

11-1940

UWOMJ Volume 11, No 1, November 1940

Western University

Follow this and additional works at: <https://ir.lib.uwo.ca/uwomj>



Part of the [Medicine and Health Sciences Commons](#)

Recommended Citation

Western University, "UWOMJ Volume 11, No 1, November 1940" (1940). *University of Western Ontario Medical Journal*. 95.
<https://ir.lib.uwo.ca/uwomj/95>

This Book is brought to you for free and open access by the Digitized Special Collections at Scholarship@Western. It has been accepted for inclusion in University of Western Ontario Medical Journal by an authorized administrator of Scholarship@Western. For more information, please contact tadam@uwo.ca, wlsadmin@uwo.ca.

11-12
1940-42

38

VOLUME XI.

NUMBER 4

UNIVERSITY OF WESTERN ONTARIO

MEDICAL
JOURNAL



VOLUME XI.

Published by the

University of Western Ontario Medical School

Undergraduate Body of the

LONDON - ONTARIO

University of Western Ontario
MEDICAL JOURNAL

Published Quarterly by
Undergraduate Body of the
University of Western Ontario Medical School
Entered as a Second Class Matter at London, Canada.



BOARD OF ADVISORS

E. M. WATSON, M.D., F.R.C.P. (Edinburgh)
R. A. JOHNSTON, M.D., F.A.C.S., F.R.C.S. (C.)
S. A. MACDONALD, B.A., M.B., Ch.B.

STAFF

JOHN D. ATCHESON, '41, *Honorary Editor*
LAWRENCE D. RUTTLE, '42, *Editor-in-Chief*

Associate Editors

RAYMOND STUBBING, '42, *Abstract Editor*
ALAN DOUGLAS, '42, *Book Review Editor*

Business Manager
JOHN LINDSAY, '43

Advertising Manager
HENRY H. LEE, '43

Circulation Manager
JOHN DOUGLAS, '44

Assistant Advertising Manager
DOUGLAS BOCKING, '44

Subscription Manager
DOUGLAS LAKE, '44

Secretary-Treasurer
STUART M. BUSBY, '44

28150

University of Western Ontario
MEDICAL JOURNAL

Volume 11 — No. 1

November, 1940

CONTENTS

	Page
Chronic Non-Tuberculous Basal Infections of the Lungs <i>W. E. Pace, '41</i>	1
The Carotid Sinus in Disease <i>Max Nareff, '42</i>	7
Hare-Lip and Cleft Palate <i>D. J. C. Miller, '38</i>	14
Sleep <i>R. Brian Holmes, '43</i>	20
Elliptical Cell Poikilocytosis <i>H. W. Boyes, '42</i>	26
Abstracts	30
Editorial	33
Book Reviews	34
Recent Accessions to the Medical School Library.....	37

SEDALEX

(TRADE NAME)

Each fluid ounce contains:—

Ammonium Chloride	- - - - -	16 grs.
Ammonium Carbonate	- - - - -	16 grs.
Sodium Citrate	- - - - -	16 grs.
Diacetylmorphine HCl.	- - - - -	$\frac{1}{3}$ gr.
Menthol	- - - - -	$\frac{1}{4}$ gr.
Chloroform	- - - - -	3 min.
Eucalyptol	- - - - -	1 min.

INDICATIONS—Sedative, Alkaline, Expectorant.

DIRECTIONS—One or two teaspoonfuls as directed by Physician.

Charles R. Will & Company

LIMITED

ETHICAL PHARMACEUTICAL PRODUCTS

London

Canada

Samples to the Medical Profession on request



How much will it cost you to borrow \$100 at the Bank of Montreal, if you are able and willing to repay the money in 12 equal monthly instalments? Just \$3.65. There's no other charge.

Consult our nearest branch manager.

BANK OF MONTREAL

ESTABLISHED 1817

"A bank where small accounts are welcome"

IMPERIAL LOYALIST Furniture

for

Doctors' Waiting Rooms

Hospital Sun Rooms

Visitors' Sitting Rooms

Staff Sitting Rooms



*The Furniture which gives atmosphere
and distinction to any room.*



Made by

Imperial Rattan Co. Limited

Stratford - Ontario

The University of Western Ontario
MEDICAL JOURNAL

VOLUME 11

NUMBER 1

**Chronic Non-Tuberculous Basal
Infections of the Lungs**

By W. E. PACE, '41.

CHRONIC cough, with expectoration, is a symptom which is frequently disregarded in the examination of a patient because its prognostic significance is not generally appreciated. This symptom often indicates the presence of a low-grade infection in the lung bases which is the forerunner of clinical bronchiectasis.

In this paper an attempt is made to describe a recognized group of cases in which chronic cough, with expectoration, is the chief complaint. These cases are further characterized by the presence of constant physical signs which are confined to the lung bases, and by the absence of the constitutional symptoms which are found in pulmonary tuberculosis. Notice was attracted to a similar type of patient in 1902, by Lord, who described 18 cases of a chronic infection of the lungs which followed a relapsing course. Since then, Hamman, Miller, Austrian, Cherry and others have described related series of cases. The term chronic non-tuberculous basal infections of the lungs used in this paper is taken from Hamman.

The clinical course of this condition is characteristic. The symptoms usually begin in childhood or early adult life but these patients most often present themselves for diagnosis between the ages of 20 and 40 years. Cough, with expectoration, is their chief complaint. It may start insidiously, but frequently occurs after a definite respiratory infection such as a severe "cold", an attack of "grippe", influenza, or pneumonia. In children, it may follow one of the infectious diseases, especially measles or pertussis.

Cough and sputum increase as the patient becomes older, and there is a definite susceptibility to mild upper respiratory infections. Remissions and exacerbations occur with changes in temperature. The symptoms increase during the cold weather but become less prominent or disappear during the summer months.

In most cases the cough and expectoration persist for many years

with remarkably little impairment in the general health; other cases go on to develop a frank bronchiectasis.

THE SYMPTOMS

Cough with expectoration is the outstanding symptom. This cough is usually intermittent and is most marked in the morning on arising, or after a change of position. It varies from time to time in intensity and character; at times, it is loose and productive, at other times almost dry.

The amount and character of the sputum also varies; often, it is scanty, tenacious, and mucoid, but during exacerbations it may become profuse and mucopurulent. The mucus is often raised in solid lumps. In amount, it varies from one drachm to four or five ounces a day. It is never raised in large mouthfuls, nor is it foul-smelling as in bronchiectasis.

Haemoptysis is relatively common. It occurs in about 50 per cent of the cases, usually as blood-streaked sputum; but frank haemorrhages are not rare. Because of blood in the sputum, many cases are incorrectly diagnosed as pulmonary tuberculosis and are sent to sanatoria where they sometimes remain for years.

The constitutional symptoms are mild, in spite of the long history. The general health is only slightly impaired. The body weight is well-maintained and there is never the fatigue which is present in tuberculosis. A low fever may occur and may persist for long periods of time, but the afternoon temperature is not above 100° F.

PHYSICAL SIGNS

Inspection and palpation of the chest usually disclose no significant variations from the normal. On percussion, an area of impaired resonance may be found over the base of the affected lung posteriorly. However, this is rarely marked. On auscultation, there may be some alteration in the breath sounds. These may be diminished, or they may be harsher than normal, rarely bronchovesicular in character.

The one characteristic finding is the presence of localized moist râles over the affected lung base. The râles may be scanty or profuse, changing markedly from time to time with variation in the amount of coughing and expectoration. They are best elicited by having the patient cough. The râles are often localized to one particular spot, so that thorough examination of the whole basal area is essential. They may disappear entirely during periods of remission, only to recur in the same area during the next respiratory infection.

ROENTGENOLOGICAL FINDINGS

Radiologists have not yet agreed upon the radiographic characteristics which are diagnostic of chronic basal disease. However, practically

all investigators do agree that the commonest finding in these cases is a generalized increase in the density of the pulmonary markings, which is most prominent in the lower half of the lung fields. By itself, this finding is not of much diagnostic importance, but, if it is accompanied by one or both of the abnormalities mentioned below, it may offer presumptive evidence of the existence of chronic basal disease.

Areas of chronic pneumonia or pneumonitis may be seen in these cases; the shadows appearing as flocculent patches in the lower half of one or both lung fields. Sometimes, they are represented by a mottling along the course of the basal trunks.

Displacement of the heart and mediastinum toward the diseased area, which is usually on the left, occurs in a good percentage of cases but must be looked for carefully if slight degrees of the condition are not to be missed. This shifting is thought to be due to atelectasis in the affected lobe which results when the bronchioles are plugged with secretion. When both lower lobes are affected, the pulling forces counteract each other and displacement may not be present.

Since the introduction of lipiodol in 1921, bronchography has shown that many cases possess bronchial dilatations of varying degree and that they are, in fact, mild cases of bronchiectasis. The importance of this finding will be considered below. Lipiodol injection cannot be made routinely, but when two or more of the above findings are present in the flat plate of the chest and are supported by appropriate history and physical findings, bronchography should be done.

DIFFERENTIAL DIAGNOSIS

Pulmonary tuberculosis must be definitely excluded. Cases of chronic basal disease do not show the marked constitutional symptoms such as loss of weight, increasing fatigue and night sweats which are associated with tuberculosis. The physical signs are localized to the basal areas in contrast to the apical involvement so common in tuberculosis. No tubercle bacilli are found in the sputum. It must be stressed that differentiation on the basis of clinical findings alone is not reliable; therefore, X-ray examination must be made in every case if tragic errors are to be avoided.

Chronic bronchitis is easier to differentiate. The symptoms of this condition do not usually appear until middle age and are often manifestations of more serious affections. Haemoptysis does not occur; fever is absent, but dyspnoea is common. On inspection, the chest shows a variable degree of emphysema and, on auscultation, moist râles or rhonchi are heard throughout both lungs. X-ray examination may show an increase in the pulmonary markings, but there is no displacement of the mediastinum, nor areas of chronic pneumonia.

Tumours of the bronchi and lungs may produce physical signs identical with basal infections of the lungs. However, the symptoms are not of such long standing, and there is gradual or rapid impairment of the general health. The cough is usually harsh and occurs in paroxysms. X-ray examination may show a rounded uniform shadow, usually in the mediastinal region, which represents the tumour mass. Atelectasis is often extensive and may cause gross displacement of the mediastinum.

Lung abscess should not be confusing. There is usually a history of sudden onset, often after a surgical operation, with chills, fever, and profuse sweats. X-ray of the chest shows the presence of a cavity with a fluid level, surrounded by pneumonic consolidation.

