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UNIVERSITY OF WESTERN ONTARIO

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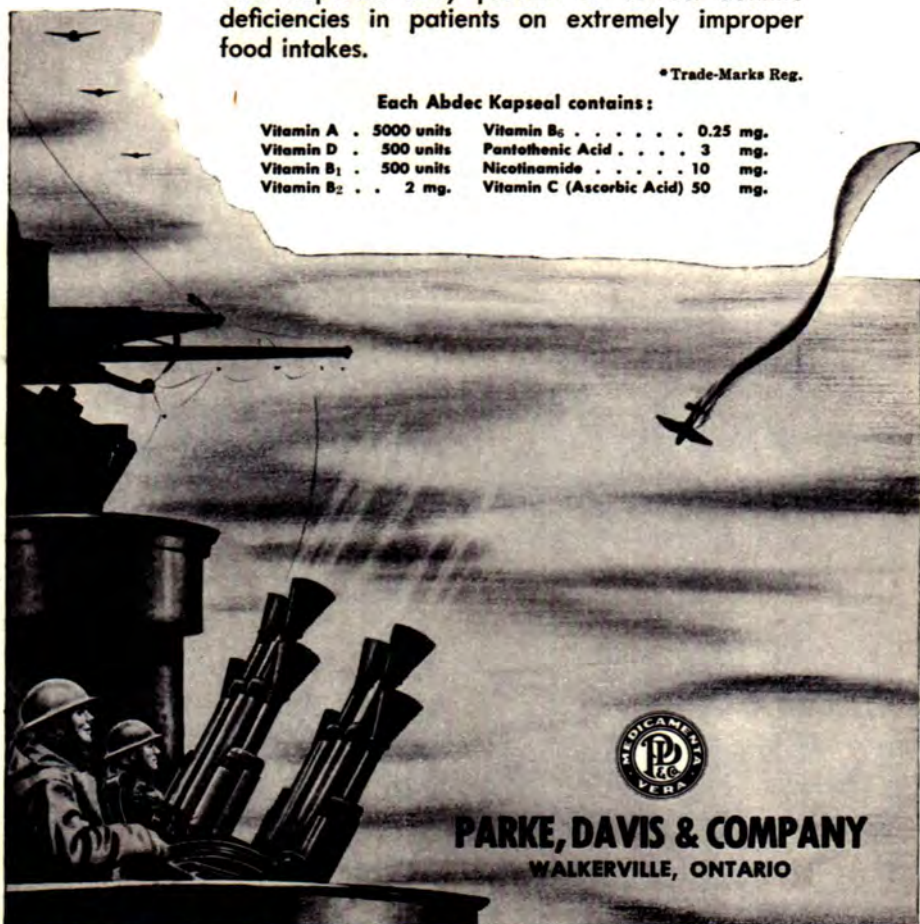
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The University of Western Ontario
MEDICAL JOURNAL

VOLUME 14

NUMBER 1

Present-Day Therapeutics

By HUGH GRANT SKINNER, M.D., M.A.

A REVIEW of the aims and status of therapeutics is timely for many a student finds himself in a confused state regarding treatment. He is twirled by this current and twisted by that — not understanding fully the meaning of any particular influence.

Therapeutics may be defined as the art and science of the treatment of disease. In its broadest sense, therapeutics includes all forms of treatment such as drugs, surgery, manipulation, physiotherapy, psychotherapy, etc. In this discussion our attention will be focused principally on drugs.

The ultimate goal of therapeutics is to be able to offer a cure for all diseases. At present we can only accomplish cures in a relatively few conditions; in others, delay the pathological process or afford symptomatic relief.

The student is continuously trying to assess the values of the courses outlined for him while in medical school. Therapeutics deserve scrutiny from this point of view. Therapeutics is based upon biochemistry, physiology and pharmacology, medicine and surgery, etc., and affords an excellent bridge between the scientific and clinical subjects. Because proper therapeutics can be practiced only by one conversant with the basic scientific subjects, the study of therapeutics gives the student an opportunity to consolidate the gains of his earlier work and fit them into his clinical training. In other words, therapeutics demonstrates the working method of science and teaches the application of these principles to the practical fields of medicine. Besides wishing to know the reason for having a subject in the medical curriculum, the student often asks "What good will this subject be in practice?" In the case of therapeutics, we can state that treatment to alleviate or cure has made and is still making great progress. The medically minded patients of today demand proper treatment and cannot be side-tracked with the pink medicine or placebo of our forefathers. A good therapist is demanded by the public.

The medical student is frequently confused by statements to the effect that the diagnosis is all important. Certainly the proper diagnosis

is important, but it is only the beginning of our obligation to the patient. It is an accomplishment to make a diagnosis but to fail to treat is an indication of an incompetent doctor. Both diagnosis and treatment are essential. It is as hard to decide which one, diagnosis or treatment, is most essential as it is to decide whether the egg or the chicken came first. A good diagnostician who understands the syndromes that respond to drug therapy should be a good therapist. As an example, a diagnostician who can diagnose heart failure in its earliest stage and apply digitalis therapy can accomplish more than another practitioner who has to wait until the clinical picture of failure is more advanced before he recognizes and treats the condition.

Therapeutics brings the doctor into relationship with the druggist. The druggist or pharmacist is trained to prepare and dispense the drugs ordered by the physician in a prescription. There are difficulties involved in this relationship that the student should be aware of so that he may meet and overcome them. Druggists may be critical of the lack of pharmaceutical niceties in the doctor's prescriptions. Although it is not the druggist's duty to suggest treatment, it is important that the physician make use of the druggist's superior knowledge in compounding prescriptions. The importance of color, taste and smell in medications has not been stressed enough. Certainly every doctor should cooperate with the pharmacist in improving the medications he prescribes.

A service the druggist renders to the doctor is in his attitude toward the patient when he gives the patient the medication prescribed. The pharmacist reassures the patient regarding the medicine and regarding his choice of physician in such a manner that the patient will remain under the doctor's care until the drugs had an opportunity to accomplish their task.

A pharmacist has an excellent opportunity to practice medicine for many people believe he is skilled in medical lore. Counter prescribing by the druggist is wrong. If a druggist refers a patient to the doctor it is common decency to instruct the patient to have the prescriptions filled by the referring druggist.

The doctor has a power over the druggist that is exerted without realizing how much money it may involve. If the doctor orders one particular trade-marked tablet the druggist is obliged to dispense only the drug made by the company who possess that particular trade-mark. This leads to complications for a druggist may have to stock dozens of brands of one drug. The drugs of all companies have to come up to the same B.P. standards. If the drug is synthetic, the product of all the companies is the same. The trade name quandry forces the pharmacist to carry thousands of the same tablets when a few hundred would make for efficiency and good treatment. The druggist's greatest criticism

comes when the doctor orders some newly detailed product a few times then forgets it, leaving a stock of an expensive mixture to deteriorate on the pharmacist's shelves. The lesson to be learned is to order B.P. or Non-Official products when possible and to make certain you have a place in your therapeutic plans for a company's specialty before you ask the druggist to stock it.

