnosis. A claim for tinnitus alone is not accepted, but if it occurs as part of a claim for hearing loss, for example,an attempt is made to determine the cause.

Because it cannot be measured, and because its existence cannot be proved, it cannot be assessed, and most jurisdictions - and maybe all - will not recognize tinnitus as a pensionable disability.

Definition

Hypertension is a condition in which recorded blood pressure readings consistently exceed the normal range. However, there is no rigid dividing line (see Sir George Pickering, The Fallacy of the Dividing Line, In LARAGH Hypertension).

Normal Blood Pressure

Blood pressures in the normal healthy individual vary over a wide range. The blood pressures of normo-tensive individuals have been monitored by exact scientific methods over twenty-four hour periods. Variations of blood pressures were noted from a high 220/90 under highly emotional conditions to a low of 90/40 during sleep. As can be seen from these figures there is a much greater range of variation of the systolic pressure (130 mm. of Hg.) than the diastolic (50 mm. of Hg.) It is well recognized that the blood pressure increases slightly as age advances. The New York Heart Association has defined hypertension - "Persistent arterial blood pressure above 140/90 mm. Hg. in persons below fifty and 150/100 mm. Hg. over the age of fifty indicates the presence of hypertension. Both diastolic and systolic hypertension are significant".

Repeated recordings are essential for a proper diagnosis of hypertension. A large scale study conducted in the UK found that on the basis of a single blood pressure reading 33% of the subjects had a measurement in the hypertensive range. However, given a series of six recordings, the proportion of true hypertensives dropped to 5%, i.e. showing persistent elevations.

Classification

Hypertension may be (1) Essential, and (2) Secondary.

1. Essential Hypertension is a primary hypertension with unknown cause. 95% of the cases of hypertension are of the essential type.

2. Secondary Hypertension occurs as an accepted manifestation of known diseases. The most important of these diseases are renal disease and certain endocrine diseases. It is, in effect, a symptom of the primary condition and would be included in the consideration of the primary condition.

Clinical Manifestations

The characteristic clinical finding in hypertension is an elevation of blood pressure above the normal range. There are usually no symptoms. On questioning, the symptoms that the individual may be aware of are early morning headache and consciousness of a forceful pulse. Frequently, the first manifestation of hypertension may be the occurrence of a vascular complication from arteriosclerosis, i.e. a "stroke" or "heart attack", at which time examination shows the presence of hypertension.

Once diagnosed, the course of hypertension is so variable that no predictions can be made. The extremes of progression of the hypertensive process can be as follows:

1. The level of blood pressure may persist at a low level of hypertension with little evidence of worsening or of complications for twenty to thirty years.

2. Those affected by accelerated (malignant) hypertension may show extremely rapid development of increasingly high levels of hypertension associated with severe

Clinical Manifestations ...CONTINUED

progressive renal damage and such individuals have been known to die in a matter of two to three months.

3. The progress of hypertension commonly is between these extremes.

Etiology

1. **Physiological Control of Blood Pressure**

This is complex and various physiological mechanisms have been suspect as contributing to Essential Hypertension. These are:

(i) Renin - Angiotensin Mechanism
(ii) Hormones - Specifically Aldosterone
(iii) Sympathetic Overactivity
(iv) Carotid Sinus and Aortic Body Baroceptors

In none of these mechanisms has evidence been found to support that they contribute to the development of Essential Hypertension.

2. **Heredity**

It has been shown that people whose parents are (or were) hypertensive are far more likely to develop hypertension.

3. **Age**

There is a slight increase normally in blood pressures with advancing age.

4. **Obesity**

This does appear to have some influence in the development of hypertension but only after the weight exceeds 20% of the normal average weight for height and age.

5. **Environmental**

(i) Diet, smoking, occupation and infection have not been found to influence the development or worsening of hypertension (excepting where diet has caused obesity as noted above).

(ii) Stress, nervousness and strain related to on-the-job pressures are experienced by all individuals, civilian and military. The reaction of each individual to stressors is a reflection of the individual underlying personality. The general consensus of medical thought is that there is insufficient evidence to conclude that such day to day stress contributes to the development of hypertension. There is no question that emotional factors will produce a temporary rise in blood pressure, both systolic and diastolic, but there is insufficient evidence to confirm that this produces any long-term elevation of the blood pressure. It has been shown clearly that in individuals suffering from psychiatric disorders, hypertension does not occur more frequently than in mentally healthy individuals.

Pension Considerations for Essential Hypertension

1. Individual Consideration of Claims for Hypertension

In the medical consideration of any claim for hypertension, it is clear that with all the variables present (some of which are noted below), each claim must be considered on an individual basis and the advice to Veterans Affairs Canada will be based on all the medical evidence which is available.

2. Definition of Hypertension for Pension Purposes

A systolic blood pressure of 140 and/or diastolic reading of 90 are widely considered as a minimal level for a diagnosis of hypertension. These figures cannot be considered abnormal if they are followed by many years of lower levels of blood pressure. In a similar fashion, these figures or even slightly lower levels cannot be considered normal if they are followed closely by recorded evidence of persistent and progressive elevation of the blood pressure, particularly in a young individual.

It is considered that diastolic readings of 100mm. Hg, or greater indicate hypertension. In recent years the elevation of the systolic reading has attained more significance than formerly and at higher levels is certainly more significant as related to the vascular complications such as strokes dissecting aneurysms.

The medical determination of the date of onset of Essential Hypertension will be determined by a consistent rise in blood pressure, possibly with post-discharge years. Systolic levels below 140 and/or diastolic levels below 90 will not usually be accepted as evidence of hypertension, unless there is significant progression to higher levels which are maintained over the subsequent five to ten year period.

3. a) Single Borderline Readings

A single borderline reading in the 140-90 range is difficult to evaluate and must be considered in the light of the applicant's age, weight, and the circumstances under which the reading was taken. Such single reading or even slightly higher systolic levels will not normally be considered as indicating the presence of hypertension unless followed by recorded evidence of persistent hypertension in the early post discharge years following service covered by the Insurance Principle.

A single borderline reading followed by a ten, twenty or thirty-year period without a recorded blood pressure reading, cannot be used to consider a later diagnosis of hypertension as service related. The cause of the 95% of hypertension is unknown. Therefore, there is no medical knowledge at this time to substantiate that indeed one single borderline reading could indicate the onset of hypertension in service.

If in the future another indicator of essential hypertension would become recognized, and demonstrated to be present during the period of latency, at times as long as 30 years, hypertension then might be considered originating in service.

Obviously, in 5% of the cases of secondary hypertension, the date of onset of the primary disease could definitely relate the hypertension to service.

Pension Considerations for Essential Hypertension ...CONTINUED

b) **Labile Hypertension**

"Labile Hypertension" will be identified by some physicians during service covered by the Insurance Principle. This term is used by the physicians to describe individuals in whom unusually wide swings in the blood pressure readings occur normally. This is often found in individuals whose vascular system reacts more readily to physical and emotional factors.

4. **Military Service in Peacetime**

Hypertension occurring in Peacetime service can rarely, if ever, be considered to have arisen out of or have been directly connected with that service except when the hypertension is of the secondary type; i.e. in 5% of the total cases of hypertension, when the basic disease, acute or chronic nephritis, occurs during service.

Summary - For use, if considered necessary, in decisions:

1. **Essential Hypertension, i.e. 95% of the cases**

Essential Hypertension is a primary condition in which recorded blood pressures consistently exceed the normal range, the cause of which is unknown. Most cases show no clinical signs or symptoms, with hypertension being found at routine examination or following a vascular complication (stroke, heart attack). Heredity, age and constitution appear to be the major factors in the etiology. Environmental factors do not affect its course with the exception of infection of the renal tract, and prolonged and exceptional physical and/or emotional stress. Both of these exceptions may play a part in the development of hypertension.

2. **Secondary Hypertension, i.e. 5% of the cases**

Secondary Hypertension occurs as part and parcel of a primary disease. Thus no separate ruling for hypertension as consequential upon the primary disease is required. This would include conditions such as glomerulonephritis, congenital and acquired conditions affecting the calibre of the major renal vessels, arteritis nodosa, and some disease affecting the endocrine glands.

References

Hypertension Manual, John H. Laragh, First Edition, 1974

Cardiac and Vascular Disease, Hedley L. Conn, Orville Horwitz, 1971

The Heart, J. Willis Hurst, Third Edition, 1986

Cecil: Textbook of Medicine, 1985

Harrison's Principles of Internal Medicine, Seventh Edition, 1977

Oxford Textbook of Medicine, 1983

References ...CONTINUED

Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels, Criteria Committee, New York Heart Association, Seventh Edition
Published Excerpts from Current Literature, including the Framingham Study.

MEDICAL GUIDELINES **THE CONCEPT OF LABILE HYPERTENSION**

This is an older term which was used to describe hypertension which appeared to be transitory. The duration of blood pressure elevations in most cases was for minutes or hours, and in the vast majority of these cases followed over the years, sustained blood pressure elevation (hypertension) did not develop.
(Madsen, P.E.R., Buch, J. Aerospace Med., 1971)

It is now recognized that the blood pressure in both normal persons as well as in hypertensive persons is to some extent labile, in that transient elevations can occur in response to the minor emotional circumstances of everyday life. Such elevations are part of the body's adaptation to the environment and are normal, and harmless, and cannot be used to support a diagnosis of hypertension. For this reason it is recommended that the designation of Labile Hypertension be discarded (Stein: Internal Medicine 1987).

For the above reasons the most meaningful blood pressure measurements are obtained under conditions of tranquillity, and it is recommended also that coffee and cigarettes be avoided in the preceding hour. A diagnosis of hypertension requires evidence of sustained elevation of blood pressure in excess of 140/90 mm. Hg.

Pension Considerations:

1. **Lower Limb Amputees**

a) Below Knee Amputees:

As a general policy, it is felt that a below knee amputation predisposes to the development of and/or aggravation of degenerative osteoarthritic changes in the opposite lower limb. This influence is felt to a minimal degree in the opposite ankle, a moderate degree in the opposite knee and a moderate degree in the opposite hip. In addition, it affects degenerative changes in the lumbar-sacral spine to a mild to moderate degree.

In the normal person the ankle joint and articulation in the bones of the foot are subject to stresses and strains during normal walking and even more so during periods of increased activity or on uneven terrain. In the amputee such compensatory measures are gone and stresses and strains are transmitted proximal to the nearest joint which in below knee amputees would be the knee. It is felt that such stresses and strains may influence the development of degenerative changes within the knee joint to a certain extent. In determining the extent one ought to consider age of onset of degenerative changes, severity of changes and the length of the amputated stump.

A longer stump would act like a lever and cause more torsional force upon the knee joint than a shorter stump. In general a claim for osteoarthritis of a knee consequential to a lower leg amputation would be considered consequential to a minimal to moderate degree. Assessment for the amputation includes any complications of the stump such as chronic dermatitis, ulcer or abscesses. It is not felt that a below knee amputation in any way contributes to nor aggravates degenerative changes in the hip joint of the amputated leg.

b) Above Knee Amputees:

As with below knee amputees, it is felt that above knee amputations contribute to degenerative changes in the opposite ankle to a minimal degree, the opposite knee to a moderate degree and the opposite hip to a moderate degree. Once again degenerative changes in the lumbar sacral spine are considered influenced to a moderate degree.

In above knee amputees the stresses and strains normally sustained by the foot, ankle and knee joints are transmitted to the ipsilateral hip joint and may influence the development of degenerative changes in this joint.

Pension considerations are as for lower amputees and claims for osteoarthritis of the hip consequential to above knee amputation should again be considered consequential to a minimal to moderate degree.

Pension Considerations: ...CONTINUED

2. **SHORT LEG PROBLEMS**

In considering the influence of a shortened leg on other joints in the lower limbs, and upon the lumbar spine, one must consider altered gait. For such alteration to be significant, that is, to conduce to significant mechanical stresses on joints, it is felt that a length discrepancy of 3 cm or more should be evident. Any less is within the ability of normal compensatory adjustments such as pelvic tilt, slight scoliosis, and perhaps the use of a shoe lift to negate by rendering distribution of weight more nearly equal. The shortening may be real or functional, as in loss of ability to fully extend the knee. Measurements of leg length are notoriously variable depending upon technique of the observer, but usually suffice to establish whether the discrepancy is significant.

The presence of a shorter leg is not felt to influence the degenerative process in any joint of the shortened limb. However, it is considered that additional workload evolves upon the opposite limb, and as a result, degenerative changes to the opposite ankle may be influenced to a minimal degree, changes in the opposite knee to a moderate degree, as also degenerative changes in the lumbar spine.

3. **DISTURBANCES THAT ALTER GAIT BUT ARE NOT THE RESULT OF A REAL OR FUNCTIONAL SHORTENING**

In general, it is not considered that an altered gait per se, in the absence of any real or functional shortening, contributes in any significant manner to degenerative changes in joints of either limb.

Pes planus, pes cavus or hallux valgus are generally not considered to alter gait mechanics to a sufficient degree to conduce to degenerative changes, either in the lower limbs or in the lumbo-sacral spine.

MEDICAL GUIDELINES **LOWER EXTREMITIES**
VARICOSE VEINS & THROMBOPHLEBITIS

DEFINITION

Varicose veins of the lower extremities are dilated, elongated veins involving the superficial venous system and the communicating veins.

ANATOMICAL AND PHYSIOLOGICAL FACTORS

The venous system of the lower extremities consist of:

1. The deep system of veins.

2. The superficial veins system.

3. The communicating (or perforating) veins which connect the first two systems.

The deep venous system is centrally placed in the lower extremity, and is surrounded by supporting muscles which prevent dilation of the veins. As a result, the deep veins do not become dilated and elongated (varicose). The superficial veins system runs in the fatty layer between the skin and the fibrous layers surrounding the muscles (fascia). They are not supported by a resistant structure and so can dilate and elongate and become varicose. The communicating veins run between the superficial system and the deep system of veins.

All three systems of veins have valves preventing downward flow of blood in the superficial and deep systems and preventing outward flow from the deep system to the superficial system in the communicating veins.

In a normally functioning venous system, the deep veins are of most importance. The muscles surrounding deep veins by their contractions force the blood from the deep system to the superficial system in the communicating veins.

In a normally functioning venous system, the deep veins are of most importance. The muscles surrounding the deep veins by their contractions force the blood from the deep veins into the abdominal veins. Following the relaxation of the muscles, there is a temporary decrease in the deep vein pressure and blood flows from the superficial veins through the communicating veins into the deep system. Whole blood does flow upward through the superficial veins to enter the deep system in the groin, a large part of the blood flow is through the communicating veins to the deep veins lower down in the extremity.