PATHOLOGY

It might be of value to discuss the present concept of the underlying pathology in chronic basal pulmonary infections. It has been shown by many investigators that this condition is not a specific disease due to the activity of one micro-organism. Different organisms are found in different cases and from time to time, in the same case. The use of lipiodol in bronchography, as mentioned above, first showed that in at least some of these cases bronchial dilatation existed. This finding suggested that chronic basal disease was, in reality, a mild or early form of bronchiectasis. More proof of this has occurred with wider use of lipiodol in recent years.

If the symptoms of chronic non-tuberculous basal infections are now examined in this new light, it is found that they fit in remarkably well with those of bronchiectasis. There is the frequent onset in childhood, the history of chronic cough and expectoration, often following an acute respiratory infection; the long duration of symptoms with slight constitutional reaction; the tendency to recurrent hemoptysis; the character and strict localization of the physical signs, and the absence of pulmonary infiltration in the roentgenogram. Added to these is the fact that not a few cases go on to develop a frank bronchiectasis, and that an occasional autopsy has demonstrated the presence of dilated bronchi.

It must be made clear that the above concept of chronic basal disease is not presented as a proven fact. Much work must be done before this view can be fully accepted, but as a working hypothesis it is of assistance in the interpretation of individual cases.

PROPHYLAXIS

It appears that many basal infections of the lungs have their beginning during childhood, even although symptoms do not appear until much later. Thus, if this type of disease is to be prevented, a

more serious attitude must be adopted with regard to acute respiratory infections in children. All colds must receive prompt attention and patients must not be allowed out of bed until the cough has disappeared. If the patient shows susceptibility to repeated upper respiratory infections, every effort must be made to build up his general health by a well-regulated, hygienic life so that the vicious circle of repeated infection may be broken. Measles and pertussis, as well as pneumonia, must be regarded as predisposing to chronic basal infections and convalescence from these infections carefully supervised.

TREATMENT

The treatment of established basal infections of the lungs is often very unsatisfactory. If dilated bronchi are already present these cannot be made to assume normal contour again. Since these infections do not threaten life, the main problem in treatment is to alleviate annoying symptoms.

General Measures: A sane manner of living is essential if the patients are to ward off the development of a frank bronchiectasis. They should have adequate rest, fresh air, and a well-balanced diet. When possible, their removal to a warm, dry climate during the winter months is a distinct advantage. Every effort should be made to avoid exposure to "the common cold". A chronic infection in some part of the upper respiratory tract is usually present in these patients. It should be treated by conservative methods only. The cure of such infections seems to have little beneficial effect upon the pulmonary disease.

Specific Measures: The emptying of the dilated bronchi of infected secretion by postural drainage is the best medical treatment. This is best attained when the patient leans over the side of the bed with the affected side of the chest uppermost so that his head is lower than his hips. This position should be maintained until drainage is complete, usually for five to 15 minutes at a time. The procedure should be repeated as often as is necessary to keep the bronchi well drained.

Repeated bronchoscopic drainage may provide considerable symptomatic relief. However, the discomfort involved is considerable, and the method is not widely recommended.

The injection of lipiodol into the affected bronchi has been of benefit in some cases, but the results are not consistently good. It may be worth a trial.

All observers agree that vaccines are of no use in the treatment of chronic basal disease.

X-ray therapy is at present being tried but it is still in the experimental stage.

Some patients do not improve under conservative management and to prevent the development of bronchiectasis, certain authors suggest the use of collapse therapy. Phrenicotomy has been tried and has controlled the symptoms in some cases. However, the use of this and other measures is a matter of individual opinion and no attempt is made to recommend them in this paper.

SUMMARY

1. The general characteristics of chronic non-tuberculous basal infections of the lungs are presented.
2. The symptoms, clinical course, physical signs and roentgenological findings are described. Special mention is made of the diagnostic value of the ordinary flat plate of the chest.
3. The importance of ruling out the presence of pulmonary tuberculosis is stressed.
4. Evidence is presented to show that chronic non-tuberculous basal infections of the lungs represents a mild or beginning bronchiectasis.
5. The methods of treatment are outlined. Emphasis is placed upon the prophylactic value of the proper treatment of acute respiratory infections in childhood.

BIBLIOGRAPHY

1. Andrus, P. M.: Chronic Nonspecific Pulmonary Disease; *Am. Rev. Tuberc.*, 41:87, 1940.
2. Austrian, C. R.: Chronic Basic Non-Tuberculous Disease of the Lungs; *Internat. Clin.*, 182:367, 1931.
3. Cherry, H. H.: Chronic Basal Non-Tuberculous Pulmonary Inflammation; *Am. J. M. Sc.*, 182:367, 1931.
4. Hamman, L. and Wolman, S.: Chronic Non-Tuberculous Pulmonary Infections; *Tr. Nat. A. Study and Prev. Tuberc.*, 12:1, 1916.
5. Hamman, L.: Chronic Non-Tuberculous Basal Infections of the Lungs; *Am. Rev. Tuberc.*, 40:363, 1939.
6. Hedblom, C. A.: Pathogenesis, Diagnosis and Treatment of Bronchiectasis; *Surg., Gynec. & Obst.*, 52:406, 1931.
7. Warner, W. P.: Bronchiectasis, Aetiology, Diagnosis and Treatment; *Canad. M. A. J.*, 27:583, 1932.
8. Warner, W. P.: Factors Causing Bronchiectasis; *J.A.M.A.*, 1666, November 23, 1935.

VITAMIN ADVERTISING AND THE MEAD JOHNSON POLICY

The present spectacle of vitamin advertising running riot in newspapers and magazines and via radio emphasizes the importance of the physician as a controlling agent in the use of vitamin products.

Mead Johnson & Company feel that vitamin therapy, like infant feeding, should be in the hands of the medical profession, and consequently refrain from exploiting vitamins to the public.

The Carotid Sinus in Disease

By MAX NAREFF, '42

THE carotid sinuses are small, bilateral, aneurysmal-like dilatations, usually restricted to the proximal part of the internal carotid artery. From them originate the carotid sinus reflexes, the homeostatic mechanisms which are important in regulating the circulation and respiration.

I.—ANATOMY AND PHYSIOLOGY

The carotid sinus possesses a rich supply of specialized characteristic nerve endings, whose fibres pass centrally as the carotid sinus nerve to join the glossopharyngeal nerve, and thence to a central termination in the medulla.

Sollmann and Brown, in 1912, were among the first to direct attention to the fact that the carotid sinus zone was a reflexogenic centre through which cardiac and peripheral reflexes were initiated. However, it is largely through the work of Hering and Heymans that the true nature of the vascular proprioceptive sinus control centre was exposed.

The carotid sinus nerve endings are pressorsensitive, reacting to changes of pressure within the vessel wall. Bronk and Stella have shown that impulses are continuously being conducted centrally in the sinus nerve. The sinus is constantly discharging; the action-potentials varying with the phases of cardiac activity. Every systole with its rise in blood pressure is accompanied by "packets" of discharge, with lessened activity in the interval. This would seem to indicate that the sinus nerves have a tonic inhibitory influence on the medullary centres involved.

Arterial pressure is maintained largely by peripheral resistance and cardiac output. Accessory factors include circulating blood volume, elasticity of large vessels and viscosity of the blood.

Increase in endovascular pressure within the sinus reflexly induces peripheral vasodilatation and slowing of the heart with reduction in cardiac output and resultant fall in arterial pressure. Here the tonic inhibitory influence on the cranial centres is increased. The afferent link of the reflex arc is the glossopharyngeal nerve and the efferent limb is the vagal and sympathetic nerves. Decrease in endovascular sinus pressure causes peripheral vasoconstriction and increased cardiac rate with resultant rise in blood pressure. Here the tonic inhibition on the medullary cardiovascular centre is decreased. Haemorrhage, by diminishing the circulating blood volume, tends to lower the arterial blood pressure. This is compensated by the pressor-proprioceptive action of the sinus reflex. Heymans has shown that in man the peripheral vasoconstriction and dilatation occur in the splanchnic vascular

area. Changes in the blood pressure result from changes in the heart rate and in peripheral resistance. Although a fall in pressure may be due to changes in peripheral vascular tone alone, with no dependence upon a slowing of the heart rate. This is shown by the fact that use of atropine will not alter the fall in pressure despite the cardiac slowing which it produces. It has been shown also that the carotid sinus has a humoral function in the sense that it reflexly controls adrenaline discharge. This may play some role in its pressor abilities, but only as an accessory, since the sinus reflex can operate in the absence of the adrenals.

Experimental evidence is contradictory as to the role of the carotid sinus in the cerebral circulation. There are at present two schools of thought on the matter; one, stating that the reflex has only an indirect action on the cerebral vessels; this occurring by means of its action on the general circulation. Endosinusal hypertension would result in vasodilatation in the general circuit but vasoconstriction in the cerebral vessels, and the opposite would occur with endosinusal hypotension. The second school claims that the reflex will affect directly the cerebral vessels producing cerebral vasoconstriction with sinus hypotension, and vasodilatation with hypertension. The authorities of both schools are agreed, however, on the fact that the carotid sinus reflexes play a paramount role in maintaining the adequacy of the cerebral blood flow (*i.e.*, cerebral vascular homeostasis). Thus, "failure of the sinus" may be manifested by cerebral symptomatology.

In addition to its pressoreceptors, the carotid sinus wall is now known to possess also sensitive chemoreceptors, which make it possible for changes in the oxygen and carbon dioxide content of the blood to influence blood pressure and respiration. Bard is of the opinion that under normal circumstances the sinus reflexes play no role in respiration. It is only under conditions of extreme degrees of oxygen lack and carbon dioxide excess that the chemoreceptors operate.

II.—THE CAROTID SINUS IN AORTIC VALVULAR DISEASE

With incompetency or insufficiency of the aortic valve, the pulse wave possesses a typical character. The pulse beat is described as water-hammer or collapsing. This is due to the sudden rise in blood pressure above normal, immediately followed by a marked collapse of the pulse coincident with the fall in diastolic pressure level. The rise in blood pressure is due no doubt to the increased amount of blood ejected by the enlarged hypertrophied ventricle. Wiggers has shown that the prime cause of the low diastolic pressure is the regurgitation of blood into the left ventricle. Peripheral vasodilatation may, however, also play a role. It is thought that the sudden ejection of a large quantity of blood into the systemic vessels stimulates the carotid sinuses to increase the inhibition on the vasomotor centre, causing

peripheral vasodilatation. Fishberg points out that many patients with aortic regurgitation possess a warm, moist skin which might indicate that arteriolar dilatation is present. Arguing now against the carotid sinus involvement, Fishberg notes that there are many "Aortics" who have a marked pallor and cold skin. To this latter may be added Heyman's observation that the carotid sinus reflex mediates vasodilatation primarily in the splanchnic area. Thus it appears that in some cases the reflex may play some sole in producing the water-hammer pulse, but in general it is not at all conclusive. Further work remains to be done on action-potentials in the carotid sinus nerves of "Aortics" before the exact mechanism is fully appreciated.