The modern pharmaceutical manufacturing house through their representatives, the drug travellers and their advertising campaigns, exert a tremendous force in moulding the therapeutic practice of the physician. It is important that the student understands this influence. There are a few aspects of this relationship that appear wrong. In the first place, the trade-mark name difficulty previously mentioned may be partly the companies' fault. The representative does all in his power to have the physician write the order for the trade name of a product instead of the official name. If a manufacturer's name means anything it should be enough to state B₁ (Thiamin Hydrochloride) manufactured by _____ and not compound some high-sounding name for confusion's sake. The pharmaceutical companies reply that the other fellow does it so we have to do the same.

The second criticism has to do with the mixtures, such as cough remedies, tonics, sedatives, etc., that are manufactured by the pharmaceutical companies. Most of these combinations are composed of B.P. or U.S.P. drugs with excellent vehicles. The point is that doctors should be able to write prescriptions that will rival the most desirable preparations. This is the place where the druggist and physician can coöperate to their mutual benefit. The mixtures of the pharmaceutical houses are good but the doses are fixed and that is a disadvantage. The other point is that the patient, often more observant than we think, soon spots the special red medicine of Dr. X and, being wise, eliminates the doctor and treats the anaemia of all his friends with "the special red medicine that did so much for my anaemia". The pharmaceutical houses argue that is the doctors' fault. There is a demand for cough, tonic and sedative mixtures. They say we are business men and we fill a demand. The moral is to learn to write varied and good prescriptions for such mixtures.

The advantages of having pharmaceutical houses are numerous. These companies put out excellent standardized products and do much to supply our demands for uniform and potent drugs. They spend large sums in research and have contributed a great deal to the advancement of therapeutics. Many of these manufacturers publish journals for practitioners. In these periodicals can be found reviews of the literature on treatments and new drugs. Most of these journals contain well-written discussions of treatment that are useful to the busy practitioner.

A few points regarding the ideal therapist will give us an idea

of what standards a student can aim at. The ideal therapist must possess common sense; the ability to apply his knowledge of physiology, biochemistry, pharmacology, psychology, etc., to the treatment of disease. He will realize that treatment is dynamic, therefore changing. He will be aware of the fact that a good deal of his knowledge of drugs must come from the expert. The type of research he can carry out in his daily practice is limited to careful observation of drugs previously introduced by pharmacologists and experimental clinicians. He must be critical of all new treatments and insist that reliable experts accept them before he uses them on his patients. He should prescribe drugs only where they will be of value. In other words, he must study the syndromes that respond to treatment, although many times a therapeutic test is legitimate. He must understand psychology and realize that drugs are less effective in functional disorders than psychotherapy. He will be in touch with humanity for no therapist can do his best for his patients if he does not understand both the patient and himself. He becomes a philosopher for a good therapist has a unique opportunity to study human nature. His success affords him happiness. His failures teach him humility. The ideal therapist is a doctor at his best.

A Fatal Case of Acute Rheumatic Fever

By K. ERIC ROGERS, '44

AN obese forty-one year old white female was admitted to the hospital on January 28th complaining of fleeting joint pains, associated with sweating, malaise and weakness of ten days' duration. During the four days prior to admission, the patient developed swelling of the ankles and hands, dyspnoea and fever.

The patient's complaints began during a period when her fourteen year old son was suffering from scarlet fever. Four days before admission she became dyspnoeic and experienced left chest pain which extended into the axilla. This pain was increased by deep inspiration and coughing. She developed a non-productive cough.

Functional inquiry with particular reference to "growing pains", rheumatism and epistaxis during childhood was negative. The patient stated that she had "inflammatory rheumatism" at 37 and 39 years of age. Slight dyspnoea on exertion and occasional ankle oedema were present prior to the present illness.

Physical examination revealed a weak female patient perspiring profusely. There were tender swollen joints and pitting oedema of the ankles. The thyroid was diffusely enlarged. The neck veins were engorged. Dullness was present in the left chest extending from the third rib anteriorly and fourth rib posteriorly to the base. The apical impulse was 13.5 cm. from the midsternal line. The apical first sound was loud and snapping. The heart rate was 120 per minute. On auscultation, the heart sound P1 was soft, P2 loud and reduplicated. In the aortic area, A1 was increased while A2 was softened and prolonged. Blood pressure was 132/80 mm. hg. The abdomen was soft but tender in the right and left upper quadrants. There was no hepatomegaly or splenomegaly. There was one plus pitting oedema of the arms and legs. No subcutaneous nodules, purpuric or erythematous lesions could be demonstrated.

The provisional diagnosis was bronchopneumonia and rheumatic heart disease. There was no response to a three-day course of sulfadiazine with a blood level of 12.5 mgm. per cent. The temperature remained constantly above 102° during the first week and above 100° the second week. The pulse rate ranged from 110 to 150. The urinalysis was negative. The day following admission the blood pressure dropped to 88/50. The white blood count on January 28th was 15,200 per cu. mm., on February 3rd 20,350 and on February 11th 34,350. Blood Wassermann was negative. Other laboratory investigations included

blood culture and throat culture which were negative. The sedimentation rate was elevated. On February 8th the total plasma proteins were 4.0% with an A.G. ratio of 0.37.

X-ray examination of the chest on January 31st showed a left-sided pleural effusion with diffuse pneumonic infiltration of the lung parenchyma on the right side. On February 5th 50 cc. of bloody pleural fluid, which gave no growth aerobically, was drained from the right chest. A pericardial tap on February 8th was unsuccessful. X-ray of the chest on February 10th revealed bilateral pleural effusion.

On February 12th the peripheral oedema was increased. A loud pericardial friction rub was noted but no murmurs could be detected. Oxygen was administered as cyanosis was marked. The patient pursued a downhill course and died in congestive heart failure on February 13th, seventeen days after admission.

DISCUSSION

A survey of the literature reveals the rarity of fatal attacks of acute rheumatic fever in adults. Judging from the patient's history, her first attack of rheumatic fever was four years prior to her fatal illness. Ball¹ states that initial attacks of rheumatic fever in adults is rare. Recurrences of the disease are less severe and less frequent among adults than among children. Cohn and Lingg² concluded that the later the disease begins the fewer are the patients who exhibit carditis, and the greater becomes the number without manifest infection. After the age of thirty, the number of severe cases is relatively small, regardless of when the disease was acquired.

This case bears out Osler's³ assertion that acute rheumatic fever rarely proves fatal, except with complications such as pericarditis, pleurisy or pneumonia. Miller⁴ points out the recognized fact that in the adult type, arthritis is prominent, and the heart and brain tend to be relatively spared. In adults an acute attack usually develops rapidly with slight warning symptoms.

PATHOLOGICAL REPORT

Heart—Gross: On opening the pericardial sac which was thickened and markedly adherent to the right ventricular wall, about 250 cc. of cloudy, bloody fluid was found. Numerous fine "bread and butter" adhesions appeared to be superimposed upon a more chronic cor villosum type of adhesions. The entire heart was hypertrophied and in the left ventricle there was a superimposed acute dilatation. Diffuse thickening of the leaflets involved the aortic, the mitral, and the tricuspid valves. In addition, all three valves exhibited small warty vegetations spread along the line of closure. McCallum's patch was evident on the left auricular wall above the posterior cusp of the mitral valve.