ETIOLOGY

Hereditary or developmental factors include:

1. Absence of valves.

2. Incomplete valves.

3. Incompetent fibrous or elastic tissues in the vein wall.

ETIOLOGY - CONTINUED

Hydrostatic pressure of the blood carried in the veins is a primary factor. The erect posture causes the vein wall and valves to be exposed to the pressure produced by the weight of the uninterrupted column of blood above the valve. In time, this extra weight of blood will stretch the wall competent valve. In time, this extra weight of blood will stretch the wall of the vein at the site of the lower valve, pull the valve apart so that it becomes incompetent and the pressure of the blood is then passed lower to the next competent valve. In this way, the dilation of the veins gradually progresses towards the foot and as the veins themselves become smaller and thinner farther down the extremity, they dilate more readily.

SECONDARY CAUSES

1. Occasionally an incompetent communicating vein develops at the site of a localized injury to the thigh or leg. This may be due to rupture of the valve, or destruction of the valve of the communicating vein following localized thrombosis and inflammation of the site of trauma.

2. In cases of deep vein thrombosis, varicose veins may arise on a secondary basis due to the deep vein obstruction.

DEEP VEIN THROMBOSIS

The process of deep vein thrombosis (deep phlebitis) frequently destroys the valves in the perforating or communicating veins. After the process subsides, the major portion of the venous blood is still returned through recanalized veins affected by the thrombosis or through dilated deep collateral veins not affected by the thrombotic process. However, due to the lack of competent valves in the communicating veins, superficial varicose veins may develop. The degree of such varicose veins is dependent upon the competence of the upward flow of blood in the deep veins and the degree of damage to the valves of the communicating veins.

Deep veins thrombosis occurs:

1. Following injury to the lower extremities, particularly with immobilization.

2. During immobilization in bed for other reasons both medical and surgical, e.g., heart disease, lung disease, postoperatively, etc. and may lead to complications such as pulmonary infarction due to an embolus.

There may be swelling of the ankle and lower leg subsequently which may improve as the deep and superficial veins re-canalize to allow a greater flow of blood.

A causal diagnosis of varicose veins is sometimes made in error when the veins are prominent but neither varicose or abnormal. Repeated examination thereafter may confirm that the veins are not varicose.

Pension Considerations:

1. A ruling should be requested on varicose veins one leg only, if the disease is limited to one leg or was limited to that leg during service and in the early post-discharge period.

2. Varicose veins may be presented for ruling as a bilateral condition if the records establish clearly that the same relationship to service exists with respect to both legs.

3. Varicose veins may be submitted for ruling as due to an injury only when the veins develop in close proximity to the site of injury.

4. Superficial phlebitis is a common complication of varicose veins and is accepted as part of the disability without separate ruling.

5. Aggravation of pre-enlistment varicose veins can reasonably be considered to have occurred if the varicose veins are larger or more extensive on discharge or in the immediate post-discharge period than prior to enlistment.

6. If an operation has been required for the varicose veins during service, it is suggested that aggravation be assessed on a minimal basis even if no assessable disability is present at discharge.

7. Aggravation of pre-enlistment varicose veins by Regular Force service should rarely be conceded, as the conditions required to produce an aggravation above and beyond the natural progress of the disease, are rarely encountered in Regular Force service.

8. When a decision has been rendered on varicose veins, not specifying right or left, the diagnosis would not normally be revised for separate rulings on right and left legs, unless revision is requested by the applicant and this is considered medically reasonable.

9. When varicose veins of unknown etiology appear in both legs at an interval of less than two years, they may be submitted for ruling as a single disease process if the claim is under subsection 21(1).

10. There is statistical evidence that when people are confined to bed (e.g., in coronary cases) there is a higher incidence of deep vein thrombosis in those persons who have varicose veins.

11. In cases of deep thrombophlebitis recent studies using labelled I. Fibrinogen, indicate that even in the absence of clinical signs the deep vein involvement is bilateral in 40% of cases. For this reason entitlement for deep vein thrombosis accepts that the condition is probably bilateral.

REFERENCES

1. Principles of Surgery - Schwartz
McGraw-Hill Inc. 1969

2. Surgery, Principles and Practice
Rhoads, Allen, Harkins, Moyer - Fourth Edition 1970

3. Peripheral Vascular Disease - Allen, Barker, Hines
Fairbairn - Juergens - Spittel - Fourth Edition 1972

4. Pathology
Anderson - Sixth Edition 1971

In multiple sclerosis there is partial loss of the myelin covering which surrounds the axons of nerve cells. This myelin is destroyed in a patchy distribution along the course of bundles of axons, and lesions may be found in the brain, brainstem or spinal cord. The cause of the destruction of myelin is not known. Research studies indicate, however, that multiple sclerosis is related to some environmental factor which is encountered in childhood, and which, after years of latency, either evokes the disease or contributes to its causation. This factor is thought to be a virus, although to date no virus has been seen in, or isolated from, affected tissue. Linked with this is the postulation of a secondary factor, an autoimmune reaction, that results in the destruction of the myelin.

A number of triggering factors, such as infection, trauma, and pregnancy, have been suggested but none has been convincingly related to either first attacks or exacerbations of the disease.

The majority of cases of multiple sclerosis have their onset between 20 and 40 years of age, but it may appear in the fifties or even sixties. The disease is intermittently progressive, with periods of exacerbation and remission. The initial manifestation may be as little as a slight weakness in a lower extremity, or as much as partial loss of vision. Often there is full recovery from this first attack, and there may be no further symptoms for as long as ten years. Eventually, however, there is disturbance of sensation and muscle power in the limbs, disturbance of gait, bladder dysfunction and a wide variety of brain stem and cerebellar deficits. Dementia and seizure disorders may develop late in the course of the disease.

Because of the variability of symptoms and their combination, assessment must be made on an individual basis, with consideration given not only to specific deficits, but also to the overall effect of these elements on independence and activities of daily living.

These include disorders of the central nervous system, that is, the brain and spinal cord, and of the peripheral nervous system, which includes the cranial nerves.

Although these disorders can be classified on an etiological basis, i.e., according to their cause, many of the conditions are of unknown, uncertain or variable cause and these are identified and discussed individually.

The types of disabilities which may result from neurological disease may be listed as follows:

Central Nervous System

-Brain

- Sensory disturbances (loss of, distortion of, or exaggerated sensation).
- Motor disturbances (paralysis, loss of fine movements, presence of involuntary movements).
- Communication disturbances (speech).
- Complex integrated cerebral function disturbances, i.e. organic brain syndrome with memory and orientation deficits.
- Episodic neurological disorders, e.g. epilepsy.

Spinal Cord

- Motor disturbances
- Sensory disturbances
- Gait disturbances
- Impairment of bladder, bowel and sexual function

Peripheral Nervous System

- Peripheral nerves
  - Peripheral neuropathy (polyneuropathy)
  - Impairment of individual nerves (e.g. ulnar neuropathy)
- Cranial nerve lesions

The following is a mixed classification, since a purely etiological classification is not practical for pension purposes:

1. Convulsive disorders (epilepsy)
2. Cerebrovascular diseases
3. Traumatic disorders of the brain
4. Brain and spinal cord tumors
5. Infectious diseases of the nervous system
6. Multiple sclerosis
7. Degenerative disorders
   - Alzheimer's disease
   - Parkinson's disease
8. Developmental and congenital anomalies
9. Diseases of the spinal cord
10. Diseases of the cranial nerves
11. Entrapment neuropathies
12. Peripheral neuropathies
13. Nutritional disorders of the nervous system
14. Headache (tension, vascular, post-traumatic, mixed)

MEDICAL GUIDELINES **NOSE, THROAT, AND RELATED CONDITIONS**

Sinusitis

Acute sinusitis is a self-limiting disease. Chronic sinusitis is curable. It rarely causes headache, nasal obstruction, or any symptoms aside from nasal and post-nasal discharge. A Medical Examiner may see pus in the nose, but the only sure way of diagnosing sinusitis is by X-ray. Radiology reports of "thickened mucous membrane" or "polyps" are not evidence of sinusitis. Only a sinus that is opaque to X-rays or contains fluid is the seat of sinusitis.

Chronic Sinusitis and Bronchitis

There is argument whether chronic sinusitis can lead to chronic bronchitis. Veterans Affairs Canada policy is that a minimal (1/5) consequential relation may exist.

Allergic and Vasomotor Rhinitis

Unlike chronic sinusitis, these conditions do almost invariably cause nasal obstruction and headache and, like sinusitis, nasal and post-nasal discharge. They are not caused by infection, the post-nasal drip is not infected, and the conditions cannot and do not lead to chronic bronchitis. They may, particularly allergic rhinitis, be associated with bronchial asthma but they do not cause it, and asthma is not a consequence of either allergic or vasomotor rhinitis.

Deviated Nasal Septum

A deviation of the nasal septum is most commonly caused by trauma, and most commonly trauma at birth. Much less commonly, it is a developmental defect. A slight deviation of the nasal septum with good airways means that the nose is normal.

There is argument whether a deviated nasal septum can lead to chronic sinusitis or chronic bronchitis. Veterans Affairs Canada policy is that a minimal (1/5) consequential relation may exist.

Tracheostomy

In some rare circumstances, for example, a gunshot wound of the larynx, a permanent tracheostomy is necessary. It causes some disability - for example, it prevents the patient from swimming.

Adipose tissue (fat) is a normal component of the human body. Obesity may be defined as an excess of adipose tissue.

Life Insurance Tables are readily available and record "desirable weights" based on age, sex, build and height. Relative weight (RW) is the relationship of measured body weight to the "desirable weight". Most authorities consider relative weights over 120% to constitute mild obesity, 140% to 200% moderate obesity and over 200% as severe or morbid obesity.

Although obesity is a common presenting endocrine complaint, the overwhelming majority of cases are due to excessive food intake or physical inactivity or both. Obesity is always the result of a positive energy balance and will not occur if "excess" calories are not ingested. This is true for all types of obesity.

Medical research has clearly established that the amount of fat in the body is not,of itself, a disease or evidence of a disease process. Nevertheless, obesity does predispose individuals to development of other disease processes.

Regulation of body fat by proper calorie consumption is a voluntary decision and no assessable disability results if caloric intake is regulated. Obesity is not considered to be a disease process which causes disability within the meaning of the Pension Act.

Provided certain clinical evidence of some pensioned condition is present, a degree of obesity consequential may be considered. These include some endocrine disorders (hypothyroidism, Cushing's Syndrome), some types of brain tumors and brain damage, and resulting side effects of certain medications such as corticosteroids or phenothiazines and certain antihistamines.

In general, if entitlement for obesity is granted, assessment should be in the range of 0% to 10%. Rarely a 15% assessment may be justified.

PREAMBLE:

The eye is the organ of vision. The diagram below shows its structure in cross section.

Rays of light strike the cornea, the window of the eye, pass through the anterior chamber of the lens where they are focused, after passage through the vitreous, onto the retina. From the light received, the retinal nerve cells generate an image, and transmit impulses, via the optic nerve, through the brain to the occipital cortex, where the images are interpreted.

The presence of two eyes enables binocular vision to be appreciated. This results from careful co-ordination of the movements of both eyes together. Objects are seen in three dimensions giving perception of depth.

VISUAL ACUITY

The visual acuity is a measure of how far a person can see. It is measured on a standard (Snellens Test chart) which is placed 6 meters (or 20 ft.) from the patient. The top letter is seen by a normal eye at a distance of 60 metres (200 ft.). The subsequent ones at 36 metres (120 ft.), 18 (70 ft.), 12 (40 ft.), 9 (30 ft.), and 6, 20/20 . The visual acuity is expressed as a fraction. The six (6) or (20) indicates the patient's distance in metres or feet from the chart, and the other figure, the letter seen. Thus a perfect visual acuity is 6/6 or 20/20. Glasses can improve visual acuity so a note should always be made as to whether the testing has been with, or without, glasses.

VISUAL ACUITY...CONTINUED

If a visual acuity is less than 6/60, but the patient is not totally blind, further testing is required, as follows:

1. The patient is advanced towards the big letter, and if it can be identified at a closer distance then 5/60, 4/60, 3/60, 2/60 or 1/60 is recorded accordingly.

2. If the top letter is not identifiable the patient counts the examiner's fingers 0.5 metre and 1 metre.

3. If this fails, identification of hand movement at 0.5 metre and 1 metre is tried.

4. Finally, the ability to perceive light is tested - if this fails, the eye is totally blind.

A blind eye has a non-reactive pupil. (There may be variations in tested visual acuity which suggest worsening of eyesight, but these findings are due to variations in testing techniques and co-operation of patients.)

The C.N.I.B. provides the following statement regarding legal blindness:

A person is considered legally blind if the visual acuity in both eyes with proper refractive lenses is 20/200 (6/60) or less with the Snellen Chart or equivalent, or if the greatest diameter of the field of vision in both eyes is less than 20°. In simple terms, if one is able at a distance of 20 feet (6 meters) to read only the large 'E'on the Snellen Chart, that person's vision is likely to be within the range of legal blindness.

It must be stressed that legal blindness does not mean total absence of vision: approximately ninety percent of the known legally blind population have some useful vision.

ABNORMALITIES OF MOTILITY

Strabismus - Squint

The movements of each eye are governed by six muscles working together as a team. Strabismus results when this mechanism breaks down.

Strabismus may be congenital or acquired.

Congenital strabismus occurs at birth or early in life. One eye deviates off the visual axis and becomes convergent (turning in) or divergent (turning out). The squinting eye usually has subnormal vision, or amblyopia.

An **acquired strabismus** may be:

i) Acute
ii) Established

i) The cardinal symptoms of an acute onset of strabismus is DIPLOPIA. The commonest cause is paralysis of one of the extra-ocular muscles. This can occur as a result of trauma, other eye diseases (Herpes Zoster, Temporal Arteritis) or general diseases such as Diabetes, Multiple Sclerosis, Myasthenia Gravis, Occlusive Vascular Disease, and brain tumour.

ABNORMALITIES OF MOTILITY...CONTINUED

ii) Established strabismus sometimes develops some years after the loss of vision of the deviating eye. It usually deviates out causing a "Secondary Divergent Strabismus".

AMBLYOPIA

Amblyopia is a developmental condition, and caused by an unconscious suppression of vision in one eye, at an early age; this is usually because suppression of vision in one eye, at an early age; this is usually because the eye has a high refractive error, and is sending a blurred image to the brain.

Pension Considerations:

It is pre-enlistment in origin, by its very nature. The actual vision of the amblyopic eye remains constant from an early age: certainly by nine years of age.

NYSTAGMUS

Nystagmus is the term applied to rapid, shaky movements of the eyes. It is independent of normal extra-ocular movement which is unaffected.

Pension Considerations:

Congenital nystagmus is always associated with subnormal vision.