Aortic stenosis is frequently accompanied by a tendency to fainting spells, dizziness and actual syncope. It has been suggested that this results from the cerebral anaemia due to the small volume of the pulse. That the stroke volume output in aortic stenosis is not impaired tends to negate this explanation. It has been suggested also that the cerebral symptoms may be linked with peculiarities in the carotid sinus reflex, since, as we shall see, if this reflex is hyperactive, dizziness, weakness and fainting attacks can and do occur. Marvin and Sullivan in an electrocardiographic study suggest that the sudden death and syncope of aortic stenosis may be due to hyperactivity of the carotid sinus reflex. In calcareous aortic stenosis the sinus wall may also be arteriosclerotic and this may play some role in the mechanism. Here again, further study is necessary since it has been shown that most cases of aortic stenosis have no apparent abnormality of carotid sinus sensitivity. In fact, Weiss states that the syncope of aortic stenosis probably depends on hyperirritability of the myocardium to normal vagal impulses, as a result of myocardial ischemia, and is not of carotid sinus origin.

III.—THE CAROTID SINUS IN CONGESTIVE HEART FAILURE

Acceleration is one of the methods by which the heart can increase its output. In congestive heart failure the cardiac output is diminished and the rate is usually rapid. Obviously with distended veins, the Bainbridge reflex is an important factor in this acceleration. Fishberg, in his classic, "Heart Failure," suggests that the tachycardia may in addition be due to action of the carotid sinus and aortic reflexes, since they might be stimulated by the lessened cardiac output. He also records that there is no available evidence to support this supposition. However, in cases of congestive heart failure, there is fall in blood pressure, particularly systolic fall. This may play some role in stimulating the sinus reflexes and aiding in the compensatory tachycardia.

IV.—THE CAROTID SINUS IN PERIPHERAL CIRCULATORY FAILURE (SHOCK)

As previously stated, the carotid sinus acting as a vascular proprioceptive mechanism tends to compensate for haemorrhage. Should the

sinus be depressed, this compensatory mechanism is impaired. Heymans has shown that "Spinal or general anaesthesia, narcotics, barbiturates, histamine, severe anoxaemia and pulmonary hyperventilation may have depressing and even paralyzing effects upon the carotid sinus." This causes Heymans to suggest the possible role of carotid sinus dysfunction in peripheral circulatory failure. It would be merely secondary to the important factors in maintaining the condition of lowered blood pressure, since the reflex restorative mechanisms would be inefficient.

In peripheral circulatory failure tachycardia accompanies the low blood pressure. This tachycardia cannot be due to the action of the Bainbridge reflexes since there is no venous distension; the veins being collapsed. Thus it might be conceivable for the sinus reflex to indulge in accelerating the heart and yet be inefficient or unable to maintain a normal blood pressure. There is no evidence for this.

V.—THE CAROTID SINUS SYNDROME

Individuals differ in their response to mechanical stimulation of the carotid sinus. In about thirty per cent of normal individuals the carotid sinus is insensitive to manipulation. In others, varying degrees of cardiovascular or cerebral phenomena are noted. Females are said to react less well than males and the response increases with age and elevation of blood pressure.

Through the efforts of Weiss and his colleagues, we have today knowledge of this interesting cardiovascular—cerebral syndrome. They have shown that an abnormally sensitive carotid sinus can be responsible for attacks of syncope and they could reproduce these attacks by pressure over one of the carotid sinuses. A hyperactive carotid sinus may be unilateral or bilateral.

The clinical manifestations are due to cerebral anoxaemia resulting from diminished flow of blood through the brain. Weiss and his co-workers classify the hyperreactors into three groups depending upon how the cerebral anoxaemia is produced.

(a) *The vagal type:* Here the anoxaemia is due to a temporary cardiac standstill due to sino-auricular or auriculo-ventricular block. There is also an associated fall in blood pressure. These attacks may be controlled by the intravenous administration of 1 mg. of a solution of atropine sulphate; the atropine inhibiting the vagus action. One-half cc. of a (1-1000) solution of epinephrine hydrochloride subcutaneously also will control these attacks, by stimulating the heart and raising the blood pressure.

(b) *The depressor type:* Here the anoxaemia results from a marked decrease in blood pressure unassociated with cardiac slowing

and resulting from peripheral vasodilatation. Atropine will, of course, have no effect here and epinephrine is the controlling agent. This type is rare in its pure form.

(c) *The cerebral type*: Here the syndrome results from reflex action on the cerebral vessels with little or no change in blood pressure or heart rate. There is no controlling drug for this type.

“Clinically the syndrome is manifested by short attacks of syncope with or without convulsions, occurring almost always with the patient in the upright position. It is usual for pressure against the neck or movements which stimulate the sinus to precipitate an attack. Although some cases apparently occur without cause. The attacks may be preceded by dizziness, weakness, blurring of vision, tinnitus, pallor, or epigastric distress. Occasionally there are no prodromes. Frequent mild attacks may occur between attacks of syncope.” Patients with the carotid sinus syndrome may or may not have pathology in the sinus wall.

The condition is diagnosed by the history and by mechanical stimulation of one of the carotid sinuses. By noting the blood pressure, the pulse rate and the effects of atropine and epinephrine the type of sinus mechanism involved can be determined.

Weiss and Baker state that the overactivity of the sinus reflex may be due to one of three factors:

1. The excitability of the afferent nerve endings within the sinus.
2. The state of the medullary centres.
3. The excitability of the efferent cardiovascular nerve endings.

Sigler suggests that all factors point to a constitutional vagotonic tendency.

Purks believes that another factor is sclerosis of the sinus wall, since this makes the application of stimuli more effective.

Lewis described a syndrome which he called vasovagal syncope. The syncope consisted of fainting spells occurring in young individuals in poor physical condition. These attacks occurred when the body had been erect for some time. Lewis concluded that a disturbance in the carotid sinus reflex mechanism might be present in such cases, but presented no evidence of hyper-excitability of the carotid sinus.

Whether the carotid sinus mechanism is involved in the syndrome of postural hypotension is uncertain.

VI.—THE CAROTID SINUS IN HYPERTENSION

Experimental evidence is somewhat contradictory as to the role of the carotid sinus in the production of hypertension. While sections of

the carotid sinus nerves can produce hypertension, it is at present not believed that abnormality of sinus action is a mechanism in the etiology of essential hypertension.

VII.—THE CAROTID SINUS IN ANGINA PECTORIS

Levine, in his "Clinical Heart Disease," states that "occasionally attacks of angina pectoris are instantly relieved by massaging the carotid sinus." Wasserman has also observed this. The mechanism involved here is obscure, but cardiac inhibition may be a factor.

VIII.—THE CAROTID SINUS AND THE ACTION OF DIGITALIS ON THE HEART

Digitalis acts directly on the myocardium, and also indirectly, through an increase of the vagal tone. The latter can be demonstrated by section of the vagi. Heymans and his co-workers have shown that perfusion of the carotid sinus with digitalis will produce cardiac slowing, which cannot be produced by perfusion of the drug through the medulla alone. This would suggest that part of the digitalis action in increasing vagal tone is through the sinus reflex mechanism. In this respect it should be mentioned also that digitalis increases the sensitivity of the myocardium to the normal vagal tone.

IX.—THE CAROTID SINUS IN THE DIAGNOSIS OF CARDIAC ARRHYTHMIAS

In certain patients, stimulation of the carotid sinus may be utilized with advantage as a diagnostic and even a therapeutic measure. The effect of carotid sinus stimulation on the heart is noted by use of the electrocardiogram. Gold points out that with a rapid regular rate varying between 150-175 per minute, it is sometimes difficult to note the exact auricular action and thus make a correct electrocardiographic diagnosis. Thus a moderate slowing of both the auricles and ventricles on the graph, as a result of sinus stimulation, indicates a sinus tachycardia. Complete temporary stoppage of the entire heart indicates paroxysmal auricular tachycardia. Complete transient standstill of the ventricle with no effect on the auricles suggests auricular flutter.

However, in many cases, the carotid sinus will have no effect and in these it is best to use some other means of vagal attack upon the heart, such as the oculocardiac reflex, deep inspiration and lowering of the head.

Therapeutically, in auricular paroxysmal tachycardia, carotid sinus pressure may abolish an attack. In auricular flutter, the effect is fleeting and so the use of the sinus is of no avail. In auricular fibrillation, carotid sinus manipulation has no effect.

The carotid sinus reflex plays a definite role in health and disease.

Many of its mechanisms in disease are still obscure and await further elucidation.

REFERENCES

- Ask-Upmark, E.: *Acta Psychiatrica et Neurologica*, Supp. 6, 1934.
 Bouckaert, J. J., and Heymans, C.: *J. Physiol.*, 79:49-96, 1933.
 Bronk, K. W., and Stella, G.: *J. Cell. and Comp. Physiol.*, 1:113-130, 1932.
 Code, C. F., and Dingle, W. T.: *Proc. Staff. Meet. Mayo Clinic*, 10:139, 1935.
 Ferris, E. B., Jr.; Capps, R. B., and Weiss, S.: *Medicine*, 14:377-456, 1935.
 Fishberg, A. M.: *Heart Failure* (Lea & Febiger), 1937.
 Gold, H.: *M.C.V.A.*, 24; May 1940.
 Heymans, C.: *New Eng. Med. Jour.*, 218:147, 1938.
 Levine, S.: *Clinical Heart Disease*, 2nd Ed. Pg. 16, 1940.
 MacLeod's *Physiology in Modern Medicine*, 8th Ed., 1938.
 Marvin, H. M., and Sullivan, H. G.: *Trans. Assoc. Am. Phys.*, 1:265, 1935.
 Purks, W. K.: *Ann. Int. Med.*, 13:270-279, 1939.
 Sigler, L. H.: *Ann. Int. Med.*, 9:1380, 1936.
 Sollmann and Brown: *Am. J. Physiol.*, 30:88-104, 1912.
 Weiss, Soma; Capps, R. B.; Ferris, E. B., Jr., and Munro, D.: *Arch. Int. Med.*, 58:407, 1936.
 Weiss, Soma, and Baker, J. P.: *Medicine*, 12:297-354, 1933.
 Wiggers: *J.A.M.A.*, 97:1359, 1931.
 Wasserman, S.: Quoted in Fishberg.

SQUIBB RELEASES SULPHATHIAZOLE

Sulphathiazole has been released for sale by E. R. Squibb & Sons of Canada Ltd., in the form of 0.5 gram scored tablets for oral dosage and in crystals for compounding prescriptions and for determination of blood concentration.