Microscopic: The myocardial fibres were moderately hypertrophied, palely stained, and edematous. There was a widespread diffuse and focal infiltration of inflammatory cells, mainly monocytes, also plasma cells and lymphocytes with numerous multinucleated Aschoff cells, especially in relation to the vascular system. Well defined Aschoff nodules were scarce. Scattered throughout the myocardium were areas of actual muscle necrosis, and about these areas cellular infiltration was most marked. Definite areas of hyaline degeneration were present in the auricular wall, which also contained small foci of polymorphs in addition to the monocytic infiltration. The gross hypertrophy of the heart and the histological picture suggests the pericardial involvement is much older than the vavular and mypcardial lesions. *The diagnosis is acute rheumatic pancarditis.*

SUMMARY

1. A fatal case of acute rheumatic fever in a forty year old female is presented.
2. The rarity of this condition in adults is emphasized.

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²Cohn and Lingg: The Journal of the American Medical Association; Jan. 9, 1943.
³Osleis Principles and Practice of Medicine.
⁴Miller: The British Encyclopaedia of Medical Practice.

* * *

(The author of this paper would like to express his appreciation to Dr. L. D. Wilcox of the Department of Medicine and to Dr. E. P. Johns of the Department of Pathology for their helpful suggestions in the publication of this report.)

Immersion Foot

By A. C. JOHNSON, '44

"**I**MMERSION FOOT" is the name used to denote a condition produced by long immersion of the feet in extremely cold water, usually associated with immobility of the limbs and constriction by boots and clothing, and characterized by painful swelling of the feet and lower limbs.

Although old records, both medical and non-medical, make no mention of immersion foot, an essentially similar syndrome known as trench foot has been recognized for some time. The first account of trench foot was written by a surgeon-general of Napoleon's army in the winter retreat from Moscow in 1812. Later, in the Crimean War, accounts say that the casualties from trench foot alone, were estimated to be in the thousands.

The trench fighting of World War I brought the problem of the cause and prevention of trench foot to the foreground again. The only exciting cause that could be suggested at the end of 1914 was the soupy mud of the trenches and its infective nature. An article appeared in the British Medical Journal, ascribing a microbic cause of this syndrome.

In addition to "trench feet" there occurred an analogous condition on the anterior surface of the lower leg which has been designated "trench skin" and also "trench knee" which occurred in the kilted Highlander.

Finally, as a result of careful observations and experimentation, it was determined that the exciting cause was continuous immersion of the feet for hours or days in cold, semi-liquid mud or water, with fatigue, immobility, and constriction of the limbs. The puttee was blamed, and rightly so, for causing unnecessary constriction of the limbs on account of its tendency to shrink on immersion in water.

Later, in peace time, trench foot occurred in some of the personnel of a Mt. Everest expedition and in one of the survivors of the Moose River Gold Mine tragedy in April 1936.

With World War II came mechanized warfare and the abandonment of static trench fighting. But as a result of enemy submarine action many survivors of torpedoed vessels have been exposed to unusual physiologic conditions with the production of immersion foot.

Immersion foot has not heretofore received adequate recognition, although it must have occurred after torpedoings in the last war and after shipwreck in northern waters since time immemorial. Perhaps some of the peg-legged sailors owed their disability to this syndrome.

Attention was first focused on this syndrome by the Medical Corps of the Royal Navy after the loss of a ship off the Norwegian coast in the late spring of 1941. The ocean temperature was around 40° F. A number of the crew were adrift for several days on a type of raft that forced the occupants to keep their feet immersed in cold water. After the survivors were rescued by trawlers (which carry no medical officers) the mistake was made of rapidly warming their numb and swollen feet before a stove. As a result, a number of these men developed gangrene, which necessitated amputation.

It was claimed by some observers that a similar picture was found in survivors landed in Virginia after drifting in the Gulf Stream where the water was between 60 and 70° F. This idea has been largely discredited in an article by White, U.S.N.R. It was fundamentally quite different from immersion foot for there was no evidence of direct cutaneous injury. Immersion in warm water per se cannot cause either edema or pain in the legs and feet. Hypoproteinemia and vitamin deficiency from starvation were considered to be largely responsible. Thus the term "immersion foot" is inappropriate in this connection and should be given up.

Causation, Incidence and Predisposing Factors

Immersion foot develops when the limbs have been soaked for many hours in cold water or mud at a temperature insufficient to freeze them. Sea water, cold enough to do the damage, is found in the North Atlantic both winter and summer.

Sea water freezes at -1.9° C. and tissues do not freeze until their temperature falls to -2.5° C. or less, so that parts immersed cannot be frostbitten.

Judging from the patients' histories, the following factors seem to influence the occurrence and severity of immersion foot:

- (1) Time of exposure and temperature of the water.
- (2) Footwear afforded some protection during short exposure. During long exposure, however, boots constricted the swelling feet and impaired their circulation. A booted foot often suffered more than one from which the boot was lost. In rare cases, where men had on loosely fitting rubber hip boots were they able to avoid this condition. Recently Commander White, U.S.N.R., had the opportunity of examining the crew members of a torpedoed vessel after three days adrift in cold water. Three of these men were protected by a new type of rubber lifesuit and suffered no thermal injury.
- (3) Immobility: The legs were usually dependent and immobile as a result of overcrowding. Men who kept moving suffered less than those who sat still. An airman exposed for 14 hours in a rubber raft,

holding tight to the centre rope, had more damage in the hands than in the feet, although both were immersed.

(4) Body cooling reduced the peripheral circulation.

(5) Seasickness and starvation may be contributory.

(6) Age: Men over 40 and under 17 died from cold sooner than those of intermediate age.

(7) Race: Some have observed that those who suffered most were Greeks, Australians, Negroes and those employed in engine-room or stokehold duties. This suggested that those accustomed to a warm environment may not have the same defences as those who have been exposed to colder climates.

(8) Morale: Those who gave up hope died more quickly or suffered more severely than their companions.

(9) Treatment on rescue has a definite bearing on the severity and course of the condition.

Clinical Features

During exposure, the immersed limbs soon became numb. Some patients described walking "as if on cotton wool". Pain, tingling and itching were unusual. Cramps sometimes occurred, usually in the calves. Swelling of the bare feet was noticed after some hours or days or otherwise not until the boots felt tight. The skin, red at first, was later pale, mottled blue or black.

The symptoms and signs observed in the affected limbs after rescue may be described in three stages:

1. The prehyperaemic stage: This stage lasts from a few hours to several days. The extremities remain cold, somewhat swollen, discoloured and numb with extensive stocking or glove anaesthesia. Peripheral arteries may be pulseless for some hours, and remain so in cases going on to gangrene.
2. The hyperaemic stage: This stage lasts from six to ten weeks. The typical findings are vascular disturbances, swelling, sensory and motor disturbances, absence of sweating, blisters, ulcers and gangrene. Each of these findings will be considered.

(a) Vascular disturbances: the skin of affected parts remain hot and red. The parts become congested when dependent and blanch rapidly when elevated. Blanching from finger pressure disappears quickly except in parts destined to become gangrenous.

(b) Swelling: often there is increasing swelling which pits on pressure. With rest it subsides in three to four weeks.

(c) Sensory disturbances: Tingling is felt early and often lasts for six weeks. Affected parts ache and throb. Pains may occur as sharp stabs and are made worse by warmth, dependency and exercise. There is loss of sensation to pain and temperature with glove, sock or slipper distribution.

(d) Motor disturbances: Muscles in the cooled area may show weakness and impaired electrical response.