Acquired nystagmus is associated either with disease of the balance mechanism of the ear, or a neurological disorder, e.g. multiple sclerosis.

ERRORS OF REFRACTION OR IMPAIRED VISION

Definition:

Emmetropia is the condition of the normal eye. Ametropia is that condition in which incident-parallel rays of light do not come to a focus upon the retina. It is divided into:

1. Myopia
2. Hypermetropia
3. Astigmatism

General:

Myopia, hypermetropia and astigmatism are errors of refraction and arise from one or more of the following conditions:

1. Abnormal length of globe - too long in myopia, too short in hypermetropia.

2. Abnormal curvature of the refracting surfaces of the cornea or lens, the curvature being too strong in myopia and too weak in hypermetropia and different in different meridians in astigmatism.

3. Abnormal refractive indices of the media.

ERRORS OF REFRACTION OR IMPAIRED VISION...CONTINUED

4. Abnormal position of the lens - displacement forwards in myopia and backwards in hypermetropia.

Accommodation is the ability to produce a clear retinal image at varying distances. This is achieved by alteration in the curvature of the lens of the eye. The lens is suspended within the circle of the ciliary body which by contraction allows the curvature of the lens to increase for the perception of close objects. Presbyopia develops in all normal subjects, when accommodation fails around the age of 40 and glasses are needed for reading.

MYOPIA

Myopia, (or shortsightedness) is the condition where the rays of light come to focus in front of the retina. Myopia may be classified as:

1. Simple
2. Degenerative

Simple Myopia

The majority of cases of myopia are termed simple because they are in no sense pathological, there being no related degenerative changes in the eye.

Degenerative Myopia

In this type of case the myopia is of a high degree and associated with pathological changes in the eye - degenerative changes in the choroid and retina, vitreous opacities and cataract formation. The refractive error appears in childhood and the degenerative changes usually appear about the fifth decade.

The only symptom in low myopia may be indistinct distant vision. In addition there may be discomfort after near work due to "eye strain".

Pension Considerations:

Myopia is genetically determined. Environmental factors such as excessive near work, working in artificial light, etc., do not influence the progress of the condition.

HYPERMETROPIA

Hypermetropia (or long-sightedness) is the condition where rays of light come to focus behind the retina.

1. Developmental

Developmental hypermetropia is the normal optical condition in infants, and persists throughout life in some 50 percent of the population in most countries of the world.

Pension Considerations:

Hypermetropia is a developmental condition, the progress of which is uninfluenced by environmental factors. Whilst excessive reading, poor lighting, etc. may give rise to symptoms, they do not in any way influence the progress of the condition.

Assessment of the degree of disability due to refractive errors is made by reference to the Table of Disabilities - Visual acuity, 8.01. The visual acuity with, and without glasses should be included in an ophthalmological report.

ASTIGMATISM

Astigmatism is the condition of refraction in which a point of light cannot be made to produce a punctate image on the retina.

Pension Considerations:

Regular astigmatism is most commonly a congenital defect. Rarely it may be traumatic following a wound, usually surgical, in the corneal scleral margin. Irregular astigmatism occurs almost entirely from diseases of the cornea, e.g. keratitis or virus infections. Injuries to the eyes may also give rise to this condition. As with myopia and hypermetropia, environmental factors including constant use, even in conditions of bad visibility, may reveal the abnormality but will never cause astigmatism, nor make it worse.

EXTERNAL STRUCTURES

The eyeball is extremely well protected. It sits in a bony socket called the orbit and is protected in front by the eyelids and tear film.

Fractures of the orbit can occur. Two have serious consequences:

1. Damage to the optic nerve can possibly cause optic atrophy.

2. Fracture of the floor of the orbit can cause double vision. It usually resolves in about three weeks. Surgical intervention is sometimes necessary. Complete recovery is usual.

Eyelids

The blink reflex is one of the most efficient of the body, and many potential dangers to the eye are averted by this means.

BLEPHAROSPASM, or excessive blinking of the eyelids, is rare, but can be associated with facial tics.

The eyelid margins hold the eyelashes. An intact, regular margin is essential for good lid function.

Pension Considerations:

Affections of the eyelid fall into the following groups:

1. Malpositions
2. Trauma
3. Infections

4. Cyst formation
5. New growth

1. **Ptosis** or drooping of the eyelid may be cosmetically disfiguring but unless the pupil is covered, there is no interference with vision and therefore is not a disability.

2. **Trauma** to the lids (lacerations) can usually be easily repaired without subsequent complications. The important thing is to align the margins of the eyelids. If this is not achieved, interruption of the tear film, with later corneal scarring, may occur. Scarring at the lid margin may also distort the direction of the eyelashes so they may grow in towards the eye and irritate it. This is known as **Trichiasis.** Hemorrhage can occur into the eyelid substance causing gross swelling, but it recovers.

3. **Infections** of the eyelid margins are known as **BLEPHARITIS**. Blepharitis may be:

   a) squamous - associated with dandruff
   b) ulcerative - due to bacteria, or;
   c) allergic

   Infections of the eyelid proper are rare - Herpes Zoster virus is the commonest.

4. **Cyst** can occur in the glands which are present along the eyelid margin. Such an example is a chalazion or meibomian cyst. They present no danger to the function of the eye and are not a disability.

5. **Tumours**. The commonest tumour is a rodent ulcer, a local form of skin cancer which can be removed. Other forms of skin cancer can occur. Benign tumours, e.g. warts, are not uncommon and can be removed.

Tears

The lacrimal (or tear) system plays an important role in the protection of the eye. Tears are constantly secreted by the tear gland, situated under the upper eyelid at its upper end, and pass over the front of the eyeball where they wash away any foreign particles which have entered the eye. They drain down the tear ducts into the nose.

Dry Eye Syndrome is well recognized and is due to a reduction of tears. Subjects affected with the condition suffer with chronically uncomfortable, often red eyes, and should live in humid environments where possible.

Epiphora is the term applied when there is an excess of tears. It is due to a block in the tear draining mechanism, which may damaged by direct trauma. It should be regarded as a discomfort, and not a disability.

Commonly, draining of tears becomes less efficient with age. Vision may be blurred by the excess tear film, but is never permanently damaged.

CONJUNCTIVA

The conjunctival sac lines the eyelids and is inserted into the peripheral circumference of the cornea, known as the limbus. It has no direct communication with the sinuses.

CONJUNCTIVA...CONTINUED

The following affections of the conjunctiva may occur:

1. Infection
2. Trauma
3. Chemical Irritation
4. New Growths
5. Allergies

Pension Considerations:

1. Most bacteria, viri, and some fungi can infect the conjunctival sac, but the disease conjunctivitis responds well to treatment, is self limiting, and recovery takes place.

2. **Trauma.** Haemorrhages and lacerations can occur, but recovery is always complete in simple conjunctival lesions.

3. **Chemical Irritation**, e.g. tear gas, is uncomfortable, but leaves no permanent damage. More serious arealkali burns which can seriously disturb the tissue, making it much more sticky so the eyelids stick together. This is known as symblepharon. Serious interference with eye function may occur. Acid burns are less serious and seldom result in complications.

4. **New Growths**. Cancerous growths of the conjunctiva are very rare. Benign pigmentary clumps of cells, melanomata or moles, are more common and of little significance.

   A **Pterygium** is not a cancerous growth. It is an invasion, led by the spearhead of a triangle of conjunctival tissue into the cornea. The cause is due to exposure, e.g. desert and arctic environments, and there is a very definite geographical distribution. Pterygia can be removed quite easily, but the recurrence rate is high. Corneal scarring, with some reduction in visual acuity, may result.

   Pingueculum is a whitish lump on the conjunctiva, usually on the limbus at 3 or 9 o'clock. It is due to fatty degeneration in the conjunctiva. It is never serious and does not affect vision.

CORNEA

This structure is the window of the eye and has no blood supply. It is an extremely important structure, forming, as it does, part of the tough outer coat of the eye. Transparency has to be maintained for good vision.

The following disturbances of the cornea may occur:

1. Congenital degenerations or dystrophies
2. Infections of the cornea - KERATITIS
3. Trauma
4. Disturbances secondary to other eye diseases.

   Infections of the cornea are usually viral, occasionally fungal. They may be recurrent and leave the tissue scarred. In such instances vision is impaired.

Infections may be secondary to corneal ulcer.

Trauma.

a) Direct. This may take any form; common causes are small lodged foreign bodies or an abrasion from a twig or baby's finger. Healing of an abrasion within a week is normal.

Mustard gas and alkalis can cause permanent corneal damage.

b) Indirect trauma from ultra violet light can cause arc eye (e.g. welding) and snow blindness. Sun ray lamps without protection can also cause eye damage. With treatment, recovery from U.V. light damage is complete.

Contact lenses are placed on the cornea of the eye, and can occasionally give rise to corneal problems.

CATARACTS

A cataract develops when opacities form in the lens of the eye. These changes in the lens usually start slowly and are progressive until full development or maturity occurs. In the early stages visual impairment is minimal, but as progression occurs it becomes more severe, and, in many cases, the cataract has to be removed to restore vision.

A cataract extraction involves removing the lens of the eye. An eye without a lens is called Aphakic. An aphakic eye has lost its power of focusing. This can be corrected by spectacles, a contact lens, or an intra-ocular implant, which is an artificial lens placed inside the eye.

Because the lens of the eye has been replaced by a synthetic one, an eye with an intra-ocular implant is not aphakic.

Spectacle correction of aphakia results in an enlarged image, which is reduced by contact lens correction, and eliminated by an intra-ocular implant. See assessment for Aphakia - 8.03 in the Table of Disabilities.

Cataracts may be congenital, acquired, or associated with a generalized disease, e.g. Diabetes, myotonia dystrophia, galactosaemia (and others).

Acquired Causes of Cataract

a) **Senility** - opacification of the lens, i.e. part of the normal ageing process, and a senile cataract is the commonest type of acquired cataract. Lens opacities occur, to some degree, in 65% of people over 50 and 95% over 65 years of age. This type of cataract is increasingly common, with the longer life spans which occur today.

b) **Trauma** - which may be either direct or indirect.

Direct trauma to a lens may occur when a foreign body penetrates the eye. This type of cataract is always unilateral.

Indirect trauma may result from:

(i) Blast
(ii) Heat - (Heat cataracts are very specialized and require expert diagnosis)
(iii) Ionizing Radiation (as from atomic explosions or radiation therapy)
(iv) Concussion, when the eyeball itself is hit without perforation
(v) Electricity (e.g. a flash of lightening, or high voltage current passing through the body)

c) **Toxins**

A toxic cataract may be caused by:

(i) Certain medications (in particular cortico-steroids and phospholine iodide eye drops)
(ii) Certain chemical - a "chemical" cataract is rare but can result from ingestion of thallium naphthalene, selenium or dinitrophenol.

Toxins in the atmosphere (e.g. mustard gas) do not cause cataracts. (A minimal association is recognized between cataract and avitaminosis).

d) **Secondary to other eye disease**

Pension Considerations:

1. The rate of progression of a cataract from the detection of the earliest lens opacities until full maturity varies, and is impossible to predict.

2. N.B. Research has shown that microwaves (as in radar installations) and Black Light (as used to detect flaws in metal) do not cause cataract.

3. Since we deal more and more with intra-ocular implantations of the crystalline, we have noticed a radical change as to the prognosis given in case of people suffering from cataracts and subject to be a candidate for a similar chirurgical procedure. See paragraph on aphakia.

RETINAL DETACHMENT

The retina may be described as the lining of the eye behind the lens which is responsible for sight. It is divided into peripheral and intermediate parts, plus the macula.

Clear central vision depends on good macula function. The other two zones are responsible for peripheral vision.

The retina is made up of several layers. A retinal detachment occurs when two of these layers separate. Usually, a break or hole in the retina occurs and fluid gradually seeps into the hole separating the layers. Thus a detachment may occur weeks or months after an injury.

Retinal detachments may result from:

a) Trauma (any trauma which damages the retina as signified by a retinal hemorrhage at the time of the injury;)

b) Peripheral retinal degenerations;

c) As part of certain generalized diseases, e.g. Marfans Syndrome. However, hypertension is not considered to be a cause.

d) Secondary to other eye disease, in particular, high myopia, chorioretinitis and retinitis pigmentosa.

OTHER RETINAL CONDITIONS

Diabetic Retinopathy:

This is a change in the retina which is secondary to the change which takes place in the small blood vessels of a diabetic person. These vessels may leak, forming plaques which affect vision if they are at the macula. They may grow new vessels, which bleed into the eye causing a vitreous hemorrhage, which seriously affects vision.

Macular Degeneration:

The macula is a specialized part of the retina, which falls right on the visual axis. It is responsible for clear vision.

Conditions of the macula seriously affect vision.

1. Macular Degeneration. By far the commonest cause of this is senile macular degeneration. It is a degenerative process in the cells of the retina which gradually becomes fewer in number, and thus vision becomes less clear. Basically, blood supply becomes reduced, new fragile blood vessels develop, bleed, plaques form, fibrosis takes place, and the whole macula may eventually become replaced with scar tissue.

2. Macular degenerations occurring early in life are almost always hereditary (e.g. Stargardts) in nature. Occasionally they are secondary to trauma.

Pension considerations:

Other macular conditions:

Chloroquine Retinopathy - A disturbance of the macula resulting from chloroquine medication. Dosage in malaria prophylaxis or treatment is seldom high enough to cause this side effect. It occurs mainly in patients receiving large amounts for rheumatoid arthritis.

Central Serous Retinopathy - Males between 25 and 40 are affected. An accumulation of fluid occurs at the macula. It reduces visual acuity to about 6/12. Recovery is complete in two to four months. The aetiology is a temporary underlying vascular anomaly at the site.

Optic Atrophy - Occurs when the optic nerve degenerates. This causes severe impairment of both visual acuity and the visual field. The most common forms are congenital. It can result from trauma when the optic nerve is damaged, or from a poor blood supply to the nerve.

Retrobulbar Neuritis - Presents in the young adult with progressive loss of vision in one eye over a period of 3-7 days until only the ability to identify hand movements remains, due to a central blind spot in the field of vision. The cause is a plaque of demyelination in the optic nerve. Recovery is usually complete and after 3-5 weeks the vision returns to normal. Very occasionally

it remains poor. Retrobulbar neuritis is often the first manifestation of Multiple Sclerosis, and 50% of patients progress to the development of the disease. Thus 50% of patients recovery is complete, and no further sequelae occur.

GLAUCOMA

Glaucoma occurs when an eye has a high intra-ocular pressure. A pressure of 21 mm Hg above atmospheric is considered potentially dangerous to the eye. The increased pressure may damage the optic nerve, the head of which is known as the optic disc, leading to cupping and pallar of the disc. This results in blind spots, known as scotomata, appearing in the field of vision. These may enlarge, several may coalesce, and lead to eventual blindness.