Sulphathiazole has received extensive clinical trial and is a noteworthy advance in the chemotherapeutic treatment of pneumococcal and staphylococcal infections. It is the third of the "sulphonamide derivatives" to be released by Squibb, the others being Sulphanilamide and Sulphapyridine. Sulphathiazole is believed to have the following advantages over Sulphapyridine:

1. More uniform absorption.
2. Less conjugation after absorption, so that a higher proportion of the total drug in the body-fluids is chemotherapeutically active.
3. Less tendency to cause serious nausea or vomiting.
4. Greater effectiveness against staphylococcal infections.

Sulphathiazole Squibb is supplied in bottles of 50, 100 and 1,000 0.5 gm. tablets and 5 gm. vials of crystals.

Hare-Lip and Cleft Palate

By S. J. C. MILLER, '38

*Department of Pathology, University of Western
Ontario Medical School*

ETIOLOGY

1. The factor of heredity is of some importance in this particular condition. This was shown as early as 1690 by Regis.
2. Vrolik and Nicati suggested that a mechanical condition was caused by a hypertrophied tongue in cases of cleft palate, which prevented the union of the palatal arches. Other possibilities are those of endocrine dysfunction.
3. The main cause is the failure of the maxillary process on one or both sides to fuse completely with the globular process, causing the formation of a fissure between the philtrum and the lateral part of the lip.

Failure of the palatine processes to fuse with each other and the nasal septum results in a cleft palate. The cause is attributed to an upset in foetal metabolism during the second intra-uterine month of existence. (Gray's Anatomy.)

4. Ritchie has shown that there is a familial incidence as 34 out of his 350 cases show a familial incidence.
5. An associated factor was the presence of multiple congenital deformities in 26 of Ritchie's cases.

INCIDENCE

1. This condition is most commonly found in the white race, where the incidence is 1:1000.
2. The incidence in the coloured race is 1:1800.
3. The condition is much more common in males than females.
4. The left side of the face is involved twice as often as the right side. The left unilateral hare-lip and cleft palate is by far the most common type found, while bilateral hare-lip and cleft palate is more common than right hare-lip and cleft palate.

CLASSIFICATION

Ritchie's classification has been adopted because it seems to be the most suitable and complete one yet devised.

1. Unilateral cleft of uvula.
2. Unilateral cleft of the soft palate.
3. Unilateral cleft of the soft and hard palates.

4. Unilateral cleft of the soft and hard palates and the alveolar ridge.
5. Unilateral cleft of the soft and hard palates, the alveolar ridge and unilateral cleft lip.
6. Bilateral cleft palate with bilateral cleft lip.
7. Median cleft.

A Second Classification

1. Prealveolar process cleft.

Here the alveolar process, hard and soft palates are normal. The defect is a cleft in front of a normal alveolar process. The degree varies from a simple notching of the vermillion border to a cleft ending at, but not involving, the nostril. More rarely, the cleft may involve the nostril and form a complete hare-lip. The deformity may be uni- or bilateral.

2. Postalveolar process cleft.

In this condition the lip and alveolar process are normally united. The hard and soft palates are cleft to varying degrees, but always in a symmetrical manner. Whenever the hard palate is involved, the soft palate is completely cleft.

- (1) Type 1/3—involving the uvula and soft palate only.
- (2) Type 2/3—involving the above and part of the hard palate.
- (3) Type 3/3—involving the uvula, soft palate and hard palate as far as the incisive foramen. This type is the most difficult to repair since the nasal septum and vomer are exposed. This resembles a double cleft, but is simply a medial cleft which has extended to the anterior palatine foramen. The optimum time of operation in this type is after the age of two, usually between the ages of three and four as advocated by Burdick. The ideal methods of approach here are—
 - A. Suture of the hard palate, leaving the soft palate for later repair.
 - B. Suture of the soft palate, raising the muco-periostium, and packing for several days. In one week a delayed suture of the muco-periostium is performed.
 - C. For type 2/3 the straight Langenbeck-Warren operation of medial suture is advocated with repair of both hard and soft palates at one sitting.

3. Alveolar process cleft.

This type involves the lip, alveolar process, hard and soft palates in varying combinations and degrees. About 75% of all cases of hare-lip and cleft palate are included in this group.

In this case, if operation is delayed until after the age of three months, the bones of the maxilla become set and firm. As a result,

mechanical moulding is very difficult to perform. This alveolar deformity should be corrected at an early age, before the above condition takes place.

The cleft in the alveolar process is always the first to be considered. The defect in the lip is always closed first, not only for cosmetic appearance and functional result, but because of its effect in aiding in the approximation of the cleft alveolar process underneath. In the case of double hare-lip fitting into this group, the premaxilla should always be pushed back into contact with the alveolar process as soon as possible.

AGE OF OPERATION

The optimum time for the repair of the cleft lip and alveolar arch is as soon after birth as the patient's physical condition will warrant. As shown by Foucar, Ritchie, Burdick and others, the bones forming the alveolar arch are more pliable, and more easily moulded up to the age of three months. Another factor is to correct the deformity as soon as possible to allow proper nursing. Thus the optimum time for the correction of hare-lip is ten days to two weeks after birth if the child is gaining in weight and the temperature is normal.

The cleft in the palate is usually too wide to be corrected at this time. However, correction of the cleft lip and alveolar arch facilitates normal growth of the bony palate. This is usually corrected between the ages of three and four.

Axhausen prefers to operate at the end of the second year, although in his series of 100 cases, only 25 were operated on before the age of three. Vaughan suggests that the operation should take place between the ages of 18 months to two years, since the tissues are in good shape at this time, and the resulting mortality is lower.

PROGNOSIS

1. Axhausen in 100 cases had no deaths.
2. Ritchie—100 cases, no mortality.

The results obtained in cases of simple hare-lip are very satisfactory, especially if the case is persevered with and followed up with secondary operations, if necessary. The end results in cases of cleft palate have been very discouraging. However, since the use of the modified Langenbeck operation, and especially the Dorrence Push-back operation performed in two stages, the results have improved markedly. The ultimate result in speech improvement as shown in the report of 100 cases by Ritchie—

- (1) 42% show excellent results.
- (2) 43% show good results.

- (3) 14% show fair results.
- (4) 1% show poor results.

RULES OF PROCEDURE

1. Repair of the alveolar process cleft should always be done as early as is compatible with the physical condition of the child.
2. Repair of the hare-lip is left to the judgment of the operator.
3. Repair of the hard and soft palates is left to the age of about three.
4. The chief factor of corrective surgery is to establish normal speech.
5. It must be remembered that these patients are never emergency surgery.
6. A proper formula should be established and be well handled by the infant before operation can take place.
7. Periodic observation from birth to time of operation should be carried out.
8. It must be remembered that no child ever dies from cleft palate, but from the surgery attempted.
9. There must be an intelligent selection of cases before the onset of speech; speech training should be started immediately after operation.

PROBLEMS TO BE REMEMBERED

1. The cosmetic appearance of the result.
2. The avoidance of scars and adhesions of the palate.
3. Voice training.
4. The prevention of inferiority complexes.

It is to be remembered that the voice disturbances in hare-lip and cleft palate are due to the inability of the patient to make explosive voice stops ranging from the palate to the lips, especially in the enunciation of the consonants g, k, d, t, b, v and p. Abnormal breathing and posture are also generally at fault and should be corrected. If surgery is not done before the age of four, the problem of mental development is involved. It is to be remembered that good anatomic results do not mean good speech, and therefore speech training following operation is of the utmost importance.

OPERATIVE PROCEDURE

The surgical techniques will be named only in passing, as they are all well outlined in any standard text book of surgery.

1. Nelaton's operation.
2. Graefe's operation.
3. The Rose operation.

4. The Mirault operation. This is the most complicated of the methods attempted, but yields the most satisfactory results cosmetically.
5. The Hagedorn operation is useful for double hare-lip.
6. The Langenbeck-Warren operation.
7. The Dorrence Push-back operation has given the best results generally of any technique attempted.

In all cases of repair of hare-lip and cleft palate, secondary operations, often plastic in nature, are required to correct various contour deformities. The most common of these are flatness of the nose, with associated depression, and flatness of the lip. This is due to a lack of forward projection of the premaxilla. Gillies and Kilner advise the use of a buccal inlay graft for the correction of these deformities.

Proper pre- and post-operative care is essential for the success of any type of surgical correction, and in all cases the success of corrective operations, for hare-lip and cleft palate is directly dependent upon the proper pursuance of strict pre- and post-operative care. Furthermore, it is of great importance that the operation be attempted by a surgeon, skilled in oral surgery, rather than the man doing extensive general surgery.

MORTALITY

As shown by Burdick, Dunning and Porter, the mortality in cases undergoing operative correction is rather high. Ritchie, in a series of 350 cases, reports a mortality of 1.4%. Axhausen, in a series of 100 cases, had 0% mortality.

POST-OPERATIVE COMPLICATIONS

1. Aspiration pneumonia.
2. Oral sepsis.
3. Shock.

It is to be noted that the anaesthetic of choice is local anaesthesia. Avertin, supplemented by gas-oxygen, endotracheally, has been used by Fitzgerald.

POST-OPERATIVE CARE

The child's elbows are splinted to keep the fingers from the mouth. Food is offered as soon as there is any prospect of it being retained. Every effort is made to keep the child from crying. Paragoric, or other mild sedatives, are given freely. If an obturator has been used, as advocated by Fitzgerald, it is not removed for one week, in order to allow the skin graft to take. It is then removed daily for cleaning.

RESULTS

1. Cosmetically, the Mirault technique has given most satisfactory results.

2. From the standpoint of voice function, the Dorrence Push-back operation, done in two stages, has given excellent anatomical results.

Author	Healed Palates	Satisfactory Functional Results
Kappeler	86.5%	53.3%
Ranzi and Sultan	69.3%	9.3%
Stahl	58.7%	15.0%
Veau	74.0%	25.0%
Dreher	60.9%	34.7%
Tschmarke	64.3%	9.1%
Turner	44.0%	13.0%

In the above group, the percentage of satisfactory functional results refers to normal speech. However, all the above-mentioned authors are able to bring about approximately 80% good voice function.