(e) Absence of sweating: The involved area coincides with that of the sensory loss.

(f) Blisters, ulcers, and gangrene: Blisters may appear during the first three days after rescue or not until a week has elapsed. Healing takes three to six weeks or longer. Parts which are to become gangrenous blister extensively.

2. The post-hyperaemic stage: This stage may last for weeks or months after the hyperaemia has subsided. There may be a cold-sensitive state, giving rise to Raynaud's phenomenon. Swelling of the feet may recur when walking is resumed. After a number of weeks there are complaints of excessive sweating of the feet.

Course and Prognosis

The mildest cases in a series of eighty recovered completely in two to five weeks. Those which passed through all three clinical stages recovered between three to twelve months. A follow-up for two years in eighteen moderately severe cases showed that seven were doing full duty, two had been invalided and the rest were doing light duty.

Pathogenesis

Coldness of the affected parts in the prehyperaemic stage indicates narrowing of arteries or arterioles. That large arteries are narrowed is shown by the absence of peripheral pulses. This narrowing is due to the action of cold, direct or through the nervous system. Intimal changes in the vessels may lead to thrombosis.

The excessive vasodilatation of the hyperaemic phase may be due to inflammation, damage to vessels or vasomotor paralysis. The inflammatory response to damage by cold has been verified by the work of Lewis. The damage to vessels explains the edema.

In the posthyperaemic stage the skin may become cooler before reflex vasomotor activity is restored. Most of the cooling is probably due to a lessening of inflammation.

The Wallerian degeneration, observed microscopically in the nerve trunks of affected limbs, is probably due to the combined effect of cold and ischemia. Blistering of the skin must be regarded as a reaction of

the cells and small blood vessels of the skin to damage by cold, although maceration of the skin by imbibition of water may play a part.

TREATMENT

Prophylaxis:

1. All seamen should be instructed in the dangers of exposure to cold air and water.
2. Waterproof, loosely fitting shoes and extra pairs of dry socks should be obtained.
3. Wet and cold boots should be discarded.
4. The legs should be moved frequently and kept elevated. Constricting pressure on the limbs should be avoided.

First Aid:

1. The patient must be carried and not allowed to walk.
2. The aim is to warm his body and to keep his extremities cool. Massage and heat are counterindicated.
3. The feet should be elevated.
4. Codeine or morphine is used when indicated.

Hospital Treatment

The rationale of treatment in the hyperaemic stage is to reduce tissue metabolism to a point where there is an adequate supply of oxygen to all the cells. This can be accomplished by cooling the skin to a point below 85° F. Lake, in 1917, first suggested treatment by continued cooling in trench feet.

Dry cooling is more satisfactory than wet for the moisture produces maceration. Dry cooling is achieved by the application of ice-bags, exposure to a fan, exposure to the air at room temperature or cooling in specially designed therapeutic cabinets.

On a theoretical basis, White advocates a period of Buerger's exercises prior to getting the patient out of bed. Another measure which may help in the early stage of edema and neuritic pain, particularly after prolonged starvation, is a diet high in protein and vitamin B. White also suggests that pressure dressings may assist in the control of edema.

SUMMARY

1. Immersion foot, secondary to exposure to cold and wet, appears to be sublethal injury to the chilled tissue cells, the cutaneous arteriolar and capillary bed, and the nerve fibres.

2. The clinical course may be divided into the prehyperaemic, the hyperaemic and the post-hyperaemic stages.
3. Treatment is directed primarily at reducing tissue metabolism by dry cooling and at preventing further injury and infection in the feet.
4. The majority of patients respond favourably to proper treatment and suffer no serious sequelae.

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The Tools of Our Craft

By RAY BAINBOROUGH, '46

"BOOKS," said Harvey Cushing, "are the most important tools of our craft when assembled in mass in our great medical libraries." It follows, therefore, other things being equal, the standing of a medical school is proportional to the usefulness of its library, for we are reminded of the adage "A workman is only as good as his tools." The usefulness of a medical library is dependent on many things. The number of books and periodicals shelved in the library and the number coming in currently are the first of these that come to mind, but these are not by any means the only important factors. Of what use would all the tools of the kingdom be if they were kept behind locked doors and could be removed only on request, one at a time and for only brief periods? Of what use would a medical library be if every time one wanted a journal or textbook, or even a dictionary, one had to produce credentials as to one's character, get the recommendation of an outstanding physician and fill out an application form in triplicate? The availability, then, of books and periodicals to the student, practitioner and research worker is of major importance. Besides these, the completeness of indexes, the reading-room space, lighting, position of the library with respect to the medical school and hospital and the arrangement of journals and textbooks add to or subtract from the usefulness of the library.

The story of the evolution of our medical libraries is hazy and their development has been discontinuous. The flood-lighted, highly-indexed twentieth century medical library is a far cry from the first libraries in the dim temples of ancient Egypt. A multitude of guinea pigs have died the martyr's death since the first real collection of scientific literature was accumulated by Aristotle. Myriads of tubs of printer's ink have been spilt since paper was first used in Europe for making records, a step which was of as great importance as the invention of printing. What a deal of spinning the kymographs have done and what a lot of pulses the clinicians have counted between those days and the present, when we have such libraries as the Army Medical Library with its catalogued million volumes and pamphlets. The libraries of medieval universities seldom exceeded a hundred volumes. Indeed, a long stride separates the handful of books possessed in the early days by the Medical Department of the College of Philadelphia (the first medical school established in America) and the 30,000 volumes shelved in our own medical library.

Of the several categories into which medical libraries can be grouped, the medical library of U.W.O. probably falls into the class of university, research and college libraries. It is comprehensive in scope,

serving the specialized needs of the research workers and medical history scholars as well as catering to the generalized needs of practitioners and students. Unlike our Arts library, the textbooks in the medical library are kept separate from the journals, else the former would be lost and smothered by their more numerous, more lively and more productive kin. The textbooks occupy only a quarter of the space on our shelves.

The impressive total of twelve hundred titles of journals are found in the medical library. Four hundred and eighty of these periodicals were streaming in currently in pre-war days. The number is slightly less now because of the ligature placed around the foreign journal channels by the war, but this is partly made up by the new inflow of military medical journals. Complete files of 340 journals are snugly stacked. So much for the bulk of the library.

No system, which might still be called a system, could give students and staff alike greater freedom. Harvey Cushing once wrote, "I have always felt that a hospital or medical school that wished to cultivate a scholarly spirit must have its library on the ground floor." And there it is as accessible as the olden "pub", and once inside you are a victim, for there is no immunity here. Once infected by the "run to the books" habit, always infected. Another generalization is, the younger the individual the more susceptible. "To inoculate a doctor with the library habit he must be caught young," said Harvey Cushing, and I do not apologize for again quoting him, because he was a man interested in libraries.

There is ample bait to tempt the most diffident medically-minded man, for all the stacks are open. Even the historical room, wherein is kept the collection of old texts along with biographies and other inspirational books, is accessible to everyone for the asking. Here one can find the first edition (1892) of Osler's "Principles and Practice of Medicine" and its younger brothers, all ancestors of the red and somewhat fatter most recent edition on the reserved bookshelf. Other old classics have assembled there, but the books which hold most browsers spellbound are those which relate the exciting adventures into the misty unknown of Fabricus and Malphigi, Da Vinci and Harvey, Priestly and Galvani. Where is there more inspiring reading than in the stories of the careful plodding of Leeuwenhoek and Koch, the lucky guesses of the wizzardly Pasteur, the wild stampedings of Metchnikoff? Nothing can equal these as appetizers for original work and the tales of all these men have crept into the historical stacks and are there at everyone's fingertips.