The intra-ocular pressure is maintained by a balance between the inflow and outflow of the aqueous humour, the fluid which nourishes the transparent structures of the eye. Aqueous humour is secreted constantly by the ciliary body, a gland behind the iris. It circulates around, before entering the anterior chamber of the eye through the pupil, then leaves by passing through the sieve-like structure known as the Trabecular Meshwork in the angle of this chamber and back into the circulation.

There are many different types of glaucoma. Glaucoma may be:

a) Primary
b) Secondary

Primary glaucoma has three classifications, viz:

1. Chronic Simple or Open Angle Glaucoma
2. Angle Closure or Congestive Glaucoma
3. Buphthalmos

1. Chronic Simple or Open Angle Glaucoma

This is the commonest form of the disease. World wide surveys suggest the incidence is in the region of 2% in the population over 40 years of age. Taking account of racial, geographical, climatic, cultural and nutritional differences, these surveys show an absence of any external factor in the aetiology of the disease. There is a definite and proven hereditary factor in this type of glaucoma, when the intra-ocular pressure becomes raised because the trabecular meshwork becomes blocked. This happens slowly over a lifetime

which is why the disease seldom becomes manifest before 40 years of age. It is asymptomatic in its early stages, but left untreated, cupping of the optic disc, visual field defects and eventual blindness occur.

2. **Closed Angle Glaucoma**

In this form of the disease, the angle of the anterior chamber is suddenly closed off and the intra-ocular pressure rises rapidly to very high levels, causing symptoms of pain and blurred vision. It usually occurs in the fifth and sixth decades of life and is more common in females. There is a definite predisposition to angle closure glaucoma, which usually occurs in far-sighted eyes and so shorter than normal, with shallow anterior chambers and narrow angles.

3. **Congenital Glaucoma - "Buphthalmos"**

This form of glaucoma is known as buphthalmos because the changes which take place in the cornea give the appearance of an "ox eye". The disease is due to congenital malformation of the angle of the anterior chamber. Over 80% of cases are diagnosed in the first three months of life.

Glaucoma may be secondary to:

1. Developmental Abnormalities (either local in the eye or systemic)
2. Acquired Defects

1. **Developmental Abnormalities**

Local developmental abnormalities include Axenfeld's Syndrome and Aniridia (absence of the iris).

More widespread disorders include:

a) Marfan's Syndrome)
Homocystinuria) both featuring dislocation of the lens
b) Neurofibromatosis - Von Recklinghausen's disease
c) The Sturge Weber Syndrome

GLAUCOMA ...CONTINUED

2. **Glaucoma Secondary to Acquired Defects**

(i) Trauma - injury to the eye can result in glaucoma. This may occur at the time of the injury, or many years later.

An increased intra-ocular hemorrhage blocks the drainage mechanisms. Delayed glaucoma is known as Angle Recession Glaucoma. In this condition, a split occurs in the angle of the anterior chamber and the resulting scar tissue damages the delicate drainage mechanisms. This causes a gradual rise in the intra-ocular pressure and hence the delay in the development of the disease.

(ii) Ocular inflammations, e.g. Iritis or keratitis.

(iii) Changes in the lens, e.g. Spontaneous rupture, dislocation, or swelling of the lens.

(iv) Vascular conditions, e.g. Central retinal vein thrombosis.

(v) Generalized diseases, e.g. Leukaemia and diabetes.

(vi) Other ocular disorders, e.g. Cysts, tumours, degenerations.

(vii) Drug induced glaucoma, e.g. Local steroid therapy.

In conclusion, glaucoma is a group of conditions in which the intra-ocular pressure is raised. The exact causes of primary glaucoma are not completely defined, but experts agree that they are endogenous and not acquired by contact with the environment. Certain external factors which may lead to the development of glaucoma have been identified.

There is no association between glaucoma and avitaminosis.

Primary osteoporosis is characterized by an absolute decrease in the amount of bone present to a level below which it is incapable of maintaining the structural integrity of the skeleton. The rate of bone formation is usually normal whereas the rate of bone resorption is usually increased. Whatever bone is present is normal morphologically; also serum calcium and phosphorus levels are normal in primary osteoporosis.

Osteoporosis is the most common metabolic bone disease and is clinically evidenced in middle life and beyond. Women are more frequently affected than men and the disease is often called "post-menopausal" osteoporosis or "senile" osteoporosis. Osteoporosis may be produced secondarily to a number of disorders including subtotal and total gastrectomy but most commonly it is of primary and unknown cause and is probably related to defective enzyme systems within the bone matrix, not in the mature bone itself.

Common causal factors in osteoporosis are:

1. Lack of activity: Moderate activity is beneficial in treating osteoporosis.

2. Estrogen deficiency: About 35% of women over 60 years of age have clinical osteoporosis. Estrogens appear to decrease bone absorption and administration of such in estrogen deficient women has been shown to markedly decrease the incidence of osteoporosis.

3. A chronic low intake of calcium has been suggested as an etiological agent.

4. Malabsorption in the operated intestinal tract may be a factor in elderly patients.

5. Deficient production of 1.25 - dihydroxy vitamin D may be a cause of some types.

Less common causal factors include:

a) Developmental disturbances: e.g. osteoporosis imperfecta.

b) Nutritional disturbances: e.g. anorexia nervosa, vitamin C deficiency, alcohol or caffeine excess, generally inadequate diet.

c) Endocrine diseases: e.g. hypopituitarism, acromegaly, thyrotoxicosis, Cushing's disease and rarely with longstanding uncontrolled diabetes mellitus.

d) Bone marrow disorder: as in leukemia or myeloma.

e) Prolonged use of Heparin.

f) Tobacco smoking: may contribute to osteoporosis.

Risk for osteoporosis are contained in the following table:

TABLE 13-7

Risk Factors for Osteoporosis

Genetic or Medical Factors	Life Style Factors
All races except blacks	High alcohol use
Previous fractures not due to major trauma	Smoking
Female relatives with osteoporosis	Low dietary intake of calcium
Thin body habitus	Lack of vitamin D
Early menopause (before age 40)	High- protein or high salt diet (promotes calciuria)
Inflammatory bowel disease; bowel resection	High caffeine use (more that 5 cups of coffee daily)
Prolonged use of corticosteroids, phenytoin, aluminum-containing antacids, or high does of thyroid	
Chronic renal failure	

In absence of any complications osteoporosis is not considered a significant disability nor disabling condition. Symptoms of backache due to osteoporosis should be assessed in the 0 to 10% range. When severe back symptoms are associated with evidence of vertebral collapse on x-ray, an assessment of 10-20% may be considered.

Fracture of long bones, commonly hip and wrist fractures, should be ruled on separately as consequential to osteoporosis. Kidney stones also require a separate consequential ruling.

X-ray evidence of osteoporosis in the absence of symptoms particulary in the skull and extremities should be assessed at nil.

Pension Considerations:

1. It may be possible to establish a consequential relationship between osteoporosis; e.g., any of several endocrine diseases, bone marrow disorders, gastric or intestinal resection or malabsorption due to inflammatory disease or enzyme deficiency of the bowel, prolonged use of certain drugs or hormones in treatment of pensioned conditions, chronic renal disease.

2. Activity may be so limited by pensioned conditions as to necessitate a sedentary life style which may conduce to osteoporosis.

3. Rarely dietary restrictions imposed by a pensioned condition may increase the likelihood of osteoporosis.

4. Generally, no specific etiologic factors can be identified in primary osteoporosis except in post-menopausal women, and any service relationship would be exceptional.

Definition

1. Paget's disease of bone is a chronic bone dystrophy in which irregular resorption and regeneration cause softening, thickening and deformity, with an increase of abnormal bone mass.

Clinical Manifestations

1. The lumbar vertebrae, sacrum, skull and pelvis are most often affected, though limb bones may also be involved. The disease may be confined to one bone, often a vertebra, or may be wide spread. It is a focal or multi-focal condition and not diffuse.

2. It typically affects the elderly, being rarely noted before the age of 50, though occurrence at an earlier age has been recorded. It is frequently asymptomatic, being discovered incidentally by X-ray or at necropsy. Deep bone pain, usually worse at night, may be a symptom. The development of deformity, spinal curvature, enlargement of the skull or bowing of the limbs may be observed. Pathological fractures may occur. Joints adjacent to affected bones are likely to develop osteoarthritis.

3. Less commonly it may present with cranial nerve lesions caused by involvement of cranial foramina, with renal stones, with heart failure due to the greatly increased blood supply to the affected skeleton, or with sarcomatous change in an affected bone. The risk of sarcomatous change is increased about 30 fold in bone affected by Paget's disease.

Aetiology

1. The aetiology of Paget's disease of bone is unknown. The disease is relatively rare in Asia and Africa, and quite common in the USA, UK, Australia, France and Germany. Men are affected more than women, and necropsy series in Germany and Britain show Paget's disease in about 3% of cases over the age of 40. A number of families have been reported with more than one member affected, but inheritance from father to son is rare. Most reported families are consistent with x-linked intermediate inheritance. Claims have been made for an inborn error of connective tissue metabolism, a disorder of hormone secretion, an acquired vascular disorder or an auto immune state, and more recently a viral aetiology has been proposed. None of these suggested aetiologies has been proven.

2. The results of numerous investigations over many years point to an endogenously determined break down in the later years of life of those processes of body chemistry which together control the maintenance of normal bone structure. There is no relationship to any specific infectious disease or infectious agent, nor is the disease affected by environment, diet, climate, stress or trauma.

Pension Considerations

1. The aetiology of Paget's disease of bone is unknown. It is accepted by leading authorities that it is a disease of endogenous origin which progresses independently of external factors.

References

Avioli L. V Diseases of Bone. In: Beeson P B, McDermott W, Wyngaarden J B, eds. Cecil Textbook of Medicine. 15th ed. Philadelphia: W B Saunders Company, 1979: 2225-2265

Catto M E. Disease of Bone. In: Anderson J R, ed. Muir's Textbook of Pathology. 11th ed. London: Edward Arnold, 1980: 875-913.

Fairbank T J. Osteitis Deformans. In Bodley Scott Sir Ronald, ed. Price's Textbook of the Practice of Medicine. 12th ed. Oxford: Oxford University Press. 1978: 977-978

MEDICAL GUIDELINES **PARKINSON DISEASE**

Parkinson's disease is a common condition that is observed in all countries, all ethnic groups and all socio-economic classes. Onset is usually between 40 and 70 years of age and the condition is gradually progressive. Pathological studies have revealed that in Parkinson disease there is a loss of pigmented cells in specific nuclei of the brain that are important in the fine-tuning of movement. The cause of this cell depletion is not known.

Lesions in, or dysfunctions of these nuclei, may occur in carbon monoxide poisoning, with certain prescription drugs, or in association with other disease processes, in which case symptoms similar to those of Parkinson disease develop. Such clinical pictures are called Parkinsonian syndrome, to distinguish them from Parkinson disease.

Parkinson disease may begin as an awkwardness in fine hand movement, but tremor and muscular rigidity are the classical signs of the condition. These symptoms are often unilateral at first, with gradual spread to the opposite side. Disturbance of gait, poverty and slowness of voluntary movement and expressionless face gradually develop. Eventually the ability to chew and swallow is affected and it may take an hour to eat a meal. Dementia occurs in about a third of cases.

Drug treatment, particularly with L-Dopa, reduces symptoms but does not prevent the slow advance of the disease. Surgical treatment may give good results in carefully selected cases. No matter what the treatment, however, the patient eventually becomes disabled to a greater or lesser extent.

The degree of disability is determined on the basis of abnormalities and deficits, and slowness of movements that result in interference with the normal activities of daily life. These include the ability to write, speak and respond to the demands of the work environment, the ability to shop and prepare meals, and the ability to perform basic self-care such as washing, dressing, eating. Deterioration of mental status is also included.

Each case must be assessed individually, on the basis of the particular symptomatology, and those who show a faster rate of deterioration should be seen more frequently to keep the assessment appropriate for the level of disability.

MEDICAL GUIDELINES **PATELLO-FEMORAL DYSFUNCTION-CHONDROMALACIA PATELLA**

Patello-femoral dysfunction refers to pain coming from the patello-femoral articulation without any gross anatomical changes in the articular cartilage of the patella. Many other names, patello-femoral pain syndrome, patellagia,patello-femoral compression syndrome, and patelo-femoral malalignment syndrome describe variants of the same clinical syndrome.

Chondromalacia patellae is a clinical syndrome similar to patello-femoral dysfunction, but involving pathological changes in the articular cartilage. Chondromalacia patella is generally classified as an overuse syndrome and as a result is frequently experienced by participants in virtually every fitness and sports activity. In some cases it is so common that it has the name of a particular activity, e.g. runner's knee.

In adolescents and young adults two types of chondromalacia patella are recognized:

1) The so-called primary or idiopathic type: this type is considered to be constitutional, usually involves both knees, and evolves insidiously and without obvious cause;

2) The secondary type, which may result from direct severe trauma or repeated minor trauma to the patella and involves only the injured knee. The trauma involved in this case is usually that of a direct blow to the patella but occasionally a severe twisting injury to the knee may be responsible.

A third type of chondromalacia patella, the adult type, is basically the beginning of degenerative osteoarthritis in the knee and etiological factors of osteoarthritis are considered to be applicable.

PENSION CONSIDERATIONS

1) Idiopathic Chondromalacia Patella is for all practical purposes a bilateral condition. Trauma does not cause this condition but may be an aggravating factor in progression of this disease. The type of trauma, severity of the trauma, and rate of deterioration of this condition are factors to be considered. As previously mentioned, the trauma is generally that of a severe direct blow to the patella. Occasionally a significant twisting injury may be responsible. It is not considered that so-called "repetitive micro trauma" play any role in development of chondromalacia patella.

2) Secondary Chrondromalacia Patella; Trauma may be an etiological agent in development of this condition. The time interval between the trauma and the onset of this condition is an important consideration. A time interval of a few weeks or months suggests a relationship, while a time interval of years certainly makes a relationship less likely. Once manifested, trauma may further aggravate the condition; once again the severity of the trauma and the rate of deterioration of the condition are factors to consider.

3) There is no medical data to indicate that chondromalacia patella is any more prevalent in service personnel that in the civilian population, and therefore no reason to believe that any alleged "repeated minor traumas" sustained during service play any role in development of this condition.