BIBLIOGRAPHY

- Blair, V. P., and Brown, J. B.: *Surg., Gyn. & Obst.*, 51:81, 1930.
 Browne, Dennis: *Br. J. Surg.*, 20:7, 1932.
 Burdick, C. G.: *Ann. of Surg.*, 92:35, 1935.
 Davis, A. D.: *Ann. of Surg.*, 52:875, 1931.
 Dorrence, G. M.: *Arch. of Surg.*, 21:185, 1930.
 Fitzgerald, R. R.: *Br. J. Surg.*, 25:816, 1938.
 Foucar, H. O.: *C.M.A.J.*, 28:373, 1933.
 Gattie, G.: *Surg., Gyn. & Obst.*, 51:224, 1930.
 Gillies, H. D.: *Lancet*, 2:223, 1932.
 Ivy, R. H.: *Int. J. of Orthodontia & Oral Surg.*, 1937.
 Johnson, J. B.: *A. J. of Surg.*, 93:103, 1939.
 Kitlowski, E. A.: *Ann. of Surg.*, 95:659, 1932.
 New, G. B.: *Surg. Clin. of N. A.*, 1931.
 Peyton, W. T.: *Arch. of Surg.*, 22:704, 1931.
 Risdon, F.: *C.M.A.J.*, 25:563, 1931.
 Ritchie, H. P.: *Arch. of Surg.*, 28:617, 1934.
 Ritchie, W. P.: *Arch. of Surg.*, 35:570, 1937.
 Vaughn, H. S.: *A. J. Surg.*, 35, 1935.
 Voorhees, I. W.: *A. J. Surg.*, 588, 1938.
 Wardill, W. E. W.: *Br. J. of Surg.*, 16:127, 1928.

THE COD LIVER OIL SITUATION

It is likely that we will experience a shortage of Medicinal Cod Liver Oil unless existing supplies are carefully husbanded. Norway naturally has been eliminated as a source of cod liver oil. To add to our difficulties Iceland, which produces much of the finest oil, had an exceptionally poor season and their yield of oil is only a fraction of the normal.

By drawing on their reserves and enlisting the co-operation of Newfoundland fishermen, E. R. Squibb & Sons of Canada will be able to supply Medicinal Cod Liver Oil in reasonable quantities of their usual high potency. Squibb Cod Liver Oil is so rich that one 5 cc. teaspoonful supplies 9000 international units of vitamin A and 1300 international units of vitamin D (far more than the accepted daily requirement for infants and growing children).

Physicians are urgently requested when prescribing Squibb Cod Liver Oil to order only 1 teaspoonful daily. Where additional vitamin D is required, rather than ordering multiple doses of the plain oil, it will be appreciated if Squibb Cod Liver Oil 10D is specified.

Sleep

By R. BRIAN HOLMES, '43

ALL plants and animals have periods of rest and activity. In the lower animals, including those with a well-organized nervous system, these alterations may not be dependent upon the brain or head ganglion. If an earthworm is cut in two, each part exhibits a regular cycle of rest and activity. In mammals, however, sleep is concerned primarily with the brain, because in an animal with the spinal cord sectioned nothing resembling sleep occurs in that part of the body served by the distal segment of the cord. The fore part of the animal still has a sleep cycle.

With the onset of sleep there occurs a feeling of lassitude and a loss of vision. The eyelids droop and are raised with difficulty. Diplopia is said to appear if attempts are made to keep the eyes open.

Kleitman lists some criteria which differentiate sleep from other forms of unconsciousness such as coma, narcosis, and hypnosis. Some of the more important of these are:

1. A loss of critical reactivity to events in the environment.
2. An increased threshold of reflex irritability and general sensibility.
3. A capacity to be aroused or brought back into consciousness or wakefulness. In contrast to the inactivity of coma and narcosis, sleep is easily reversible.

DEPTH OF SLEEP

The depth of sleep is not constant but varies from hour to hour. This has been studied in experiments in which the subject was aroused by minimal auditory stimuli. Another method of measuring this is by recording the movements of the sleeper, assuming the amount of movement to be inversely proportional to the depth of sleep.

The depth of sleep follows a characteristic curve with individual differences. In the majority of normal adults, sleep is at its deepest level at the end of the first hour. After that, the depth lessens rather rapidly to the end of the third hour, after which it gradually diminishes until the time of awaking. In children, the sleep curve has two maximal levels, the first as in the adult, the second at about the eighth or ninth hour, after which it rapidly decreases till the time of awaking. Sleep in the daytime is usually intense, partly due to lack of a conditioned reflex. The requirement of sleep gradually lessens from infancy to old age.

PHYSIOLOGICAL CHANGES DURING SLEEP

1. The heart rate decreases.
2. The blood pressure falls. Its lowest level is reached about the

fourth hour, and then gradually rises again toward the waking hour. In work done by MacWilliam it was found that exciting dreams could cause the pressure to rise well above the waking level. The volume of the arm, hand, or foot is said to be increased.

3. The pulse rate is 10-30 beats slower.
4. Respirations are slower, but more regular in rate and amplitude.
5. Rectal temperature is slightly lower.
6. The metabolic rate is reduced about 10 to 15 per cent below basal levels.
7. There is increased secretion of the sweat glands. The amount of sweat secreted per hour during sleep is equal to that secreted in an hour of heavy muscular exercise. Lacrimal and salivary secretions are lessened; gastric secretion is not appreciably changed.
8. Urine volume is reduced but the amount of urinary phosphate and acids is increased. The specific gravity of the urine is raised.
9. Very marked changes in nervous properties occur. Muscle tone is minimal. In some experiments on human beings, Kleitman found that at the end of waking periods of 60 to 114 hours, knee jerks were still obtainable, but were not so immediately after the subjects fell asleep. Positive Babinski was obtained. The righting reflex was lost, although the light reflex was retained.

THEORIES OF SLEEP

1. **NEURONE THEORY:** This is related to the contiguity theory and synapses. The workers claimed that there was a retraction of the dendritic processes with a break between the neurons. This is not accepted today.

2. **HOWELL'S THEORY:** Howell worked on the volume of the extremities during sleep, and suggested that there was a fatigue of the vasomotor centre, causing peripheral vasodilatation and a cerebral ischemia. This is demonstrated by the fact that one feels drowsy after a hearty meal, probably because the blood is diverted to the splanchnic regions. Subsequent workers found no reduction of cerebral blood supply during sleep, although it is possible that there may be a reduction to a certain area whose activity is necessary for the waking state.

3. **CHEMICAL THEORY:** Some workers think that sleep may be caused by chemical products of muscular activity, due to the ease with which one can fall asleep after extreme muscular activity. Against this theory is the fact that one need not be fatigued in order to sleep. Other workers suggest the formation by the brain tissue of a substance called "hypnotoxin" which causes sleep. After injection of cerebrospinal fluid from a sleeping animal into the cerebral ventricles of a normal one the latter showed signs of fatigue and fell asleep. Others think the pituitary is connected with productions of sleep in some way.

4. **HYPOTHALAMUS AND SLEEP:** This brings up the question of a sleep centre. Often tumors of the floor and walls of the third ventricle are accompanied by hypersomnolence, as are some lesions of the upper thalamic region. Hess claims that mild electrical stimulation of the diencephalon towards the anterior end of the cerebral aqueduct produces sleep.

5. **PAVLOV'S THEORY:** Pavlov thinks that sleep and internal inhibition are the same thing. Sleep is the spread of the inhibition over the whole cortex, with the subsequent involvement of the subcortical levels. Examples of this are the instances of drowsiness from being read to in a low, even voice, a dull lecture, or boredom from whatever cause. The preparations for sleep probably serve as inhibitory conditioned stimuli. A dog which has fallen asleep during previous experiments may do the same when merely brought into the room where the experiments have been performed. In some cases inhibitory processes only involved the cortex but did not descend to subcortical levels governing equilibrium and postural reactions of skeletal muscles. Here the animal assumed a trance-like state in which muscular tone was retained, the general attitude being described as one of alertness. This is looked upon as a transition stage between wakefulness and sleep. It is suggested also that during sleep all cortical areas are not necessarily under the inhibitory influence. This may be exemplified by the mother apparently in deep sleep who is alert to the slightest noise made by her baby. Here it is the auditory area which has remained outside the inhibitory influence. Dreams are evidently due to cortical activity since they involve memory and the ability to associate various sensory impressions.

6. **KLEITMAN'S THEORY:** This states that sleep is due to the inactivity of the cerebral cortex because of a reduced number of afferent impulses, especially from the muscles. In the onset of sleep, the most important factor is considered to be the fatigue of the neuromuscular mechanism mediating muscle tone, with consequent suppression of impulses from muscle proprioceptors. Loss of muscle tone is a constant precursor of sleep. Normally, in the process of going to sleep at night, all impulses from the postural muscles are stopped. The darkness stops the optical impulses, and the quietness stops the auditory impulses. Here also cortical activity, whether from psychic causes, anxiety, excitement, worry, etc., prevents sleep. Kleitman and his co-workers found that after long periods of wakefulness, the only way to stay awake was to keep moving about, or to remain in a standing position. On lying down and permitting their muscles to relax, they were immediately overpowered by sleep.

Decorticated dogs show periods of sleep alternating with periods of wakefulness, showing that sleep is not dependent upon the cortex entirely. The sleep rhythm is not related to day and night here, but is

made up of a number of shorter or longer periods throughout the twenty-four hours. The sleep periods occur most constantly after meals. In summarizing his theory of sleep, Kleitman postulates:

1. Sleep is an easily reversible activity of the higher functional centres of the cerebral cortex.

2. The inactivity is due to a functional break between the cortex and lower centres.

3. The break results from a decreased number of afferent impulses from the sensory organs, especially those of proprioceptors which may become fatigued.

4. In the absence of such fatigue, voluntary or involuntary relaxation may produce the same effect.

5. Diurnal wakefulness and sleep is a conditioned reflex.

6. Sleep can be correlated with decrease in muscle tone and body temperature.

ELECTROENCEPHALOGRAM AND SLEEP

In 1929, it was found that changes in electrical potential could be recorded from the head of a human being by the use of pad electrodes applied to the scalp, or needle electrodes in contact with the periosteum of the skull. Normally three waves can be recorded.

The alpha waves are rhythmical oscillations in electrical potential, at a rate of 10 per second, with an average voltage of 50 microvolts. They appear with the eyes shut or open, but only with a plain field of vision before them. An attempt of the subject to fix his eyes on any detail causes a disappearance of the waves. Any mental effort, such as mental arithmetic, also causes the waves to disappear.

The beta rhythm is considerably faster, but of lower voltage, 25 to 50 per second, and 5 to 10 microvolts. It is obtained best over the precentral region.

The delta waves are only 1 to 5 per second, but they have a much higher potential, from 20 to 200 microvolts. They are rare in the normal adult while awake, but occur during sleep.

When one is sleeping, the pattern varies with the depth of unconsciousness. In light sleep, delta waves appear, and the alpha waves are superimposed on the slower, larger waves. In deep sleep, the alpha waves disappear, leaving only the deltas. As consciousness returns, the alpha waves return, intermittently at first, then continuously as the waking state is reached.