The circulation of some 20 thousand volumes last year is evidence of this availability. Telephone calls, both local and long distance, amount to one hundred a year with the number growing rapidly. More than 400 people enter the library weekly and last year 457 other than

students and staff members used the library. A considerable increase in the use of the facilities of the library has been observed since the beginning of the war, due to the speeding up of the medical course, war research and the use of the library by the medical services of the armed forces.

Of special significance is the large number of indexes to be found in the library. Indexes which are in our library but which are not common to other medical libraries are: (1) "War Information", which keeps up-to-date new references not yet indexed in the Cumulative index, (2) a file containing a record of recent symposiums and of progress of work complete to date on the various specialties, (3) a serial list of publications on file in the library, giving details of periodical holdings, whether or not the periodical is received currently, and if the file is complete to date, (4) a card index containing the location of portraits in the library, (5) the "Where-to-look Index", (6) Index of publications of the faculty and alumni (not yet completed), and (7) a list of war publications, old and new and of current medical military periodicals.

Besides this rich supply of indexes, the library has one or two other special features worthy of mention. There are compiled in book form newspaper clippings since 1921 covering some forty subjects. These are clippings dealing with everything from cancer research to the progress of the Dionne quintuplets and from C.O.T.C. activities to doings in Victoria Hospital, St. Joseph's Hospital, Westminster Hospital and the Queen Alexandra Sanatorium. Here lies an opulent source of information for the medical historian. Much interest has been shown also in the displays in the reading room, where all the recent literature on timely topics is gathered together. Nor must we forget to mention our collection of book plates which might well be the pride of any specialized library.

This, in brief, is our medical library. In conclusion, may I again quote the late Harvey Cushing, whom I feel would not have hesitated to refer to our medical library, as he did once in speaking of another library, as a library made useful not as a passive but as an active force; one that is "not vocational but cultural, not final but initiative"; one that will serve as a common meeting ground, where the different streams of knowledge may coalesce; one where an interest in the history of our great profession will so flourish so as to permeate into all departments of a much divided school; a place from which the appeal of scholarship free from pedantism will radiate to long generations of future students—a place, in short, where Medicine, the foster mother of the sciences, once more in close contact with her whole family, will imbue them all with the spirit of that ancient phrase, "Where there is love of humanity, there will be love of the profession."

Newer Method in the Treatment of Phlebothrombosis and Thrombophlebitis

By L. G. STEVENSON, '44

THERE are two major types of intravascular thromboses, namely, thrombophlebitis and phlebothrombosis. "In the former, the clotting is believed to be the result of injury to the vascular endothelium from mechanical trauma, bacterial invasion or chemical injury, whereas in the latter it may be due to venous stasis and to alterations in the cellular and fluid constituents of the blood which increase the clotting tendency. The clinical significance of this distinction lies in the fact that in thrombophlebitis the clot is usually firmly adherent to the vein wall and is, therefore, less likely to become detached and result in embolism. In phlebothrombosis, on the other hand, the thrombus is loosely attached to the vessel and is, therefore, more liable to cause embolism."¹

This distinction, stressed by Ochsner and his associates at Tulane University, is given little weight by Jacob Fine and other Harvard surgeons. Dr. Fine says: "With reference to Dr. Ochsner's distinction between thrombophlebitis and phlebothrombosis, I think . . . that from the point of view of embolism this distinction is not helpful clinically, for we have seen embolism in individuals with outspoken thrombophlebitis."

Treatment

(1) **SULFONAMIDE DRUGS:** The sulfonamides have been used in conjunction with classical treatment, or some other form of therapy with a view to combatting the infective element believed to be an important ætiological factor in thrombophlebitis. Phlebothrombosis does not seem to be an indication for chemotherapy, but the distinction must often be difficult.

(2) **ANTICOAGULANT THERAPY:** (a) *Heparin*—The application of this agent as a powerful anticoagulant has been extensively reported in literature. Its therapeutic value, therefore, covers the field wherein thrombus formation must be prevented, and its contraindications include all those conditions where hemorrhagic tendency already exists or is anticipated. One of the larger fields of heparin therapy is encountered in spontaneous and post-operative thrombosis. While evidence of its efficacy is accumulating, it remains difficult to establish criteria in the presence of so unpredictable a complication as embolism from thrombotic fields. This is true of post-operative episodes as well

as those occurring in the course of both thrombophlebitis and phlebotrombosis. To establish premises in these conditions, cases must, therefore, present doubly convincing evidence.

TECHNIQUE OF ADMINISTRATION: The object of this treatment is to obtain over a considerable number of days a sustained clotting time of between 15 and 20 minutes. Since the action of a single dose is said to become evanescent in about four hours, continuous intravenous therapy is the method of choice. The length of administration is a matter of judgment. It appears that in most instances, from seven to fourteen days' treatment are required to tide the patient over a critical period, or in other instances to permit drug action to become efficacious without the interference of fibrin formation. A technique lately proposed is designed to incorporate the essential requirements:

Start with 1,000 cc. normal saline intravenously, at the rate of about 40 drops per minute. After this solution has run for a few minutes add six 5 cc. vials of heparin per 1,000 cc. of saline. After four hours, determine the clotting time; repeat this test every four hours until the clotting time is fifteen minutes — if it reaches twenty minutes or more, reduce the dose of heparin. On the other hand, when the clotting time is less than fifteen minutes add 5 cc. of heparin per 1,000 cc. of saline.²

The use of heparin has certain obvious disadvantages. Since there are no reliable earmarks by which to identify individuals susceptible to thrombosis, the routine use of heparin for successful prophylaxis against thrombosis would make the trouble and expense involved prohibitive. Its use for prophylaxis against embolism in patients with recognizable thrombosis or with nonfatal embolism may have real value. But heparin will not dissolve a thrombus, so that when it is discontinued, a propagating clot may form at the site of the thrombus or a new thrombus may form. There is no way of knowing when the drug may be discontinued safely. Bleeding or hematoma formation at the site of operation and elsewhere may result from its use. It is believed to provide an inadequate safeguard against embolism, since embolic phenomena have been known to take place during the course of heparinization.

(b) *Dicoumerol* — Jörgen Lehmann, in Gothenburg, Sweden, has reported extensive trials of this anticoagulant, which was first obtained from spoiled sweet-clover hay and which he calls "AP" (antiprothrombin). It was given orally to 100 cases of established venous thrombosis in the lower limb (by which he apparently means phlebotrombosis) and 32 cases of thrombophlebitis.

At the first sign of thrombosis 0.5 g. of AP was given by mouth. The prothrombin index was followed daily and kept within the limits of

20-60 by controlling the dose. A too rapid return of the prothrombin index to normal should be avoided because of the risk of a recurrence of the thrombosis. Medical treatment was combined with active movements of the leg in half the cases. Movements of the ankle were allowed from the first day, and after a few days the patients were also allowed to move the knee, provided there was no pain or other contraindication. In cases of thrombosis of the femoral or pelvic veins, movements of the knee were begun after 6-8 days or when the local pain had about gone.