MEDICAL GUIDELINES **PERIPHERAL NEUROPATHIES**

GENERAL CONSIDERATIONS

The peripheral nervous system includes all neural structures which lie outside the brainstem and spinal cord. The olfactory bulbs and optic nerves are not included because they are highly specialized extensions of the brain itself. Cranial nerves III to XII, unlike the spinal nerves, may contain only motor fibres or only sensory fibres, or a mix of motor fibres, sensory fibres, and autonomic fibres. In contrast, all the spinal nerves are formed by separate motor and sensory roots arising from the spinal cord and receive autonomic fibres somewhere along their course. Cranial nerves exit through foramina in the skull, while spinal nerves exit through the intervertebral foramina formed by adjacent vertebrae. Many of the spinal nerves enter complex plexuses or networks where their fibres are rearranged into peripheral nerves which supply specific groups of muscles and adjacent skin.

When a nerve is affected by disease or injury, there may be an initial irritation which causes abnormal sensations, pain, muscle twitching, or cramps, but in most cases there is eventually a loss of muscle power and sensation. Nerve fibres react by degeneration of the nerve fibre itself, degeneration of its insulating sheath of myelin, or degeneration of both these elements. In some instances, such as trauma, regeneration of a peripheral nerve is possible. It is a very slow process, however, especially when the nerve fibre (axon) is affected, and may take as long as 2 or 3 years.

Polyneuropathies

Polyneuropathies, as the name implies, are conditions in which more than one peripheral nerve is affected. They may be acute or chronic, affect both sides symmetrically, or involve predominantly one side or one limb depending on the underlying cause.

Although uncommon, a number of genetically inherited polyneuropathies have been identified. In some a metabolic abnormality is responsible, in others the underlying mechanism is unknown.

The vast majority of polyneuropathies, however, develop in association with diabetes and the vitamin B deficiencies associated with chronic alcoholism. Other disease processes that occasionally give rise to polyneuropathies are hypothyroidism, the end stages of liver and kidney disease,and certain tumours.

Inadequate diet with a specific deficiency of the B vitamins also leads to the peripheral nerve disorders seen in beriberi and pellagra.

Toxins and poisons are other causes of polyneuropathy. The diphtheria bacillus produces a toxin which may act not only locally, paralysing the laryngeal muscles, but distally on the nerves of the limbs as well. Heavy metals such as arsenic, lead, mercury,and industrial solvents such as those used in the manufacture of plastics, may cause nerve damage. As well, certain drugs have been found to cause neuropathies in some patients.

In cases of severe trauma, such as injury in the region of a nerve plexus or extensive burns, there may be multiple nerve damage.

Finally, there are cases in which, despite intensive investigation, no cause can be identified, and the polyneuropathy must be labelled idiopathic.

Mononeuropathies

The great majority of mononeuropathies are due to local trauma. Gunshot, shrapnel and other penetrating wounds may crush or sever a nerve with recovery of muscle power and sensation directly related to the extent of the injury.

Where they lie close to a bone or joint, nerves may be damaged by fractures and dislocations. For example, fractures of the shaft of the humerus may involve the radial nerve, and elbow injuries the ulnar nerve.

Entrapment neuropathies develop where a nerve is subjected to abnormal pressure as it courses through a tight canal. A common site is the intervertebral foramen. Here, a herniated or degenerated disc presses on a spinal nerve as it eaves the spinal canal. The carpal tunnel, composed of carpal bones and a tough more superficial ligament, may be narrowed after carpal fractures or in rheumatoid arthritis, causing pressure on the median nerve, but in most cases there are signs of entrapment without discernible cause. Nerves may also undergo compression where they pass through two tendinous heads of origin of a muscle or between muscle layers. Nerve conduction studies may be valuable in identifying the site of such problems.

The most significant cranial nerve conditions are those which affect hearing, vision, or sense of smell (I, II, III, IV, VI, VIII). Besides these, there are two which deserve special mention; trigeminal neuralgia (tic douloureux) and Bell's palsy. Trigeminal neuralgia in those over 50 years of age is usually idiopathic in origin. It is characterized by intense facial pain which is triggered by activities such as chewing. Bell's palsy is an idiopathic facial paralysis of acute onset which usually recovers completely, although in some cases residual paralysis is seen.

Assessment Considerations

Where a neuropathy is part of the symptomatology of another disease entity, it is included in the assessment of that disease. Thus, diabetic neuropathy is assessed with diabetes. Similarly, spinal neuropathies caused by herniated discs or osteoarthritis of the spine are part and parcel of these conditions. Nerve injuries are included in assessments of the gunshot wounds, fractures, and dislocations which caused them. (Values are given in the sections on the upper and lower limbs.)

The main factors in assessing decrease or loss of nerve function are: the effect on present employment (if under retirement age), the effect on activities of daily living, and any pain or discomfort in the affected part.

MEDICAL GUIDELINES **PERIPHERAL NEUROPATHIES**

GENERAL CONSIDERATIONS

The peripheral nervous system includes all neural structures which lie outside the brainstem and spinal cord. The olfactory bulbs and optic nerves are not included because they are highly specialized extensions of the brain itself. Cranial nerves III to XII, unlike the spinal nerves, may contain only motor fibres or only sensory fibres, or a mix of motor fibres, sensory fibres, and autonomic fibres. In contrast, all the spinal nerves are formed by separate motor and sensory roots arising from the spinal cord and receive autonomic fibres somewhere along their course. Cranial nerves exit through foramina in the skull, while spinal nerves exit through the intervertebral foramina formed by adjacent vertebrae. Many of the spinal nerves enter complex plexuses or networks where their fibres are rearranged into peripheral nerves which supply specific groups of muscles and adjacent skin.

When a nerve is affected by disease or injury, there may be an initial irritation which causes abnormal sensations, pain, muscle twitching, or cramps, but in most cases there is eventually a loss of muscle power and sensation. Nerve fibres react by degeneration of the nerve fibre itself, degeneration of its insulating sheath of myelin, or degeneration of both these elements. In some instances, such as trauma, regeneration of a peripheral nerve is possible. It is a very slow process, however, especially when the nerve fibre (axon) is affected, and may take as long as 2 or 3 years.

Polyneuropathies

Polyneuropathies, as the name implies, are conditions in which more than one peripheral nerve is affected. They may be acute or chronic, affect both sides symmetrically, or involve predominantly one side or one limb depending on the underlying cause.

Although uncommon, a number of genetically inherited polyneuropathies have been identified. In some a metabolic abnormality is responsible, in others the underlying mechanism is unknown.

The vast majority of polyneuropathies, however, develop in association with diabetes and the vitamin B deficiencies associated with chronic alcoholism. Other disease processes that occasionally give rise to polyneuropathies are hypothyroidism, the end stages of liver and kidney disease,and certain tumours.

Inadequate diet with a specific deficiency of the B vitamins also leads to the peripheral nerve disorders seen in beriberi and pellagra.

Toxins and poisons are other causes of polyneuropathy. The diphtheria bacillus produces a toxin which may act not only locally, paralysing the laryngeal muscles, but distally on the nerves of the limbs as well. Heavy metals such as arsenic, lead, mercury,and industrial solvents such as those used in the manufacture of plastics, may cause nerve damage. As well, certain drugs have been found to cause neuropathies in some patients.

In cases of severe trauma, such as injury in the region of a nerve plexus or extensive burns, there may be multiple nerve damage.

Finally, there are cases in which, despite intensive investigation, no cause can be identified, and the polyneuropathy must be labelled idiopathic.

Mononeuropathies

The great majority of mononeuropathies are due to local trauma. Gunshot, shrapnel and other penetrating wounds may crush or sever a nerve with recovery of muscle power and sensation directly related to the extent of the injury.

Where they lie close to a bone or joint, nerves may be damaged by fractures and dislocations. For example, fractures of the shaft of the humerus may involve the radial nerve, and elbow injuries the ulnar nerve.

Entrapment neuropathies develop where a nerve is subjected to abnormal pressure as it courses through a tight canal. A common site is the intervertebral foramen. Here, a herniated or degenerated disc presses on a spinal nerve as it eaves the spinal canal. The carpal tunnel, composed of carpal bones and a tough more superficial ligament, may be narrowed after carpal fractures or in rheumatoid arthritis, causing pressure on the median nerve, but in most cases there are signs of entrapment without discernible cause. Nerves may also undergo compression where they pass through two tendinous heads of origin of a muscle or between muscle layers. Nerve conduction studies may be valuable in identifying the site of such problems.

The most significant cranial nerve conditions are those which affect hearing, vision, or sense of smell (I, II, III, IV, VI, VIII). Besides these, there are two which deserve special mention; trigeminal neuralgia (tic douloureux) and Bell's palsy. Trigeminal neuralgia in those over 50 years of age is usually idiopathic in origin. It is characterized by intense facial pain which is triggered by activities such as chewing. Bell's palsy is an idiopathic facial paralysis of acute onset which usually recovers completely, although in some cases residual paralysis is seen.

Assessment Considerations

Where a neuropathy is part of the symptomatology of another disease entity, it is included in the assessment of that disease. Thus, diabetic neuropathy is assessed with diabetes. Similarly, spinal neuropathies caused by herniated discs or osteoarthritis of the spine are part and parcel of these conditions. Nerve injuries are included in assessments of the gunshot wounds, fractures, and dislocations which caused them. (Values are given in the sections on the upper and lower limbs.)

The main factors in assessing decrease or loss of nerve function are: the effect on present employment (if under retirement age), the effect on activities of daily living, and any pain or discomfort in the affected part.

Basic Information

The Canadian Hong Kong Force in World War II consisted of two regiments- the Winnipeg Grenadiers and the Royal Rifles of Canada from Quebec. The Force arrived in Hong Kong on the 16th of November 1941; the first Japanese activity was an air attack on December 8, 1941, and the first Japanese land attack was repulsed on December 9, 1941. The Japanese finally landed in Hong Kong on December 19, and the surrender took place on December 25, 1941. Those soldiers who survived were held in captivity for forty-four months in Hong Kong or Japan.

Other prisoners of the Japanese included both military and civilian (mainly Merchant Marine) personnel captured at various intervals after the fall of Hong Kong. The exact number involved is not known. Members of this group were, in general, subjected to the same deprivation and indignities as the Hong Kong Force itself, though, in most cases, for shorter periods of time.

Statistics

Original Numbers of Force	1,975
Died of Wounds or While in Captivity	557
Survivors Returning to Canada in 1945	1,418
Deaths of Survivors up to 1966	135
Survivors Alive at the Time of Richardson's Study	1,283
All Surviving POW's of the Japanese of March 31, 1976	1,153

Richardson Study:

In December 1963, the Parliamentary Standing Committee on Veterans Affairs recommended that a special study and survey be made of the problems, and particularly the disabilities, of Hong Kong veterans. The Minister of Veterans Affairs Canada instructed the Canadian Pension Commission to undertake this study, the results of which are contained in "A Study and Survey of the Disabilities and Problems of Hong Kong Veterans 1964-65" which was authored by Dr. H.J. Richardson, the former Chief, Medical Advisor of the Commission. The various factors leading to the disabilities of prisoners-of-war of the Japanese as described below have been accepted.

Stresses of Captivity:

1. Diet - Grossly inadequate in calories and protein.
   - No nutritional balance.
   - Vitamin deficiency was universal, particularly vitamin B-Complex.
   - Loss of weight averaging 20%, but up to 40% in some instances.

2. Medicines - These were stored in prison compounds and were in short supply or not available and at times deliberately withheld by the Japanese.

3. Lack of Communication - Isolation was almost complete with no mail to or from families, no radios, no newspapers, or other sources of reliable news from the outside world and disturbing rumours were rife.

Stresses of Captivity:...CONTINUED

4. Forced labour was common.

5. There were cases of deliberate brutality and torture by the Japanese guards.

Organic Basis of disability:

Postmortem examinations revealed that there was an organic basis for residual disability resulting from Avitaminosis and dietary insufficiency. Demyelination of the posterior and anterior columns of the spinal cord was found especially in the thoracic area. Optic atrophy was also noted. These organic nerve changes were considered to be due largely to the lack of vitamin B-Complex.

Avitaminosis with Residual Effects:

This term has been used to identify the symptom-complex as preferable to other synonyms such as beriberi, pellagra, malnutrition-deprivation syndrome, etc.

Residual Disabilities Recognized as Related to
Avitaminosis and Dietary Insufficiency:

1. **General** - Easy fatigability, lack of stamina, non-specific musculo-skeletal complaints and impaired motivation. Usual assessment range 5% to 15%. Non-specific musculo-skeletal complaints are not necessarily related to the development of defined arthritic disease many years later.

2. **Ocular** - Permanent optic atrophy with non-progressive central scotomata, some peripheral field loss and increased susceptibility to glaucoma because of scar tissue are accepted. We have noticed a temporary weakness in the ocular muscles regarding the accommodation and the convergence, but this weakness had disappeared in 1946 and 1947. The permanent optic atrophy has been judged non-progressive when the normal feeding and the metabolism were reestablished.

   The degree of permanent optic atrophy was thought to be non-progressive once normal nutrition and metabolism were established. Deterioration of vision and of the optic atrophy from other causes may occur subsequently with an apparent progression of the optic atrophy in the recent past. Due to the difficulty in precisely identifying these other causes acceptance of some deterioration of the optic atrophy may be necessary in the assessment. Spectacles are provided by D.V.A. Treatment Services without charge.

3. **Neurological** - (Subjective and Objective) - Distressing paresthesia, weakness and atrophy of muscular tissue during captivity were followed by a paresthesia of feet and legs described as "hot feet". There was impairment of sensation of cold; with some cases of frost-bite resulting, requiring a number of sympathectomies. It was common practice to leave the feet uncovered at night. This type of symptom, commonly mentioned at first, is perhaps less severe now, but many pensioners still have the symptoms. The symptoms are at least as distressing as those of foot strain, are not relieved by medication or arch supports and disturb sleep.

Avitaminosis and Dietary Insufficiency: ...CONTINUED

3. **Neurological - (Subjective and Objective) ...CONTINUED**

Usual assessment range, 5% to 10%. Impairment of sensation and facility in the use of the hands has been documented by Dr. E.V. Kral and associates of Montreal. Other findings, disturbed balance and ataxia are taken into account when the assessment is being established. Both of these symptoms validate the concept of widespread neurological damage.

4. **Gastrointestinal** - In 1945 and 1946 there were many complaints of non-specific gastrointestinal symptoms. There were flatulence, surfeit, belching, morning anorexia and nausea affecting almost all P.O.W.'s of the Japanese. These non-specific symptoms not typical of any specific syndrome largely disappeared within a matter of weeks and months, and are not considered to have any significance in relationship to the later appearance of organic disease. They were common enough in this group of veterans to justify automatically accepting them as being related to incarceration by the Japanese. These symptoms have been assessed under the diagnosis of Avitaminosis with Residual Effects, often at 10%, as the equivalent to a symptomatic healed peptic ulcer.