To experiment with the human brain potentials at the onset of sleep, Davis and other workers had a subject hold in his hand a rubber bulb which was attached to a signal pen. He squeezed the bulb when-

ever he realized that he had just drifted off for a minute. He was instructed to signal twice if he felt he had just awakened from real sleep. Results showed five stages in the process of falling asleep

1. As the subject settles down, his alpha waves increase slightly in voltage and regularity.

2. The next step is the complete interruption of alpha activity for periods of one to five seconds. Sometimes the alpha activity does not disappear entirely, and during these gaps small and irregular alpha waves are present.

3. The interruptions of alpha activity become longer, although at the end of each gap the alpha waves return suddenly, usually with their normal maximal amplitude. At this stage, an increase in the voltage of the longer random delta waves can be first clearly identified.

4. The wavelength of the delta waves increases. Alpha waves when appearing show a slower frequency than in the normal waking state. This slowing of alpha rhythm below its normal frequency is a characteristic phenomenon associated with the approach of real sleep.

5. This shows a complete loss of alpha waves and the appearance of delta waves of voltage of 150 microvolts or more.

These workers conclude that real sleep is present when slow waves (recorded from the vertex), had reached 150 microvolts or more and lasted half a minute. They found also that in clinical study of brain potentials the drowsy state must be avoided because of the similarity of patterns of very early sleep to those described for many abnormal conditions.

In general, during sleep, at first with an increase in the depth of sleep, delta waves appear before alpha waves are gone. Later, during diminished depth of sleep, the deltas disappear before the alphas return. There are minute to minute fluctuations in brain potentials throughout the night, superimposed on a gradual trend from hour to hour.

Subjective reports of sleep and dreams can be correlated with potential patterns, sometimes quite sharply.

Kleitman and other observers, after extensive work on human beings, came to a number of conclusions, some of which are as follows:

1. With respect to every sleep characteristic there is considerable variation from subject to subject, and in the same individual from night to night.

2. Seasonal differences are apparent in the motility during sleep. The lowest motility is during the springtime. The tendency to go to sleep with ease and the incidence of dreams are both highest in the spring.

3. Naps during the waking period do not affect the ease of going

to sleep. The degree of sleepiness, of course, has a direct influence in that direction.

4. External influence of weather and temperature have no effect on motility during sleep.

5. Amount of food and beverage during the evening meal and activity before going to bed do not influence motility in any way.

6. An 8-ounce glass of hot or cold water or milk increases noticeably the incidence of dreaming.

7. When the subject's health is under par, sleep is unfavorably affected, but much less so in frank illness.

8. None of the above influences on sleep affect all subjects in the same way, or a particular subject the same amount on different nights.

SUMMARY

1. All plants and animals have periods of rest and activity.
2. Sleep differs from other forms of unconsciousness.
3. The depth of sleep varies from hour to hour, and sleep is strongest at the end of the first hour.
4. There are marked changes in heart rate, blood pressure, pulse, respiration, metabolism, and nervous properties.
5. There are many theories of sleep, the true properties of sleep probably being described in none of these, but touched on in all of them.
6. There are definite relationships between sleep and electroencephalogram patterns.

BIBLIOGRAPHY

- Bard: MacLeod's Textbook of Physiology, 1938 edit.
Best and Taylor: Physiological Basis for Medical Practice, 2nd edit., 1939.
Blake, H.; Gerard, R. W., and Kleitman, N.: Potentials During Sleep; *J. Neurophysiology*, 2:48, Jan. 1939.
Davis et al: Human Brain Potentials at the Onset of Sleep; *J. Neurophysiology*, 1:24, Jan. 1938.
Grinker, R.: *Neurophysiology*, 1934.
Kleitman et al: *Sleep Characteristics*; U. of Chicago Press, 1937.

Elliptical Cell Poikilocytosis

By H. W. BOYES, '42.

THE first case of elliptical cell poikilocytosis or ovalocytosis to be reported was that of a student in the Ohio State University in 1902. This student while working in the histological laboratory noticed the peculiarity of his own red blood cells. The case showed a normal haemoglobin and red cell count. An elaborate study was made for a period of four months and the same characteristics were noted continually. The student was a healthy mulatto, 22 years of age. Unfortunately, this student died about four months after the original observation. However, he died of a cardiac failure after an attack of acute inflammatory rheumatism which had nothing to do with the blood condition. The fact that in this case the physical condition was good, with a normal number of red and white cells and normal haemoglobin with absence of normoblasts and megaloblasts made the diagnosis of a new blood abnormality necessary. Unfortunately no family history was obtainable here.

In subsequent cases fragility tests have been done and were found to be normal and the cells examined by various methods—slides dried rapidly and slowly, blood diluted with Hayems, etc., and the cells always appeared elliptical. A case reported by Bishop of New York in 1914 was a male, age 41 and, in this case, the blood of his sister was found to be the same. In both these cases the haemoglobin, white blood count and red blood count were normal. The case reported was in hospital for appendicitis making an uneventful recovery and his sister had never been sick in her life. The condition was now being suspected of being congenital.

From that time until 1939 approximately 162 cases have been reported in the literature.

Hereditary Characteristics: Vischer in 1938 reported a study of 44 cases in three generations among whom he found twelve cases who were all healthy with otherwise normal blood. Hunter and Adams in 1929 reported a Dutch family of 18 members in three generations. Cheney reported a family with 14 cases in four generations. Johns Hopkins Hospital has studied a negro family of which twelve individuals in two generations were examined and four showed the condition. None of the cells were suggestive of sickle cells.

Other families have been studied in a similar way and similar results found. All members of a family reported by Cheney, of San Francisco, transmitted the characteristic to one or more offspring. It is therefore proven to be hereditary and is thought to be transmitted according to the Mendelian Law.

This condition is not limited to any race in contrast to sickle cell anaemia found only in negroes. It has been found in Americans of Dutch,

Italian and Scotch-Irish descent; in Germans, Italians, Russian Jews, American negroes and Australian mulattoes. It is common to both sexes and is therefore not a sex-linked characteristic. The abnormality has been found in ages varying from 82 years to 11 months. All blood groups are represented in the condition. With one exception, the abnormality has never been known to skip a generation.

Elliptical Cell Occurrence in the Absence of Hereditary Trait: At Johns Hopkins Hospital, a study of the presence of elliptical cells in anaemic and non-anaemic individuals was made. Elliptical cells were found in 89 per cent of non-anaemic individuals and in 98 per cent of cases of anaemia. In the red cells of the non-anaemic, there were only one to 15 per cent elliptical cells and in 12 per cent of the anaemic patients the elliptical cells were as high as 25 per cent of red cells. This was done merely to show the presence of elliptical cells in normal and abnormal routine bloods. In contrast to these findings, in individuals with elliptical cell trait, the percentage of elliptical cells was found to be high, often around 90 per cent.

Physical Properties of the Cells: The etiology is unknown. It has been thought to be faulty erythropoiesis, influences of blood plasma, inherent quality of the cell, racial characteristics, and anoxaemia. A permanent transmission cannot be affected by transfusion and normal cells are unaltered by the plasma of persons with the anomaly. Autopsies, bone marrow studies and splenic punctures show that the cells are not elliptical when they are formed. Mechanical effects, temperature, twenty-four hour hanging drop preparations, normal blood serum, single isotonic saline washing, hypotonic saline washing, picric acid, carbon dioxide, nitrous oxide, oxygen, cholesterol, anticoagulants and the appearance of a state of anaemia all fail to affect the constancy of the ellipsoid state of the erythrocytes.

Elliptical erythrocytes are more resistant to haemolytic agents and have a decreased fragility. Bleeding time, clotting time, clot retraction, platelets, icteric index and Van den Bergh are all normal. Elliptical cells are heavier than normal cells. Potassium cyanide has been noted to increase the number of elliptical cells rapidly. Lecithin has been found to change the elliptical cells to round cells. In no case has there been 100 per cent elliptical cells in the blood.

It is distinguished from sickle cell anaemia by the absence of crescent-shaped erythrocytes, although elliptical cells are associated with sickle cell anaemia. Also, in elliptical cells there is no increase in the number of abnormal cells on standing in wet preparation, and no decrease in carbon dioxide mixtures, as is always observed in sickle cell anaemia.

Elliptical cells of a donor have been shown to disappear from the normal circulating blood of a recipient in two months. This, evaluated in the light of their increased resistance to haemolysis, may be of some significance in consideration of the life duration of the erythrocyte in

vivo. This condition may be of medicolegal value in the determination of paternity, etc.

Relation Between Elliptical Cell Anaemia and Other Conditions: At the Mayo Clinic, out of eight cases, three cases showed the typical features of haemolytic icterus. All three cases were members of the same family. This may or may not have any significance.

It has been thought to be related to anaemia, especially pernicious anaemia, and hypochromic anaemia, but there seems to be no sound basis for any relationship to anaemia.

This condition appears to be related to no other condition and all cases reported have been picked up by routine blood examinations and the conditions which have brought the patients to the hospital have had no relationship to the disease.

SUMMARY

The presence of elliptical erythrocytes in human blood is familial phenomenon appearing as a non-sex-linked Mendelian dominant characteristic which is not associated with any known disease and is unaffected by any tried therapy. The etiology is unknown. The elliptical erythrocyte is not a poikilocyte of any of the known anaemias including sickle cell anaemia. It appears to be a congenital anomaly compatible with an otherwise normal healthy individual.

REPORT OF A CASE

Mr. S.—Admitted to Victoria Hospital, May 10, 1940. Male, age 62.

Complaints: Patient admitted with extreme photophobia, could not tolerate his eyes uncovered even in a dark room but could see well.

History: The eye trouble followed typhoid fever in 1903. His eyes had been weak ever since. He suffered snow blindness in March, 1940, and the eye trouble was worse following this. The photophobia had been progressive.

Physical: The eyes showed marked blepharospasm without much conjunctivitis. Fundi showed normal discs and vessels, but there were numerous tiny bright fatty collections, most numerous below the discs, and also present all around the discs. These did not interfere with vision.

No cardiovascular or glandular abnormalities were found.

Laboratory Studies: Urine studies showed no abnormalities.

Routine blood study:

Haemoglobin—90.	Differential
Red blood count—4,180,000.	Neutrophils—66.
Colour index—1.09.	Mature—40.
White blood count—6,850.	Young—26.
	Lymphocytes—32.
	Monocytes—2.

Red blood cells showed marked variation in shape and size. The

majority were elliptical in shape. There were many poikilocytes. There was hyperchromia and monocytosis.