The effect of AP was estimated by noting the period between the beginning of treatment and the time when the leg was flaccid and painless with diminution in swelling. The time for recovery ranged from 1 to 3 weeks. The mean duration of treatment of thrombosis in Sweden before anticoagulants were used was 5-8 weeks. Active movements shortened the time for recovery significantly. In 2 cases embolism occurred during treatment. All patients survived.

There is a well-marked individual variation in response to AP, so that the dosage must be adjusted by observation of the prothrombin index of each patient. The sedimentation rate of the red cells should be followed and the treatment continued until the rate is below 60 mm. in an hour, provided it is not raised as a result of other diseases. AP treatment can be continued for 4-6 weeks without any complications, if the prothrombin index has not been kept too low.

The dosage of AP adopted in thrombophlebitis was the same as for thrombosis. Mean recovery time was 5-6 days. No difference was noted with and without movements. AP combined with sulfathiazole proved no better than AP alone.

The anticoagulant was also given prophylactically to 170 patients after various gynaecological operations during 1942. Only one case of venous thrombosis and one mild case of embolism were seen. There were no deaths from these causes. In 1941 there had been 9 cases of thrombosis and 5 of embolism, with 3 deaths in a comparable series. In about one case in ten hemorrhage occurred as a complication but in only one in a hundred was it at all profuse, and in these it was readily controlled with vitamin K and blood transfusions.

Only operative cases known to be disposed to thrombosis were selected for treatment. On the day after operation 0.5 g. of AP was given, and the prothrombin index was then kept between 30 and 60, with subsequent doses of 0.25 g. In most cases the second dose was given 6 days after operation. Usually only three doses were required, making a total of 1 g.³

James A. Evans of Boston says that dicumerol is still experimental and that in his hands it has proved too erratic and too dangerous. "In 25 cases," he writes, "I have had one death, one barely saved from death by repeated transfusions, and two other minor hemorrhages. The doses used in these patients who suffered hemorrhages were no greater and were often less than in the patients who failed to respond with prolongation of conglutation time. An intravenous salt of dicumerol given once in 24 or 48 hours may prove more reliable by avoiding the element of variable in absorption." Evans does not record whether or not his technique involved frequent determinations of the prothrombin index and its maintenance near a certain definite level.⁴

(c) "*Thrombocytopen*": This crystalline substance, recently isolated by H. G. Skinner from beef spleens, is capable of lowering blood platelets, prolonging bleeding time, eliminating clot retraction and increasing capillary fragility. This work has not yet been reported.

The fact that the increased platelets are an important factor in the production of post-operative and post-partem thrombophlebitis and embolism led to the attempt to lower the increased platelet count of post-operative patients. This lowering was accomplished. Final proof of the efficiency of "*thrombocytopen*" in the prevention of embolism awaits further investigation.⁵

3. PARAVERTEBRAL SYMPATHETIC BLOCK: This form of treatment was first described by Leriche and Kunlin, of France, in 1934. They reported three cases of acute post-operative phlebitis in which treatment by novocain block of the lumbar sympathetic ganglia was successful. Since 1934, this method has been used successfully in Europe by Aufrere and others, while credit for its further clarification belong to Ochsner and De Bakey.

According to Leriche, clinical manifestations in thrombophlebitis are due to the establishment of a vasomotor reflex as a result of impulses originating in the thrombosed venous segment. He is of the opinion that there are three dominant factors in this process: (1) Amount of vein involved; (2) arteriospasm; and (3) venospasm, the latter being most constant and significant. Ochsner and De Bakey, after much clinical and experimental work, feel that arteriospasm is of equal or more importance than venospasm. That a localized thrombophlebitic process can initiate a marked arteriospasm is illustrated by numerous reports in the literature in which the onset of phlebitis was considered to be an arterial embolism.

The technique for injection of the paravertebral lumbar sympathetic ganglia is very simple. Injection is performed with 10 cc. of a 1 per cent procaine hydrochloride solution. Following the injection, the leg feels subjectively warmer, pain is markedly decreased, and the

patient generally has an area of hyposensitivity to a pin prick, extending from the iliac crest to just below the knee on the injected side. Ochsner feels that sympathetic ganglion block should be done daily until the patient's temperature returns to normal, because it is believed that as long as fever persists, vasospastic impulses can originate in a thrombophlebitic segment.

In a series of 25 cases of thrombophlebitis reported by Nicholson, the results obtained from paravertebral sympathetic block were gratifying. Pain was generally relieved within the first half hour and relief usually was permanent after the first or second treatment.

The results obtained in the treatment of chronic thrombophlebitis by this method has not been so encouraging. Paravertebral sympathetic block combined with bed rest has generally given these patients a period of freedom from pain, with return of the leg to normal size. When normal activity was resumed, edema reappeared after the leg had been in a dependent position for six to eight hours. In treatment conservative measures such as encouragement of reflex vasodilatation by immersing the hands and arms in hot water for twenty minutes two or three times a day, avoidance of vasoconstrictor influences such as smoking and exposure to cold, may be beneficial. These patients also should be instructed to prevent edema from occurring in the extremities by applying a compression bandage before arising and elevating the extremity whenever possible. If these conservative measures fail, then the more radical method of producing vasodilatation will be necessary, and this can be had through lumbar sympathectomy.⁶

The chief criticism directed against paravertebral sympathetic block is that expressed by the advocates of ligation and division of the femoral vein. They claim that the injection method, while affording symptomatic relief, is not an adequate safeguard against pulmonary embolism.

4. PROXIMAL LIGATION OF VEIN: Believing that anticoagulant therapy, paravertebral sympathetic block, and other means of treatment already described do not provide sufficient insurance against embolism, many workers are attempting to interrupt mechanically the route by which emboli travel. This is an old principle of treatment. John Hunter used compresses for the purpose, and this is a method which still has a place in therapy. But it has obvious limitations, and the next logical step is ligation and division of a proximal venous segment.

Jacob Fine, Howard A. Frank and Arnold Starr, all of Boston, have made a careful study of this question and conclude that ligation and division, confirmed and controlled by venography, comprise the surest guarantee against pulmonary embolism. They write: "Homans and,

after him, several others divided the femoral vein as a prophylactic measure against embolism in isolated instances of thrombophlebitis of the lower leg. This procedure has not acquired the vogue which we believe it deserves. In a first series of patients so treated we observed, in agreement with Homans, that (1) embolism did not occur and (2) the phlebotic process in the leg subsided—in some instances at a remarkable rate. Pain decreased or disappeared in a day or two, and edema, when present, frequently began to subside more quickly than might be expected. The result was quite analogous to that obtained by novocain block of the paravertebral sympathetic trunk and was presumed to be due to a similar mechanism.”⁷

They mention as an additional advantage of treatment, though a secondary one, the short convalescence. “According to Ochsner and De Bakey, a shortened convalescence is also achieved by paravertebral novocain block, but this technic does not, we feel, provide protection against the discharge of an embolus.”

“In a previous report,” they continue, “routine division of the femoral vein was recommended in all patients known to have or suspected of having thrombophlebitis of the deep veins of the lower leg. This conclusion was derived from the fact (1) that there was no reliable way to predict that an embolus would occur; (2) that the incidence, morbidity and mortality from emboli were far greater than is generally realized. Since a dependable venographic technic has become available, we no longer divide the femoral vein on the basis of suspicion, but always if the signs and symptoms are obvious or if a filling defect is demonstrable in the venogram.”