Infectious hepatitis did occur during captivity but was not well documented because of lack of records. Any upper gastrointestinal symptoms which might be considered residuals of this disease would be assessed under this gastrointestinal component. Small and large bowel disease during service included amoebiasis, hookworm, and other minor infections, as well as irritations from inappropriate foods and physical-chemical contaminants. This has resulted in later years in the so-called "irritable bowel" and little else. Such persistent symptoms of "irritable bowel" represent a minor disability. Usual assessment range, 5% to 10%.

5. **Psychiatric** - Most, if not all, men returning from Japanese captivity took longer to re-adjust to civilian life than other veterans. This has been shown by more than usual difficulties in social and economic adjustments.

Impaired tolerance to the strains of daily life associated with anxiety, tension and depression are much commoner and more severe in these veterans than in any other large population of veterans. Corroboration of this point of view comes from a study of American and Australian prisoners-of-war both in Japan and Korea.

The neuropsychiatric disability is assessed without ruling in most cases under the diagnosis of Avitaminosis with Residual Effects, particularly where the diagnosis is in doubt, assessment is made in accordance with the Table of Disabilities for neuro-psychiatric conditions.

Avitaminosis and Dietary Insufficiency:...CONTINUED

6. **Vascular** - The majority of P.O.W.'s of the Japanese had shortness of breath, palpitation and chest pain on release from captivity, usually temporary but persisting in some cases without evidence of organic heart disease. The death rate from arteriosclerotic heart disease was excessive up to about 1960 to 1965, and since then has returned to normal. Non-specific persistent symptoms would be assessed, if found, under the diagnosis of avitaminosis with residual effects. Peripheral vascular symptoms in association with sympathetic over-activity resulted in some sympathectomies in the early post-discharge period. These symptoms overlapped with those of the neurological component and, when etiologically related to the neurological damage, are assessed with that component.

7. **Genito-Urinary** - It was initially thought that Avitaminosis had led to an excess incidence of ureteral colic during captivity but there has been to date no evidence of more than usual long-term disease of the renal tract.

   There was no evidence of sterility, but documentary, personal and hearsay evidence suggest an increased incidence of relative impotence in the fifth and sixth decades. This disability has been assessed in the 10% range as an addition to the neurological component.

8. **Dental Disease** - As this was very prevalent following captivity, all P.O.W.'S of the Japanese were granted full treatment rights for dental disease by D.V.A Treatment Services. Under these circumstances, an entitlement ruling for treatment purposes is unnecessary.

Post-Discharge Rulings:

Post-discharge conditions may be claimed as consequential to Avitaminosis with Residual Effects. When such rulings are favourable, the conditions are assessed according to the Table of Disabilities. The degree of relationship of a condition claimed as consequential upon Avitaminosis with Residual Effects varies inversely with the time interval between captivity and the onset of symptoms or signs diagnostic of the disease. Some such conditions appearing in the early post-discharge period have been pensioned in full as related to the effects of captivity. Other claimed conditions of recent origin with no relevant non-specific symptoms or continuity of complaints are not considered to have any relationship to the period of captivity.

Specific Areas of consequential Rulings:

1. **Ocular** - Glaucoma and cataract, when diagnosed, require ruling to determine whether and to what degree they may be related to Avitaminosis.

2. **Gastrointestinal** - Peptic ulcers are commonly ruled upon in P.O.W. 's of the Japanese. These were significantly more common than in any other large group of veterans known to have been studied.

3. **Osteoarthritis and disc disease** - Where there is continuity of musculo-skeletal discomfort related to specific joints or areas of the spine, consequential rulings for osteoarthritis of individual joints and for degenerative disease of the spine have been given.

Specific Areas of consequential Rulings: CONTINUED

4. **Arteriosclerosis** - Consequential rulings can be considered for arteriosclerosis relative to its three major manifestations - arteriosclerotic heart disease, cerebral vascular disease and peripheral vascular disease.

5. **Nervous Conditions** - Psychotic illness is the only area of nervous disease that requires a ruling, and this is necessary only when undoubted psychotic illness is present. All other nervous conditions are assessed without ruling under the psychiatric component of Avitaminosis with Residual Effects. As with all pensioners, a P.O.W. of the Japanese may claim for any other condition on a consequential basis which he considers is related to the Avitaminosis with Residual Effects. Such claims will be considered on their individual merits.

Pension Considerations

1. **Avitaminosis with Residual Effects** - Six factors are recognized as possible components of the disability related to this entitlement.

   They are as follows:

   - General
   - Ocular
   - Neurological
   - Gastrointestinal
   - Peripheral Vascular
   - Psychiatric

Taking into consideration the psychological trauma and multisystem symptomatology that may be quite non-specific for any organic illness but still considered related to the avitaminosis and dietary insufficiency suffered by prisoners of war of the Japanese: it is suggested that a minimum assessment of 50% for the avitaminosis would be appropriate.

When an assessment above 50% is considered appropriate, the specific factors identified should be listed on the VAC 865 and appropriate individual assessments. These, in turn, will be bracketed to show the total disability related to avitaminosis with residual effects.

Pension Considerations

Example #1

1. AVITAMINOSIS WITH RESIDUAL EFFECTS 50%
   Pens. W.W.II
   (VAC Initial 68.02.12)

Conditions accepted as consequential in whole or in part upon avitaminosis with residual effects are listed and assessed separately.

Example #2

1. AVITAMINOSIS WITH RESIDUAL EFFECTS

   Pens. W.W.II
   (VAC Initial 68.02.12)

   - General 20%)
   - Neurological 20%) 60%
   - Gastrointestinal 15%)
   - Ocular 5%)

2. DUODENAL ULCER
   Pens. 2/5ths conseq. re. #1 (2/5 x 20%) 8%
   (VAC Initial 69.03.09)

N.B Under the provisions of Bill C-100 which was proclaimed as law, 7 May 1986, the aggregate of all disability assessments for Hong Kong veterans plus compensation given to them under special P.O.W. legislation can now reach a maximum of 150% and will be paid at that rate.

Major types of disorders coming under purview of the psychiatric section include conditions which can be grouped under the following general headings:

1. Psychoneuroses (synonym: neuroses)
2. Functional Psychoses (the Schizophrenias, Paranoid Disorders and Major Affective Disorders)
3. Organic Brain Syndromes
4. Psychological Factors Affecting Physical Condition
5. Mental Retardation
6. Personality disorders
7. Substance Abuse Disorders

Of these only the first three groups (Psychoneuroses, Functional Psychoses and Organic Brain Syndromes) have direct relationship to the pension process. The remainder relate only insofar as they may interact with the three main groups.

Psychiatric conditions are commonly termed invisible illnesses because except during exacerbations they show no visible signs. Diagnosis thus is very dependent on carefully taken history, appropriate questions, the willingness of the person to divulge problems, the motivation of the subject and on a series of observations of maladaptive behaviours consistent with a pattern of Psychoneurosis, Functional or Organic Mental disorder.

Virtually all humans, from time to time or under special circumstances are subject to episodes of anxiety or its concomitants, of mood swings (elation and depression) or of brief delirium or confusion (toxic, traumatic, infectious events). Diagnosis of a psychiatric disability is made when such phenomena are continually or repeatedly present to a disruptive degree and are not merely transitory symptoms within the normal range of human reactions. That all humans are subject to brief occasional or persistent mild irrational thoughts, feelings and behaviours is a truism. "All the world is a little bit queer except thee and me, and sometimes I wonder about thee". Thus psychiatric conditions can be fairly conceptualized as exaggerations beyond the normal range of human emotional behavioral and thought aberrations, rather than as aberrations of a quite different order than experienced by all. The aberrations become diagnosable as psychiatric conditions when the quantity, intensity, duration or recurrences increase to the point of constituting a disability in significant areas of living.

Disabling psychiatric conditions may be abrupt or very insidious in onset. When insidious, dating the approximate time of onset of the illness must be done retrospectively through careful analysis of available history. During the early stages the person may be reticent about divulging problems, denying them and keeping them hidden, due to shame, guilt, or fear of being labelled insane, or because of lack of insight. On reviewing the history, careful differentiation must be made between symptoms and signs consistent with the eventual clear diagnosis and those which in retrospect were unrelated.

1. Psychoneuroses

For pension purposes, these conditions are characterised basically by continuing excessive anxiety and derivations thereof. Subjectively, anxiety is a sense of apprehension and foreboding over an impending or anticipated ill. Objectively, it includes physiological changes which are described in the following text.

Within limits anxiety and fear are universal characteristics of humanity. When appropriate for the situation, anxiety is a protective mechanism. When excessive, inappropriate, intensely recurrent or prolonged, it becomes a disabling condition and is termed psychoneurosis. A period of psychoneurosis may be followed by essentially complete recovery.

Anxiety, the Basic Neurosis

Effects which result from emotional stressors depend upon the genetic endowment and the person's life experiences from infancy onward.

Being subjected to vigorous playful tossing may be a delight to an agile infant, but distressing to a feeble one.

Competitive sports produce a eu-stress or dys-stress response in childhood and youth depending on innate abilities and past experiences. What one perceives as fun and challenge, the other sees as dangerous.

In all animals the alarm reaction (stress response) to a perceived threat (the stressor) is "fight or flight", in which a variety of body hormones are mobilized and physiological changes occur. These enable the animal in the natural state to escape or combat the threat. In milder form the alarm reaction may be simply hunger mobilising the animal to obtain food, thus restoring homeostasis. It is only in the trained or "civilized" animal or the human that nervousness or the anxiety reaction and dys-stress related physical illnesses occur.

In a severe alarm reaction as in the prey of the animal seeking food, body changes occur which are protective in the wild state but temporarily deleterious in the civilized state.

The body reactions include:

a) Hair standing on end. This makes the threatened animal look larger and is protective. The aggressor gets a bite of hair rather than of flesh.

   In the human, it gives only a creepy feeling.

b) Pupil of the eyes dilate. This increases the visual scope and improves low light vision. It is thus protective although sharpness is reduced.

   In the human, it usually just causes blurring of vision.

c) A surge of blood pressure shunts all available blood, and thus energy, to the muscles in preparation for fight or flight. In nature, the sole priority is immediate survival. Cool logic, digestion and carrying material in the mouth, stomach, intestines and bladder are not helpful. Blood goes to the muscles from the brain, the digestive tract, the bladder and the skin. The digestive tract and bladder contract to expel the excess baggage.

   In humans, the experience is of inability to think clearly, of nausea, vomiting and diarrhea, of involuntary micturition or "nervous pee" and of going pale.

d) The skeletal muscles obtain a large injection of blood and nutrients giving unusual strength and energy. In a primitive situation, the energy is expended mindlessly with no untoward effects. In the civilized state, such physical flight or fight is not available. The physical energy cannot be directly expended, leaving the muscles in an excited tremulous state.

e) With immediate survival, as fight or flight goes on, blood returns to the skin. Flushing and sweating occur to cool the overheated muscles. This is followed by return of blood to the brain and internal organs. With success, after a period of rest, full restitution occurs.

The reason anxiety is called a disease of civilization is that our animal nature and the demands of civilization call for opposite responses. As a comedian said, "my head tells me to stay but my feet tell me to go".

The child in class well prepared for a test accepts it with equanimity (eu-stress). The one who feels unprepared or incapable feels threat (alarm or dys-stress) with a fight-flight reaction. This gives gastro-intestinal upset, urinary urgency, difficulty focusing vision, reduced mental acuity and concentration, going pale, then flushing and sweating. Because of inability to put the skeletal muscles to their natural propensity for action, they show tremulousness.

All the above are the physiologically produced components of a situational acute anxiety reaction. Continuance or repetitiveness of anxiety-provoking events tends to lead either to generalized "free-floating" anxiety or to fears focused on specifics. Though time, the originating stressors are repressed or forgotten.

The person beset by anxiety of uncertain origin rather frantically seeks explanations or adopts avoidance behaviours to escape being in a state of ominous foreboding or fear. These attempts at restitution of equanimity result in the various behaviours and mental mechanisms that belong in the general diagnostic group of Neurosis or Psychoneurosis.

Common mental and behavioral adaptations by which the anxious person avoids the misery of continuous anxiety or panic attacks include:

a) Compulsions - excessively repetitive behaviours and rituals which are attempts to keep occupied and in control.

b) Obsessions - excessively repetitive intrusive thoughts or ideas which divert the mind from generalized anxiety.

c) Hysteria - either by conversion of general anxiety to functional disability of a body part, such as hysterical blindness or paralysis; or by dissociation to forget the fear, as by hysterical amnesia.

d) Phobias - focusing all anxiety on one subject thus freeing the person from generalized anxiety.

e) Post-Traumatic Stress Disorder - this may be acute and in some cases of limited duration. In other cases it may become chronic but be long delayed in being exposed. The "sine qua non" for diagnosis is the existence of a stressor, outside the range of usual human existence, of such severity that it would evoke significant

symptoms of distress in almost any previously normal person. Other essential features include (a) recurrent intrusive recollections, nightmares, feeling or acting as though the event were re-occurring; (b) decreased interest and detachment from others with restricted emotional responses; (c) Hyperalertness, insomnia, survivor guilt, difficulty concentrating the memory, avoidance of or upset in situations reminiscent of the traumatic event.

2. **Functional Psychoses**

For pension purposes, included in this category are major disturbances of function of thought processes and of mood (affect) in which probable structural or chemical changes in brain tissue are as yet of unknown or incompletely defined nature. Diagnostic types are the group of Schizophrenias, the major affective disorders and the Paranoid Disorders.

The essential features of the schizophrenic disorders are the presence of psychotic features (loss of contact with reality) of at least six months duration, accompanied by delusions, hallucinations and disorganization in the form of thought. There is a tendency to follow a deteriorating course. Even following a single schizophrenic break, it is generally accepted that some residual impairment remains permanently.

The essential feature of the major affective disorders is a pattern of severe pervasive disturbances of mood or affect with manic and/or depressive periods of disabling degree.

The essential feature of the paranoid disorders are persistent persecutory delusions or delusional jealousy, commonly associated with resentment, anger, grandiosity and social isolation.

3. **Organic Brain Syndromes**

For pension purposes the essential feature of these disorders is a psychological or behavioral abnormality associated with permanent dysfunction of the brain, due to structural deficits or disruptions of the neural substances. The clinical and laboratory findings in this group are extremely varied depending on the part or parts of the brain affected, the pre-existing personality, the occupational and social setting, and interactions with other disabilities. While the commonest causative condition is Alzheimer's Disease, more than fifty other causes have been identified.

4 **Psychological Factors Affecting Physical Condition**

Included in this category is a wide variety of afflictions commonly termed psycho-somatic, psycho-physiologic and so called "functional" medical disorders.