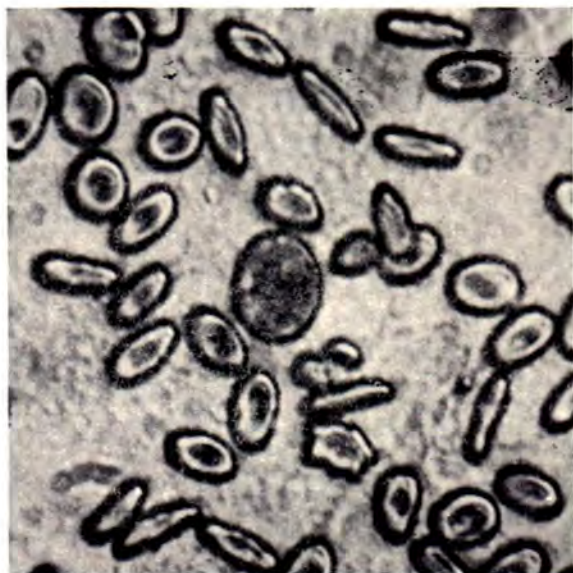
Kahn and Blood Wasserman were negative on two occasions.

Electrocardiogram showed no abnormality.

Chest X-ray was negative.

Glucose Tolerance Test was normal.

Blood Cholesterol was normal.



Photomicrograph of a stained smear of cells taken from Mr. S.

Diagnosis: The photophobia was considered to be a mental problem since there was no organic cause. The elliptical cell anaemia here, as in other cases reported, had absolutely no relationship to the condition which brought the patient to the hospital, and was picked up as in other cases in the routine blood examination. No family history of the disease was obtainable.

BIBLIOGRAPHY

1. Archives of Internal Medicine, Vol. 14, 388-392, 1914 (2).
2. Transactions of the Association of American Physicians, Vol. LIV, pp. 355-358, 1939.
3. American Journal of Clinical Pathology, Vol. 8, pp. 391-397, 1938.
4. Johns Hopkins Hospital Bulletin, Vols. 62 and 63, pp. 209-219, 1938.
5. Journal of American Medical Association, Vol. 98, Part 1, pp. 878-881, 1932.

Abstracts

THE TREATMENT OF ACUTE ATTACKS OF BRONCHIAL ASTHMA BY INTRAVENOUS INJECTION OF AMINOPHYLLIN

By H. A. CARR

J. Lab. and Cl. Med.; 25:1295, 1940

The article deals with the intravenous injection of aminophyllin to relieve bronchial asthma. It begins by giving a brief history of the various substances used in the treatment of bronchial asthma. The first purine body found to have an effect on asthma was hot coffee which sometimes relieved an acute attack. Other purine compounds were used in the treatment, such as aminophyllin, Xanthine group and theophyllin. It was also observed that aminophyllin intravenously apparently restored sensitivity to adrenalin.

In this series, 41 cases of acute bronchial asthma were treated over a period of 10 months on the wards of the 4th Medical Division of Bellevue Hospital. It was found that 22 of these cases were refractory to adrenalin, and their acute attacks were treated by intravenous injections of aminophyllin; 0.48 gms. in 2 cc.'s of aqueous solution, injected within two minutes. It was found that dizziness, vomiting and nausea occurred in about one-third of the cases, but there were no serious results. Twenty-two of the patients who had a disease duration of one to 20 years were given 78 injections of the aminophyllin, and in one-half the cases relief was immediate; in the others relief was evident in 20 to 30 minutes, with duration of benefit from one hour, to absence of recurrence over a period of days. In no patient was it necessary to give more than one injection within a period of less than two hours. Twelve patients were treated by intravenous injection of aminophyllin who had no history of previous medication, and in eight cases relief was immediate; in four cases it occurred in 10 minutes. Two patients who had other complications did

not have relief lasting over a period of one hour. Thus, aminophyllin, given intravenously, is a most efficacious drug in the treatment of intractable acute attacks of bronchial asthma, especially in those cases which are refractory to adrenalin.

—CLARK WILLOUGHBY, '42.

THE RELATIONSHIP OF MIGRAINE TO HYPERTENSION AND TO HYPERTENSION HEADACHES

By J. W. GARDNER, G. E. MOUNTAIN
and E. A. HINES, JR.

Am. J. Med. Sc.; 200:50, 1940.

The authors made a study of 100 patients at the Mayo Clinic who had been seen in consultation because of hypertension, all with a systolic blood pressure of more than 160 m.m. of mercury. These patients were questioned as to the presence of migraine, or a previous history of migraine; also the presence of a hypertensive headache. A control of 100 patients with normal blood pressure was used.

The results of this investigation showed that migraine occurred approximately five times as frequently among those who have hypertension as it does among those who do not have hypertension. Vasoconstriction, which is an important factor in the production of both migraine and hypertension, may be influenced by genetic factors, especially concerning the inheritance of a certain type of personality—the ambitious, meticulous and exacting individual. The patients who were noted to have recovered from attacks of migraine before or at the time of onset of their hypertension frequently experienced headaches of the hypertension type; and certain patients had both types severely. It was obvious that the individuals who had migraine were more likely to experience the hypertension type of headache than those not similarly afflicted.

The authors describe migraine as a headache with attacks of a periodic nature, the pain confined to one side of the head, with visual and gastrointestinal disturbances; the hypertensive headache is one that is present when the patient awakens, reaches its greatest intensity before breakfast, and disappears after breakfast; it is often occipital, but seldom unilateral.

—M. ELIZABETH FORBES, '42.

**ACUTE CHOLECYSTITIS PRECEDING
NEOPLASTIC COMMON BILE
DUCT OBSTRUCTION**

By ROBERT E. ROTHENBERG and
SHEPARD GERARD ARNASON

Ann. of Surg.; 112:400, 1940

A series of eight cases are reported in which acute cholecystitis occurred in the early stages of neoplastic common bile duct obstruction. At operation, the following was found: gangrenous cholecystitis in two cases, empyema of the gall bladder in one and acute inflammatory cholecystitis in five. In none of these cases did the surgeon suspect malignancy at operation. Enlarged lymph nodes were noticed in some cases but were considered inflammatory in origin from the acute gall bladder pathology. The period between the operation and symptoms of malignancy varied from a few days to twelve weeks.

It is admitted that cholecystitis probably plays an unimportant rôle in the etiology of neoplasm of the bile ducts. However, the short interval between acute cholecystitis and the onset of malignant symptoms leads one to believe that the gall bladder infection caused the underlying tumor to grow more rapidly. Biliary stasis, increased intraductal pressure, and the pressure by a tumor on cystic vessels might cause an acute cholecystitis.

Although an extensive abdominal exploration should not be carried out in the presence of an acute suppurative lesion, still it is suggested that a careful search be made in the region of the common bile duct and head of the pancreas in such cases.

—ALLAN HOGG, '42.

ADIE'S SYNDROME

By JOHN McDOWELL MCKINNEY and
MAURICE FROCHT

J. Am. Med. Sc.; 199:546, 1940

The authors add seven more cases of this interesting syndrome, which simulates tabes, to the medical literature. They regard it as a disease of unknown etiology, perhaps heredodegenerative in nature. It occurs in younger people, more commonly females, and runs a benign chronic course, alone or in conjunction with other diseases. It is characterized by: (1) Loss of deep reflexes, typically confined to the ankle jerks and most often unilateral; (2) Pupillary changes, typically a unilateral tonic pupil which does not react to light and reacts tonically to accommodation. The pupil differs from the Argyll-Robertson pupil in that (a) it is unilateral, (b) mydriatic, (c) regular, (d) there is no atrophy of the iris, (e) there is sustained tonic contraction in accommodation. A classification of atypical cases is also presented.

—MAX NAREFF, '42.

HYPOTENSION: THE IDEAL NORMAL BLOOD PRESSURE

By S. C. ROBINSON

N. E. Jr. Med.; 223:11, 1940

Hypotension has been looked on as a disease entity. A case of low blood pressure lacked stamina, had cold extremities and showed an inability to do prolonged mental or physical work. "They are not exactly ill, yet they are rarely well." (Norris).

Between one-fourth and one-third of the adult population have blood pressures under 110 mm. (systolic) and 70 mm. (diastolic). Such pressures are designated hypotension.

It is commoner in young women and in slender builds. Low pressures do not tend to rise with age; rather they maintain an even level. The mortality rate is lower than that of average pressures. There are no symptoms peculiar to or due to low blood pressure.

Neither fatigue nor vitality is a function of the level of blood pressure;

rather, they are related to the daily physical activity of the individual. In fact, those noted for endurance, vitality, viz., trained athletes, farmers and other active groups are also noted for their high incidence of low blood pressure.

Hypotension is not a disease; it is an ideal blood pressure level.

—LOUIS LAGER, '42.

HISTAMINE IN ANAPHYLAXIS AND ALLERGY

By LAURENCE FARMER

Bull. N. Y. Ac. of Med.; 16:618, 1940

The author reviews the evidence favoring the histamine theory of allergy and himself subscribes to it. He points out that in the treatment of allergic conditions there are very large numbers of possible allergens, and has introduced histamine phosphate as a possible common denominator to be used for desensitization of allergic persons rather than a specific allergen preparation.

He outlines the method and dosages used at Lennox Hill Hospital, and cites two cases successfully treated.

—WARD REASON, '42.

NON-SURGICAL CASES SIMULATING ACUTE APPENDICITIS IN CHILDREN

By HENRY L. HEYL

N. E. J. Med. 223:42, 1940.

This article deals with nine case histories each of which presents symptoms very similar to those of acute appendicitis. The cause of the symptoms in each case lay outside the peritoneal cavity. The final diagnoses on these cases were typhoid fever, measles, migraine, diabetic acidosis, allergic gastroenteritis, rheumatic fever, lobar pneumonia, sickle cell anemia and leukemia.

The author points out that, with the exception of pneumonia, pyelonephritis, and mesenteric adenitis, most non-surgical conditions simulating appendicitis are associated with a low white blood count. He warns, however, that the finding of a leucopenia does not rule out appendicitis.

Many of these differential considerations are seldom encountered in practice and most children with symptoms of appendicitis have it. The doctor should not be confused and should not make unusual diagnoses. However, the performance of an appendix operation is still attended by a considerable mortality and should be avoided where possible.

W. LOVEGROVE, '42.

Editorial

THE JOURNAL STAFF'S DILEMMA

There comes a time in the history of every organization when those responsible for its activity must stop and take stock. Our Journal is in just that position today.