“It is evident that femoral vein division cannot prevent thrombosis proximal to the site of division. This constitutes the chief limitation of this procedure for effective prophylaxis against embolism. But since it is an uncommon occurrence, we feel justified in adhering to our position that routine division of the common femoral vein is the procedure of choice in lower leg thrombophlebitis.”

“Though on uncertain ground in deciding whether surgical intervention is justified before infarction has occurred, we should not hesitate to operate after a first infarct. To wait for a second infarct before operating is too arbitrary as a rule to be reliable. We regard a first infarct, however slight, a sufficient indication. The type of surgical procedure which should be adopted is by no means established. Taeven, Kulenkampff, and de Takats and Jesser limited their efforts to clot extraction without division of the vein. Our view is in agreement with that of Homans, that division of the vein, or at least ligation, should be done whether or not complete extraction of clot or thrombus is achieved.”⁸

The analogous treatment of acute superficial thrombophlebitis in an incompetent venous system of the lower extremity is described by Heyerdale, Clagett and Anderson: "The greater saphenous vein is divided at the saphenofemoral junction and a sclerosing solution is injected into the distal segments (5% solution of sodium morrhuate in amounts varying from 0.5 to 3 cc.). In the presence of thrombophlebitis the activity of the sodium morrhuate is increased. Consequently less sclerosing solution is used in the veins affected by phlebitis than in uncomplicated varicose veins. Warm, moist packs may be applied to the affected extremity postoperatively for the patient's comfort. The patient is dismissed from the hospital a day or two after ligation, to return to the clinic for further sclerosing therapy to eliminate the remaining patent varicose veins."

The advantages of the described treatment are listed as follows: (1) duration of the acute stage of the disease is shortened, (2) varicosities which require ultimate treatment are obliterated at the same time, (3) there is less possibility of recurrence of phlebitis and (4) the likelihood of the development of pulmonary emboli from the thrombosed portion is reduced.⁹

SUMMARY

1. A survey of the recent literature on the treatment of phlebothrombosis and thrombophlebitis is presented.
2. The results of treatment with the sulfonamides, the anticoagulants and paravertebral sympathetic blocks are discussed.
3. Stress is laid upon the value of venous ligation in the prevention of pulmonary embolism from thrombophlebitis in the lower extremity.

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Acute Cholecystitis

By SHIRLEY E. DRESSER, '44

FOR the past few years there has been much discussion about the problem of acute cholecystitis, resulting in some differences of opinion as to the correct treatment of the condition.

By acute cholecystitis is meant a clinical picture characterized by the following: severe pain, fever, leucocytosis, local tenderness, nausea, vomiting, and a palpable mass in the upper abdomen. In these patients the gall bladder is engorged, oedematous or necrotic or an abscess or suppurative process may be present in the gall bladder, the bile ducts or the liver.

Everyone is in agreement that surgery is the means of ultimate cure of this condition but the problem lies in deciding when to operate, especially as to whether this should be undertaken during the acute stage. While it is true that the majority of patients will recover spontaneously from their acute attack and therefore can be treated surgically at a later date, it is also true that more than a few cases have terminated fatally from peritonitis following gangrene and perforation of the gall bladder during the course of conservative management. Thus, the key to rational therapy lies in understanding and correlating the pathology and the symptomatology with the application of such knowledge to each individual case.

AETIOLOGY AND PATHOLOGY:

Two factors play a part. The first is acute inflammation of a varying degree affecting all layers of the gall bladder but usually most marked in the submusoca. The irritant causing the inflammation is usually infective, but other cases are non-infective, and here the irritant is trauma, *e.g.*, stones or reflux of pancreatic ferments.

The second factor is acute obstruction of the gall bladder outlet either by a stone impacted in the cystic duct or occlusion by inflammatory oedema. This factor is important because certain characteristics arise only when the outlet is obstructed. Hence, the condition may be called ACUTE OBSTRUCTIVE CHOLECYSTITIS.

The importance of obstruction depends upon whether it will, or will not be relieved. Rutherford Morrison has pointed out the various changes following acute obstruction of the gall bladder. One of three conditions results, depending upon the amount of bile present when the blocking-off occurs. These are (1) The gall bladder slowly distends, due to its own secretion, and forms a chronic painless lump in the right hypochondrium (hydrops of the gall bladder). (2) The gall bladder distends and becomes inflamed, forming a tender mass in the right

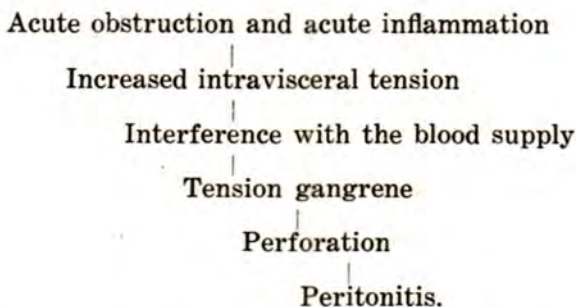
hypochondrium (empyema of the gall bladder). (3) The distended gall bladder becomes gangrenous (gangrenous cholecystitis).

In hydrops, the organ will have been emptied before the obstruction and then gradually becomes refilled by secretion. In gangrenous cholecystitis, the stone becomes impacted first and the gall bladder remains full of infected bile. The means by which gangrene results can be explained by the following principle: if, in any hollow muscular viscus, active inflammation is superimposed upon obstruction, the intravisceral tension increases and may become so acute as to interfere with the circulation to such an extent that partial or total gangrene followed by rupture and spilling of its contents will result.

In acute cholecystitis, the gall bladder shows both inflammation and obstruction. Intravisceral tension is raised by increased fluid contents caused by exudation from inflammation and transudation due to congestion of the blood vessels following forcible smooth muscle contraction. The thickening of the gall bladder wall is also caused by this exudative and transudative process. The relative importance of obstruction and inflammation vary from case to case, but these variations represent only different degrees of severity of the same disease rather than separate pathological entities.

The increasing distension of the obstructed gall bladder compresses the blood vessels in the tense wall thus interfering with the circulation and may eventually occlude the vascular channels with resulting death of the tissues supplied by them. The "tension" gangrene always begins at a spot farthest from the source of the blood supply, *i.e.*, at the fundus. It appears as a black or grayish, rounded or oval patch which steadily spreads, depending upon the extent of blood supply occluded. The mucosa is affected first and most severely. In the early cases, the mucosa may be gangrenous while the rest of the wall is not yet involved. It must be remembered that gangrene may also occur at the site of an impacted stone, due to direct pressure. Denton's theory that vascular interference is due to pressure on the vessels by stone in the cystic duct has been disproved because in many cases no stone is found in the cystic duct.

The termination of an attack of cholecystitis by resolution, fibrosis, partial or total destruction of the gall bladder would appear to depend more on the obstructive than on the inflammatory element of the pathology because when the gall bladder is successful in emptying itself the attack usually subsides. Clinically, partial or total gangrene never occurs except in those cases in which the gall bladder is tensely distended and obstruction present. The following is a short summary of the pathology of this condition:



If left to nature, the increased intravisceral tension may be released in one of two ways:

(a) The obstruction is overcome or removed by natural means, *e.g.*, dislodgment of a stone at the outlet, thus allowing the contents to escape by the natural route. In this case the patient will recover spontaneously from the attack without surgical interference and elective surgery can be undertaken later.