For pension purposes these disorders are so beset by contradictory, complex and uncertain scientific research findings mixed with a multitude of folk beliefs, and are so heterogeneous that no single theory or description can encompass all. Neither can a generalization be made that, because a cause and effect relationship may be established in one case, similar results are to be expected in other persons.

Medical consensus is that psychological factors play, at most, a minimal role in either causing or permanent worsening of psycho-somatic conditions.

Constitution, life-style,other physical conditions, and the many vicissitudes of living, have been clearly documented as fully accounting for the vast majority of the so-called psychosomatic conditions. However, it is generally accepted that when there is a close temporal connection between initiation or exacerbation of a psychiatric disorder and initiation or exacerbation of one of the psychosomatic disorders, that some degree of cause and effect relationship may well exist. It must be noted that despite the paucity of medical and scientific support the benefit of doubt has very frequently been invoked for the granting of entitlements in this respect. (See also articles on hypertension, arteriosclerosis and peptic ulceration.)

5. **Mental Retardation**

This term refers only to significant intellectual limitations resulting from hereditary, prenatal and perinatal influences. Intellectual loss occasioned by brain damage resulting from trauma, infection or other cause, during service life are dealt with as Organic Brain Syndromes.

Mental Retardation of mild to moderate degree, if not accompanied by physical stigmata, may well escape detection when circumstances are simple, familiar, repetitive and relaxed. However, it may predispose to development of or worsening of psychoneurosis or psychosis when the afflicted person is placed in a position of having to adapt to new situations, learn new techniques or to use initiative in situations beyond the ability to comprehend.

6. **Personality Disorders**

Every individual has unique enduring patterns of perceiving, relating to, and thinking about the environment and oneself. These derive from hereditary, constitutional and early environmental factors and are exhibited in a wide range of important social and personal contexts. Such unique characteristics of a person are termed "Personality Traits".

When "Personality Traits" are inflexible and maladaptive to the extent of causing repeated difficulties in inter-personal transactions they are then said to constitute "Personality Disorders". The manifestations of Personality Disorders are generally recognizable by adolescence or earlier and continue basically unchanged by environmental factors through much of adult life. However, they often become less obvious in middle or old age through slow emotional maturation or lessening of primitive energies.

Diagnosis of Personality Disorder is generally made as a result of cumulative observations of disciplinary or other disruptive interpersonal problems rather than of intra-personal behaviour problems. Thus it is rarely diagnosable on a single cursory examination. Symptoms which may accompany Personality Disorder are generally transitory and situational, such as when caught in a misdemeanour.

For pension purposes diagnoses of Personality Disorders, per se, have no direct bearing. However, such diagnoses, in retrospect, when followed by an acceptable diagnosis of psychiatric disability, may be viewed as a developmental phase of that disability.

7. Substance Abuse Disorders

For pension purposes Substance Abuse and disorders arising therefrom are considered with rare exceptions, to be voluntary acts not caused by any specific psychiatric disorder.

Use of certain substances to modify mood or behaviour under certain circumstances, generally termed recreational, is commonplace in our society. Most commonly used are alcohol and caffeine, but a wide variety of other drugs are used in this matter.

Use of small amounts of the common mood altering substances of recreation, stimulation or for control of anxiety and tension are not causative of permanent psychiatric disorders. Even occasional abuse is unlikely to lead to permanent deleterious mental or physical problems.

Abuse of mood altering substances, i.e. use of excessive amounts of alcohol or other drugs to the point of intoxication is not tension or anxiety relieving and is not used for these purposes. Rather such abuse is a form of pleasure seeking behaviour, often with untoward mental and physical effects, but unrelated to a pre-existing or concurrent psychiatric disability.

Substance abuse and dependency derive from the personality makeup, cultural, genetic, social and economic factors rather than from psychiatric disablement.

Exceptional consideration to the above may be granted in individual cases of severe persistent psychotic disorders and of Benign Essential Tremor, (hereditary). For the former, it is because of defective judgement. In the latter, it is because alcohol is very rapidly effective in abolishing the tremor, but so short lived in its effect that frequent dosage may be resorted to. Unfortunately, the overuse of alcohol eventually causes more tremor and a vicious cycle occurs.

Exposure of all living plants and animals to small amounts of ionizing radiation is a universal phenomenon with effects ranging from beneficial to negligible. Effects of larger amounts range from temporarily or locally harmful through all degrees of immediate or delayed adverse effects to rapidly catastrophic and fatal.

The effects of various levels of absorption of ionizing radiation on the body have been observed, measured and carefully documented in medical settings throughout the 20th century. Thus, the safe limits of exposure and absorption, both acute and cumulative, for known types and dosages of radiation have become clearly established. However, precision of measurement is not achievable under the uncontrolled conditions immediately following accidents or in nuclear warfare. In such circumstances assessment of effects must be based more on observed biological changes attributable rather than on theoretical radiation exposure.

The intensity of radiation energy from the electro-magnetic spectrum depends upon the frequency of the wavelength progression rate: - At the top of the list - (the shortest) - are the Gamma and Cosmic rays - coming down the spectrum we have: The x-rays; the ultraviolet waves; visible light waves; infrared waves; microwaves; and, radio-radar waves at the lower end of the spectrum.

The speed of propagation of the wave is constant in all frequencies; i.e.: 186,000 miles per second. - The energy involved, of vast importance to the human race, is inversely proportional to the wavelength: - i.e., a long radio wave having very little energy has no significant radiation effect on the human. The shorter the wavelength (from crest to crest) the more energy is involved: - (i.e., a boat at sea riding the crest of a wave with a long trough or length "goes with the flow" comfortable and with no damage; whereas, the same boat buffeted by multiple short waves of increased energy is shaken to pieces.) The more significant effect on a human being similarly comes from the high energy short waves.

Thus harmful effects of radiation are dependent on the duration and intensity of exposure, the types of radiations (x-rays, neutrons, gamma rays, alpha or beta particles) and the specific body tissues absorbing radiation.

A classification of the sequelae or harmful effects of radiation exposure may be considered as:

1. No effect or change in the tissue cell(s), in the case of low negligible exposure.

2. Immediate (instant to several days) changes:

    a) Mildly stimulating; transient only, leaving no sequelae.

    b) Varying degrees of cell damage from mild burning through 3rd degree burn phases with blistering and deep tissue damage. Such effects result in direct tissue cell breakdown and destruction, which is apparent and manifest within a short period of time with the death of the cell(s) resulting. There would be no long-term effect to be considered after the death of the part involved.

3. Long-Term Sequelae:

The effects of the radiation are not strong enough to destroy tissue cells but may affect the genetic or chromosomal makeup of the cell. Such an effect would be involved with the first reproduction of the tissue cells which varies for varying types of body tissues and systems. This genetic effect would somewhere down the road in an unknown and indeterminate period of time, become manifest as the number of "changed" cells become dominant; and this manifestation may take the form of:

a) A malignancy.

b) A benign form of change such as fibroid tumor or other tissue deformity which might not progress further.

The U.S. National Committee on Radiation Protection has set the maximum permissible radiation exposure for occupational exposed workers over age 18 years of 0.1 REM* per week for the whole body (but not to exceed 5 REM per year) and 1.5 REM per week for the hands. A routine chest x-ray would deliver 01.-0.2 REM.

Four hundred to six hundred RADS* of x-rays or gamma radiation applied to the entire body at one time may be fatal within 60 days. Levels of 1000 -3000 RADS to the entire body destroy intestinal mucosa leading to toxemia and death within 2 weeks. Total body dose above 3000 RADS cause widespread vascular damage, cerebral anoxia, hypotensive shock and death within 48 hours.

The clinical findings of acute ionization radiation effects on normal tissue may be considered as follows:

a) Injury to skin and mucous membranes: may result in erythemia, destruction of fingernails or epidermolysis.

b) Injury to deep structures:

1. **Hematopoietic Tissues:** Injury to bone marrow may cause diminished production of blood elements. Lymphocytes are most sensitive and erythrocytes least sensitive. Damage to blood forming organs may vary from transient depression to complete destruction.

2. **Cardiovascular:** Peri-carditis with effusion or constructive carditis may occur after months or years. Myocarditis is less common. Smaller blood vessels are more easily damaged than larger blood vessels.

3. **Gonads**: In males a single small dose of radiation (200-300 R) causes temporary aspermatogenesis and larger doses (600-800 R) may cause permanent sterility. In females a single dose of 200 R may cause temporary cessation of menses and 500-800 R may cause permanent castration. Moderate to heavy radiation of the embryo utero results in injury to the fetus or embryonic death and abortion.

4. **Respiratory Tract**: High or moderate doses of radiation may cause a temporary pneumonitis followed in weeks or months by an area of fibrositis.

5. **Salivary Glands**: May be depressed but large doses are required.

b) Injury to deep structures: CONTINUED

6. **Mouth, Pharynx, Esophagus and Stomach**: mucositis with edema and painful swallowing of food may develop within hours or days of exposure.

7. **Intestines**: Inflammation and ulceration may follow after moderately high doses.

8. **Endocrine Gland and Viscera**: Hepatitis and nephritis may be delayed effects of therapeutic radiation. The normal thyroid, pituitary, pancreas, adrenals and bladder are relatively resistant to low or moderate doses of radiation.

9. **Brain and Spinal Cord**: High doses cause damage due to impaired blood supply.

10. **Peripheral and Autonomic Nerves**: Are highly resistant to radiation.

c) Systematic Reaction (Radiation Sickness):

Anorexia, nausea, vomiting, weakness, exhaustion, lassitude and in some cases prostration may occur. With x-ray therapy it is most likely to occur when the therapy is given in large dosage to large areas over the abdomen, less often over the thorax and rare when given over the extremities.

The long term effects of radiation are difficult to evaluate because they take years to become apparent. Furthermore, it is difficult to differentiate effects presumed due to radiation from abnormal conditions known to occur spontaneously in the population at large.

Skin scarring, atrophy and telangiectasis, endarteritis, pericarditis, pulmonary fibrosis, hepatitis and intestinal stenosis, nephritis and other late effects are known to occur.

The incidence of neoplastic disease including leukemia is increased in persons exposed to excessive radiation. The latency period between exposure and development of cancer may be thirty years or more.

Pension Considerations

Canadian military personnel have readily assisted their country in peace and war related nuclear events. Most notable have been their participation in two major clean-up operations following accidents at the Chalk River Nuclear Laboratories (CRNL), (1953 and 1958), and in observation of the atomic bomb tests blasts in Nevada (1955 and 1957) and Australia (1956 and 1957). Some of these participants have developed serious health problems and several have died.

In early March 1982 the Department of Epidemiology and Community Medicine of the University of Ottawa was called upon by the Department of Veterans Affairs to conduct an investigation of the effects of exposure to radiation among these Canadian veterans. The results of this study became available August 1984.

This study failed to detect any increase in mortality or in the occurrence of premature mortality among the Canadian military personnel who participated in the CRNL clean-up or in the atomic tests blasts in the U.S. or Australia. The general mortality experience among the exposed was quite similar to a central group who were of the same age, military service and rank and engaged in similar trades at the time the events under study occurred.

Pension Considerations CONTINUED

There was no elevation in case - specific mortality for those cases which are known to be associated with radiation exposure. In particular, mortality from cancer was very similar among the exposed and control groups. The main follow up period for the survivors among the exposed and central cohorts was about 26 years. This latency period was felt by the study group to be adequate for most of the known radiation induced malignancies to be manifest.

This study, however, deals only with mortality and no conclusion could be reached about current morbidity due to cancer or non-fatal illnesses in exposed individuals.

Radiation claims must each be assessed individually and on their own merit taking into consideration results of the above study as well as known acute and long term sequelae of ionizing radiation as contained in the medical literature.

* In radiation terminology, a rad is the unit of absorbed dose and a rem is the unit of any radiation dose to body tissue in terms of its estimated biologic effect. Roentgen (R) refers to the amount of radiation dose delivered to the body. For x-ray or gamma ray radiation, rems, rads, and roentgens are virtually the same. For particular radiation from radioactive materials, these terms may differ greatly (e.g. for neutrons, 1 rad = 10 rems). In the Systeme Internationale (SI) nomenclature, the rad has been replaced by the gray (Gy), and 1 rad + 0.01 Gy. The SI replacement for the rem is the Sievert (Sv), and 1 rem = 0.01 Sv.

Chronic Bronchitis

This is an irritant, inflammatory lung condition characterized by recurrent cough, excessive expectoration of mucus and some shortness of breath. The American Thoracic Society specifies that objective signs should be present on most days for at least three months of the year during two or more consecutive years in order to be designated as "chronic", i.e. bearing a degree of permanent disability. Rhonchi, wheezing, and even rales can be heard over the lungs. Air flow may be obstructed in Chronic Bronchitis.

Causes of chronic bronchitis may include smoking, air pollution, and dusty-laden occupations. Climatic conditions should be considered insignificant.

Acute Bronchitis (or plain "bronchitis"), Subacute Bronchitis, Recurrent Bronchitis, "Chest Colds", Pneumonia

These acute lung conditions are ordinarily self-limiting leaving no permanent disability.

Upper Respiratory Infections, Common Cold (Acute) Coryza, "head cold",Rhinitis, Pharyngitis, Laryngitis

These are conditions affecting parts of the respiratory system located at and above the larynx and its vocal cords. These are often claimed as consequential relationships in which a chronic lung condition is claimed to be related to an upper respiratory condition. These will be mentioned under "Consequential Relationships".

Tracheitis, Pharyngo-tracheitis, and Tracheo-bronchitis

The trachea is the first part, and the widest conducting airway just below the larynx. Tracheitis is usually an acute disease characterized by cough without sputum and by upper substernal chest pain.

Asthma

This is an obstructive lung condition characterized by wheezing caused by the narrowing of airways. A history of childhood or young age respiratory difficulty, variable shortness of breath on exertion, possible allergens, attacks of wheezing with relief from bronchodilators, a positive family history, attacks precipitated by infection, and normal pulmonary function tests between attacks are characteristic. Obstruction of airflow is obvious on pulmonary function testing during attacks but may also be present between attacks.

Bronchiectasis

This is a permanent abnormal dilation of airways causing them to serve as a reservoir of often very abundant and stagnant secretions, at times with bloody streaks, pus or thick blood. The dilatation usually occurs because of weakened bronchial walls, but it can be congenital. A bronchogram substantiates the diagnosis.

Pulmonary Emphysema

This is a chronic lung condition wherein the efficiency of gas exchange (oxygen in, carbon dioxide out) is reduced due to destruction of alveolar walls. Alveoli are tiny air sacs, walled by delicate membranes, at the ends of the air passages in the lung tissue. Here oxygen is passed to the blood and carbon dioxide expelled. When the walls separating alveoli break, the total membrane area for gas exchange is reduced and lung efficiency decreased.