In November, 1930, the Journal came into being through the efforts of Dr. J. P. Wells (now wearing the King's uniform), who gathered together enough money, by contributions from doctors in London, to print the first issue of the Journal. Since that time, the students have worked hard and enthusiastically, and as a reward for their efforts have seen the Journal grow steadily in circulation and, we feel, in popularity and usefulness. Today it can pay its own way. However, during the intervening years a debt amounting to \$600 has accumulated. The present advertising covers the cost of printing the Journal but will not pay off the debt. The most astounding fact is that, although there are 1,300 subscribers, only 65 paid their subscription fee last year. We know that the Journal is being read by many more than those who are paying their annual fee of One Dollar, because we are continually being asked when the next issue is coming off the press. The interest is there, but the money isn't!

The Journal staff and the undergraduate body acting under the guidance of the Board of Advisors are doing their level best to make the Journal well worth the subscription fee of One Dollar annually. We must have the co-operation of the subscribers in order to carry on. One Dollar is not very much. Will you send it now? It will mean a great deal to the Journal staff, not only for the opportunity to begin this year with a clean slate but for that warm feeling which comes from the knowledge that we have the support of the graduates in what we are attempting to do.

LAWRENCE RUTTLE.



NEOPLASTIC DISEASES

By JAMES EWING, A.M., M.D., Sc.D., LL.D.

*Professor of Oncology, Cornell University Medical College and
Consulting Pathologist, Memorial Hospital, New York.*

(Fourth Edition, 1160 pp., 581 illustrations, \$15.50. W. B. Saunders
Company, Philadelphia and London, 1940.)

The recent revision of this world-wide known book has been made all the more valuable since it has been carried out by the original author himself. Dr. Ewing, now more than three score years and ten, probably the greatest living authority on neoplastic diseases today, still maintains a very close and active connection with Memorial Hospital which, in its new quarters, is the largest institution for the diagnosis, study and treatment of tumors in the world.

It was many years ago that Dr. Ewing, with his keen foresight, realized that cancer, a peculiar and mystifying disease, should be dealt with by men specially trained in its diagnosis and treatment in institutions specially staffed and equipped. Dr. Ewing's tremendous wealth of knowledge, compiled after a long and intimate experience with every conceivable form of tumor growth, has been closely woven into his book. When one is confronted with an unusual tumor or a difficult problem in tumor diagnosis, one's first and natural impulse is to reach for a copy of Ewing's "Neoplastic Diseases."

This book is of inestimable value to pathologists in general and especially those chiefly concerned with tumor diagnosis and classification. It is also a very useful reference book for senior students with some clinical background and for clinicians particularly interested in the clinical course and prognosis of tumors. For the student approaching the subject for the first time, it would seem to be too detailed and complex a treatise for him to master. After he has gained more knowledge and some practical experience, he will come back to the book and appreciate it. The new sections bring the subjects concerned up to date. The book is profusely illustrated, almost entirely with photographs of a high standard.

—DR. J. H. FISHER.

NOMENCLATURE AND CRITERIA FOR DIAGNOSIS OF DISEASES OF THE HEART

By THE CRITERIA COMMITTEE OF THE NEW YORK HEART ASSOCIATION
(4th Edition, 282 pp. Published by N. Y. Heart Ass'n, 1940; \$2.00.)

This book was first published as an answer to the confusion which exists among physicians and students with regard to the nomenclature of cardiac disease and the criteria for the diagnosis of various cardiac conditions. Prepared by experts in their field, these criteria have been found extremely useful in the correct diagnosis of many cardiac disturbances.

Since the publication of the third edition, John Wyckoff, chairman of the New York Heart Association, has passed away, and it is to the memory of his good work that this latest edition is respectfully dedicated.

In former editions, only structural changes were considered to constitute heart disease, but in the 4th edition, functional disturbances are also included.

A new section is present which outlines the pathological diagnosis of cardiovascular diseases and anomalies. Throughout the outline synonymous terms are placed in parentheses in order that one may appreciate when a proper term is being used.

Numerous illustrations are scattered throughout the appendix—a feature somewhat lacking in other sections of the book. The diagnosis of functional capacity has also been revised. Formerly confined to cases only with organic heart disease, this section now also applies to those who may have only functional disturbance. This book is not recommended to those students who wish to depend on one text for their sole information as to cardiac disease. But for those who have read widely in this subject, it forms a useful, reliable and comprehensive summary of cardiac disorders.

—J. C. KENNEDY, '42.

FOETAL AND NEONATAL DEATH

By EDITH L. POTTER, M.D., and FRED L. ADAIR, M.D.

(198 pp., 31 illustrations. Published by The University of Chicago Press, 1940; \$1.50.)

The authors begin with an introduction and a first chapter so full of tables and statistics that the reading is heavy and dull. These tables, however, prove an excellent source of reference.

A very good review of embryology follows in which the authors

stress mainly the degree of development of the foetus at term. Since definite care must be exercised in the post-mortem examination of such small subjects, a somewhat special technique is described for autopsies.

After dealing with these topics in the first half of the book, the principal causes of foetal and neonatal death are listed and described. This is followed by an extensive chapter discussing special pathological conditions. These last two chapters are excellent but should, in the opinion of the reviewer, have come earlier in the work to sustain interest throughout.

A complete bibliography follows each chapter and numerous diagrams, illustrations and photomicrographs are interspersed throughout the volume.

The reviewer considers the work too detailed for easy reading but excellent as a reference text.

—L. J. CALVERT, '42.

SUBSCRIPTION FORM

UNIVERSITY OF WESTERN ONTARIO MEDICAL JOURNAL

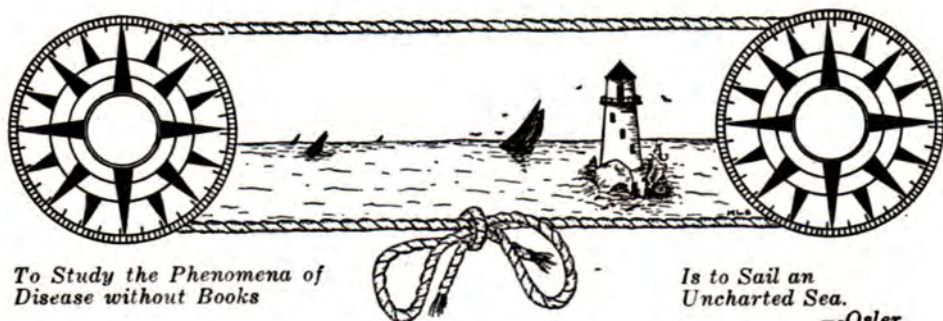
Please find enclosed One Dollar for One Year's Subscription to the
U. W. O. MEDICAL JOURNAL (issued quarterly).

Name

(Please print Name and Address)

Address

Fill in this form and mail with subscription to the University of Western Ontario Medical
Journal, 330 Ottawa Avenue, London, Ont.



*To Study the Phenomena of
Disease without Books*

*Is to Sail an
Uncharted Sea.*
--Osler.

RECENT ACCESSIONS TO THE MEDICAL SCHOOL LIBRARY

- Alvarez—An Introduction to Gastro-Enterology; 3rd ed. 1940.
 American Medical Association—Council on Medical Education—Graduate Medical Education in the United States, 1940.
 American Medical Association—Council on Medical Education. Medical Education in the United States 1934-1939. 1940.
 Ashford—A Soldier in Science. 1934.
 Bailey—Text-book of Histology; 10th ed. 1940.
 Barnes—Electrocardiographic Patterns. 1940.
 Bateman—Barkeley Moynihan - Surgeon. 1940.
 Bodansky and Bodansky—Biochemistry of Disease. 1940.
 Boyd—Pathology of Internal Diseases. 3rd ed. 1940.
 Boyd—Preventive Medicine; 6th ed. 1940.
 Brock—Injuries of the Skull, Brain, and Spinal Cord. 1940.
 Canadian Medical Association—Committee on Nutrition. Nutrition in Everyday Practice. 1939.
 Cannon—The Wisdom of the Body. 1939.
 Castle—Mammalian Genetics. 1940.
 Christopher—Minor Surgery; 4th ed. 1940.
 Dickson and Diveley—Functional Disorders of the Foot. 1939.
 Dill—Life, Heat, and Altitude. 1938.
 Evans—Evans' Recent Advances in Physiology; 6th ed. 1939.
 Ewing—Neoplastic Diseases; 4th ed. 1940.
 Fischer—Christian R. Holmes, Man and Physician. 1937.
 Frank Howard Lahey—Birthday Volume. 1940.
 Gellhorn—The Vasomotor System in Anoxia and Asphyxia. 1939.
 Grant—A Method of Anatomy; 2nd ed. 1940.
 Haden—Principles of Hematology; 2nd ed. 1940.
 Hauser—Diseases of the Foot. 1939.
 Hawes and Stone—The Diagnosis and Treatment of Pulmonary Tuberculosis; 2nd ed. 1940.
 Hill—The Histamine and Insulin Treatment of Schizophrenia and Other Mental Diseases. 1940.
 Homans—Practice of Surgery; 5th ed. 1940.
 Jones and Chamberlain—Electrocardiograms. 1939.
 Jordan—A Text-book of Histology; 8th ed. 1940.
 Katz—Electric Excitation of Nerve. 1939.
 Lewis—Research in Medicine and Other Addresses. 1939.
 Levine—Clinical Heart Disease; 2nd ed. 1940.
 MacCallum—Text-book of Pathology; 7th ed. 1940.
 Maher—Electrocardiography; 3rd ed. 1940.
 Markowitz—Text-book of Experimental Surgery. 1937.
 May—Manual of the Diseases of the Eye; 16th ed. 1939.

- Meakins—Practice of Medicine; 3rd ed. 1940.
Muncie—Psychobiology and Psychiatry. 1939.
Newsholme—Medicine and the State. 1932.
New York Heart Association—Nomenclature and criteria for diagnosis of Diseases of the Heart; 4th ed. 1939.
Osler—Counsels and Ideals from the Writings of William Osler. 1906.
Podolsky—Medicine Marches On. 1934.
Potter and Adair—Fetal and Neonatal Death. 1940.
Rogers and Muir—Leprosy; 2nd ed. 1940.
Rolleston and Ronaldson—Acute Infectious Diseases; 3rd ed. 1940.
Rooyen and Rhodes—Virus Diseases of Man. 1940.
Shohl—Mineral Metabolism. 1939.
Society of Chemical Industry, London; Food Group—Vitamin E; a Symposium. 1939.
Strecher—Beyond the Clinical Frontiers. 1940.
Virus and Rickettsial Diseases—A Symposium Held at the Harvard School of Public Health. 1940.
Von Diringshofen—Medical Guide for Flying Personnel. Tr. by V. E. Henderson. 1940.
Walters and Snell—Diseases of the Gallbladder and Bile Ducts. 1940.
Wechsler—The Measurement of Adult Intelligence. 1939.
Youmans—Essentials of the Diagnostic Examination. 1940.