Due to the loss of a multitude of membranous walls, chest x-rays show a characteristic hyperlucency. The emphysema may be generalized or localized.

There are several disease conditions in which emphysema is a characteristic feature. The effect of excessive bronchial secretions in such conditions as Chronic Bronchitis and Chronic Obstructive Lung Disease is to form one way valves in bronchioles thus trapping air and promoting hyperinflation, with stretching and braking of alveolar walls. The main causes of these conditions are inhalation of industrial irritants , other environmental pollutants, and tobacco smoke.

An additional known cause in some cases is the inborn deficiency in the tissues of an inhibitor (alph-1-anti-trypsin). This deficiency allows body enzymes to disintegrate alveolar walls.

Chronic Obstructive Lung Disease

Chronic obstructive lung disease (C.O.L.D) represents an advanced stage of several lung diseases such as chronic bronchitis and bronchial asthma, with persistent hyperinflation. Damaged lung tissue (parenchyma) loses the elastic recoil for lung deflation.

Respiratory failure is a condition documented by low blood oxygen and accumulated carbon dioxide in the blood as a result of the failure of the respiratory system to be able to adequately compensate for any load factors placed upon it.

Chronic Restrictive Lung Disease, Diffuse Interstitial Lung Disease,
Idiopathic Pulmonary Fibrosis

Diseases belonging to this group show a stiff lung as a result of fibrous tissue around the alveolar walls, thus limiting the ability of the lung tissue to expand. The blood flow through the capillaries of the alveolar wall is decreased because of lost lung tissue, and from compression of blood vessels by fibrous tissue. shortness of breath on exertion and in severe cases at rest, crackling fine rales heard over the lungs, and x-ray showing extensive reticular (honeycomb-like) on nodular pattern, or a mixture of the two elements, lead to the diagnosis. Causes are frequently unknown; however, micro-organisms, mineral dusts, allergic reactions to allergens, drug reactions, collagen diseases, therapeutic radiation, could all be proven to represent known etiologies. Note that the restrictive features of pulmonary tuberculosis (T.B.) or extensive pleural adhesions do not put T.B. into this group.

a) **Carcinoma of the Lung, Bronchogenic Carcinoma**

As it will appear under pension considerations, the microscopic pathology has some bearing on the policies presently in use. Three standard histo-pathologic types of primary bronchogenic cancers are considered:

1. Small cell carcinoma, (one subtype being highly undifferentiated "oat cell" carcinoma).

2. Adenocarcinoma (large cell carcinoma) from mucin secreting bronchial glands.

3. Squamous cell or epidermoid carcinoma originating from the bronchial epithelium.

b) **Mesothelioma, and Asbestosis-related Bronchogenic Carcinoma**

Mesothelioma is in fact a rare tumor of the pleura that is caused by exposure to asbestos dust. Decades later the tumor originates from the pleura and by proximity extends into lung tissue as well. There is an increased incidence of carcinoma of the lung in asbestos workers who are also cigarette smokers.

Pension Considerations:

Disabilities that are service-related and permanent in nature are pensionable under the insurance or the compensation principle. Established pre-enlistment conditions are pensionable for the part of the disability that is service related.

Acute diseases - such as acute, subacute, recurrent bronchitis, "chest colds", upper respiratory infections, and pneumonia would only become pensionable when the expected self-limiting pattern is disturbed by a complication which results in a long-lasting disability. Theoretically such self-limiting respiratory conditions which occur during service and/or on duty might result in entitlement; however, because complete recovery occurs from such condition, the assessment would be nil even if the condition recurs.

Chronic obstructive lung disease (C.O.L.D.) develops through a common path from a number of the already mentioned lung conditions. The worsening of the lung conditions into C.O.L.D., whether caused by smoking or by occupational irritants, is part of its natural history. The assessment is that of the lung condition as a whole.

In pensioned lung conditions such as Chronic Bronchitis or Asthma, shortness of breath may be taken as representing a measure of the disability present. However, shortness of breath, may also be linked to a separate heart condition and in such a case, it could not be taken as representing a measure of a pensionable lung disability.

In the same fashion, a person may be afflicted with both Chronic Obstructive Lung Disease and a Restrictive Lung Disease. Shortness of breath may be a symptom of both. It the Chronic Obstructive Lung Disease only is pensioned, it is necessary to attempt to distinguish the amount of disability arising from each of the conditions and to then give an assessment only for the Chronic Obstructive Lung Disease in the given example. This may be aided by such as pulmonary functions tests.

Pension Considerations in Some Consequential Relationships

Consequential claims are frequently seen involving lung conditions. The lung disease in some cases is claimed to cause or worsen another disease. The other disease therefore could be claimed as fully or partly consequential upon the lung condition.

In another group of cases, a pensioned disease may be claimed to cause or worsen a lung condition. A few examples should illustrate the above-mentioned two groups. Here the lung disease may be claimed as fully or partly consequential to the primary pensioned condition.

1. Longstanding chronic bronchitis or C.O.L.D. is considered by some physicians to play a partial role in subsequent cancer of the lung (see remarks on carcinoma of the lung). For such partial alleged relationship with adenocarcinoma only a minimal award is recommended at the present time.

3. Pulmonary tuberculosis (T.B.) may cause a significant change in the normal architecture of the lung tissue, promoting infectious bronchial secretions. Therefore, T.B. could promote the development of chronic bronchitis. Obviously other conditions, such as excessive smoking and industrial pollutants, may play a role. A minimal consequential award would represent this relationship.

3. Chronic sinusitis, often as accompanied by postnasal drip, is considered by a number of specialists in the field to provide infective aerosol which is aspirated into the airways. A one-fifth consequential relationship is recognized at this time for chronic bronchitis subsequent to pensioned chronic sinusitis.

4. Frequently a consequential claim is made for pensioned chronic bronchitis aggravating symptoms of an ischemic coronary artery disease. Both diseases represent a state of poor health. Both may cause a greater total cardio-pulmonary distress. However, present knowledge does not indicate that either causes or worsens the other physical condition.

MEDICAL GUIDELINES **SEIZURE DISORDERS (EPILEPSY)**

Epilepsy is an intermittent derangement of the nervous system resulting in an almost instantaneous disturbance of sensation, loss of consciousness or psychic function convulsive movements, or some combination thereof.

The term epilepsy is used interchangeably with the term "seizure disorder".

An isolated seizure may occur during the course of many medical illness and does not necessarily imply that a "seizure disorder" is present. Fever, certain drugs, drug withdrawal, hypoglycemia and other acute metabolic disturbances may all result in a seizure. The term "seizure disorder" applies when seizures are recurrent over a significant period of time (months, years) and are caused by a primary disorder of the nervous system rather than by the effects of non-neurological medical conditions.

Epilepsy may be primary (idiopathic), in which case no underlying cause is identified and one is left to postulate that the underlying cause is a scar resulting from disease which went unnoticed, or occurred in utero, at birth, or in infancy in parts of the brain too immature to manifest signs. Seizures may also be secondary to head trauma, cerebrovascular disease, brain tumour, or alcoholism.

The principal clinical types of epilepsy encountered are:

- Generalized grand mal seizures
- Petit mal seizures ("absences")
- Partial (localized) motor seizures (Jacksonian)
- Temporal lobe seizures (Psychomotor epilepsy)

Pension Considerations

It is of prime import to ascertain that the individual is indeed suffering from a seizure disorder, as opposed to an isolated seizure, pseudoseizures, hysterical seizures, simple syncope ("fainting spells") transient ischemic attacks or one of the migraine variants.

In cases of idiopathic epilepsy, although the underlying lesion, likely a cerebral scar, dates from birth or infancy, the onset of epilepsy is deemed to be the date of the first documented seizure.

Post traumatic epilepsy deserves special consideration; this diagnosis is made when there has been a head injury of sufficient severity so as to result in an ongoing seizure disorder. This may occur with or without a skull fracture, but head injuries with skull fracture are 20 times as likely to result in seizure disorder than are head injuries without skull fracture. The usual interval between the brain injury and the first seizure is 9 to 15 months, but it may be as brief as a few months or as long as several years. In an individual who has had a head injury and who at some later time develops epilepsy, the decision as to whether or not the two are related must be based on a careful clinical consideration of the facts and, in the end, is a question of medical judgement; obviously, the severity and nature of the initial head injury, the time relationships, and the characteristics of the seizure disorder must be carefully weighed.

MEDICAL GUIDELINES **SEXUALLY TRANSMITTED DISEASES**

S.T.D.'s are those infectious diseases which are usually transmitted from one human to another, principally, but not exclusively, by means of sexual intercourse or other similar sexual contact.

Discussion

The list of S.T.D.'s shall include Gonorrhoea, Syphilis, Chlamidia, Trichomoniasis, Haemophilus Vaginitis, AIDS, Herpes Simplex Type II, and a number of other infectious, but less common diseases.

AIDS in particular may also be transmitted by the use of contaminated parenteral injection (I.M. or I.V.) equipment, or by contact with infected blood or genital secretions. In the latter case, the chief risk is if the person's own blood stream comes in contact with the infected material, e.g. in receiving a blood transfusion.

With regard to Regular Force Service, duty factors would not normally lead to the transmission of these diseases. Exceptions to this could occur in the case of AIDS, where a service person, for example, in a medical role, could be shown to have come into hazardous contact with such secretions, in the performance of duty.

Regarding the above, it should be noted that AIDS is an exception to the S.T.D.'s, in that the other S.T.D.'s are not usually transmitted via the blood route.

MEDICAL GUIDELINES **STRESS**

A. **Introduction**

The word "stress" is commonly invoked as causative of a wide variety of illnesses as though it were a well understood, specific and noxious agent. In fact, even after some 50 years of research and innumerable studies both the concept and the relationship to disease, for the most part remain unproven. Scientific consensus of the validity of relationship to disease causation has been reached in only a few very circumscribed circumstances using scientific (not popular) definitions.

As will be noted below the primary cause of the failure of this very complex concept to be clarified has been due to the use of a common word with opposing meanings (stress = cause; stress = effect) for one intended precise scientific meaning (stress = effect).

The unfortunate result of the imprecision of basic definitions is that what is accepted as an important concept of health and disease remains vague, controversial and of less value than it probably deserves in scientific medicine.

Following are excerpts of Selye's definitions and a commentary on problems of relating the "stress" concept to disease processes.

B. Forty Years of Stress Research: Principal Problems and Misconceptions, H. Selye, CMA Journal, July 3, 1976, Volume 115.

Definitions from Selye:

Stress is the non-specific response of the body to any demand. It includes Eustress and Distress. (For clarity these are hereafter termed Eu-stress and Dys-stress.)

A Stressor is an agent that produces stress at anytime.

The General Adaptation Syndrome (G.A.S.) represents the chronological development of the response to stressors when their action is prolonged. It consists of three phases; the alarm reaction, the stage of resistance and the stage of exhaustion and affects the whole body directly or indirectly.

The Local Adaptation Syndrome (L.A.S.) is the response to non-specific demands made upon only one part of the body.

Eu-stress is that form that is beneficial to the organism.

Dys-stress is that form which appears as maladaptation - fatigue, exhaustion and unproductive or destructive body and mind reactions.

C. **Commentary**

Webster's Dictionary gives two definitions which destroy Selye's attempt at a single precise definition. It defines "stress" as either: (a) a physical, chemical or emotional factor that causes bodily or mental tension and may be a factor in disease causation. (This by Selye is a "stressor"; i.e., a cause); (b) a state resulting from a "stress", especially one of bodily or mental tension resulting from factors that tend to alter an existing equilibrium. (This by Selye is "stress"; i.e., the response, which he divides into Eu-stress and Dys-stress.)

A number of commentators on the subject have suggested that had Selye used the word "stress" only for "eu-stress" and the word "strain" in place of "dys-stress" the concept would be more readily understandable.

The principles of the "stress reaction" are similar for all living organisms including the human.

A plant develops extended roots, stalk, branches and leaf attachments through exposure to stressors, mainly variances in moisture, nutrients, temperature, light and wind. It is only when the environmental variances become extreme beyond the genetic capability of the plant to adapt that dys-stress (or strain) occurs.

For example, reduced moisture (the stressor or agent) stimulates the plant to extend its roots deeper (the stress response or alarm reaction) until sufficient moisture is obtained (adaptation). It is only when the inherent genetic endowment of the plant for root extension to obtain water is exceeded that the stage of exhaustion and maladaption (disease) occurs.

A human or other animal develops growth, energy and vigor through exposure to a stressor, mainly low blood sugar. The response (stress) is hunger which stimulates the animal to obtain the food required for health (adaptation).

If for any of a number of reasons, physical, chemical or emotional, the animal or person does not get the eu-stress response (appetite, hunger) to the stressor, or is deprived of food, then the response will become dys-stress with strain on body functions (maladaptation).

Temporary fasting or starvation,whether imposed or voluntary has no permanent deleterious effect and may be beneficial, as when excess energy stored as fat is utilized. It is only when the limits of adaptation are braced to the point of exhaustion of body resources that on-going pathology may result.

The degree of stress response (eu-stress or dys-stress) to a given stressor depends upon a wide variety of constitutional, personality and total life experience factors unique for each person. Thus, it is wrong to generalize that a given stressor or set of stressors will cause similar reactions in different individuals. Only extreme stressors outside the range of usual human experience are likely to cause the same stress response (dys-stress) throughout a given population.

D. **The Relationship of Stressors and Stress Reactions to Disease**

In the present state of knowledge specific correlations between Stressors, Stress Reactions and mental or physical disease in a human are at least as difficult to delineate and as fraught with variables as the making of correlations between climatic conditions (sun, rain, heat, cold, soil types, pollutants, etc.) and plant or animal stress reactions and diseases. A further parallel can be drawn between the difficulties of making a long term prognosis from an acute body reaction and long term weather forecasting from a down pour of rain.

In the human the problem is further complicated because in addition to physical, chemical, constitutional and genetic factors, there are emotional factors which are unquantifiable but present in everyone as unique personality characteristics.

The cumulative evidence in scientific literature does point to some personality traits as predisposing some persons to some physical and mental diseases, but no specific causal relationships have been established.

No scientific evidence has established that any specific psychiatric disability is causative of any specific physical disability.

Thus, while stressors may cause a stress reaction, which if the stressors are extreme or of long duration, leads to the development of a disease in a given individual, this disease does not cause other stress related conditions to develop.

For example, there is no evidence that anxiety (or psychoneurosis) causes hypertension or causes peptic ulcer. Dependent upon a wide variety of genetic constitutional and environmental factors, two or more psychiatric and physical disorders frequently co-exist. While they may have one partial common root (the stressor) the relationship between two such disorders is parallel, not cause and effect.