(b) The gall bladder becomes gangrenous and ruptures, allowing the contents to escape through the perforation with resulting peritonitis. In such a case surgery should intervene thus preventing perforation and its sequelae.

SYMPTOMATOLOGY IN RELATION TO PATHOLOGY:

Acute cholecystitis is more common in females and shows its greatest incidence in middle life, although it may occur from young adulthood to extreme old age. In many cases a history of previous attacks usually extending over several years can be elicited.

Nausea and vomiting have been found to be the most constant symptoms. It is slight when there are no stones and no peritonitis but if either of these is present it may be constant.

Pain varies and may be preceded by upper abdominal discomfort for several days before severe pain has its onset suddenly. If a stone is attempting to pass along the duct it is very severe and colicky in type. Pain may be localized to the gall bladder region, diffused over the right hypochondriac region or even felt over the top of the right shoulder or scapula. It is more widespread when peritonitis is present.

Fever is not usually very high, ranging from 100 - 103° F., depending upon the extent of the inflammation and the virulence of the infective process. Fallis and McClure have used the maximum pre-operative temperature to evaluate prognosis. They state that if this reached or exceeded 102° F. the mortality is three times that in patients with lower temperatures.

Constipation is a common occurrence, especially when there is local peritonitis involving intestinal coils in this region.

Local tenderness and muscle rigidity indicate distension of the gall bladder beyond its usual size with associated irritation of the parietal peritoneum. Spreading muscle rigidity even if no mass becomes evident indicates the advisability of surgical intervention because it signifies progression of the pathological process. In some cases of cholecystitis there may be no protective muscle spasm.

The development of a tender palpable mass in the right upper quadrant indicates that the disease has progressed to the stage where the intravisceral tension is so great that unless relieved by surgical means the pathological changes will progress rather than regress, *i.e.*, tension will continue to increase until gangrene supervenes. If the mass be seen early, it has the typical characteristics of a distended gall bladder—shape, definition, dullness on percussion and relations. Later, when the inflammatory process extends to the peritoneum, the gall bladder becomes wrapped up in the omentum and the mass loses its shape and definition, becoming more fixed.

If this organ is examined, it is found to be enlarged, partly due to thickening of the wall by oedema but chiefly due to distension by the increased volume of fluid content which may vary from dark appearing bile to frank pus. In the early stages, the wall is red, later becoming plum or blue in colour similar to a strangulated bowel and finally black or gray as gangrene appears. The wall is tense and if a small incision or puncture is made the contents are forcibly expelled, indicating the pressure within the viscus. The etiology can be seen to lie in obstruction of the cystic duct as inflammation alone would be insufficient to raise the pressure.

Recovery without interference will occur only when the intravisceral tension is relieved and by the time the tender palpable mass appears. Saint believes that spontaneous recovery can be placed in the category of wishful thinking. Progression is much more apt to be the outcome. Thus, a tender palpable mass indicates that the time for operation is at hand.

It has been reported occasionally that in acute cholecystitis, when a greatly enlarged, distended and possibly gangrenous gall bladder has been found at operation, no mass had been palpated pre-operatively. This may be explained by inability to palpate satisfactorily due to tenderness and muscle rigidity. However, if percussion be carried out, an area of dullness will be found in this region which in the normal abdomen is always resonant.

The pulse rate is often a valuable guide to the progress of the pathological process. A steady, continuous increase in pulse rate cannot

be ignored even although other clinical features tend to conceal the progress. Usually, however, other clinical features confirm the behaviour of the pulse.

Leucocytosis is present but does not always indicate the severity of the disease. Saint states that when the white cell count and the differential count are repeated, they tally closely with the clinical progress. As with the temperature, a falling white count with simultaneous progression of other clinical manifestations suggests a failing resistance of the patient.

Jaundice occurs in approximately 25 per cent of the cases. It can often be elicited if specifically inquired about when the history is taken and it indicates that a gall stone has been passed along the duct previously. Jaundice is indicative of obstruction of the outlet and may be associated with generalized pruritis. Clay coloured stools and highly coloured urine are further indications of biliary obstruction.

The above symptoms and signs are seen in the typical cases. On the other side of the picture are the so-called atypical cases in which the symptoms do not apparently parallel the pathological changes. Heuer states that this is often the case but other writers disagree for the following reason: The gall bladder is a hollow muscular organ and a sudden obstruction to its outlet causes its smooth muscle to contract forcibly attempting to overcome the obstruction and resulting in biliary colic. If the obstruction is relieved, *e.g.*, by expulsion of the stone into the common duct or by its falling back into the gall bladder, the contractions cease and no further clinical features appear. But, on the other hand, if the obstruction is relieved, after a time the muscular walls become fatigued and the forceful muscular contractions cease, resulting in relief from the severe pain—even although the outlet is still obstructed. When acute inflammation is superimposed on this organ, the intravisceral tension rises and the pathology described above follows. Thus, at certain stage in acute obstructive cholecystitis, pain may be absent and the patient feeling better while pathology is progressing. This phase may last hours or days before gangrene occurs but during this stage the gall bladder will become palpable. Operation should be advised without further delay.

When gangrene occurs, the nerves in the wall of the gall bladder become devitalized and fail to convey pain impulses. This is similar to the appendix in which there is often a lull before the storm of perforation.

TREATMENT:

Zollinger has followed a series of cases in the Massachusetts General Hospital. His treatment programme individualizes each patient. Regardless of how slight the signs and symptoms may be, as soon as

the diagnosis has been established, immediate hospitalization is urged because of the potential gravity of the disease. This early hospitalization affords opportunity to institute treatment which will prepare the patient for surgery at the proper time. Physical examination evaluating the extent and severity of the inflammatory process and the patient's general condition is carried out. Conservative measures are then begun:

- (a) Relief of pain by morphine.
- (b) Semi-sitting position with heat to the abdomen.
- (c) Administration of fluids intravenously or subcutaneously to maintain proper water balance.
- (d) Constant gastric suction if the patient is vomiting or distended.
- (e) Vitamin K may also be given.

Routine blood and urine laboratory tests are carried out. Each case is then assessed in order to decide the subsequent course of treatment. The majority of patients fall into the group whose signs and symptoms abate after adequate therapy of rest, fluids and the relief of pain. The second group includes those whose temperature, white count and physical findings remain constant with no response to initial treatment. In this group, operation should be performed within 36 to 48 hours after adjustment of the fluid balance. The third group is small and consists of those patients who become rapidly worse. These require simple drainage of the gall bladder as soon as possible.

TIME FOR OPERATION:

This is the cause of much dispute, but it may be said that fixed rules are hard to lay down.

Emergency operation is rarely necessary before the patient can be given proper pre-operative care. It is advisable to carry out early operation when the patient is a good risk and seen soon after the onset, when a case who has been making satisfactory progress under conservative management develops an exacerbation; when there is no response to medical treatment; and finally when the patient is acutely ill, showing signs of generalized peritonitis.

The operation of choice is cholecystectomy. The gall bladder is usually brought into view and its contents emptied through a trocar. This makes it easier to remove as well as lessening the danger of infection. Cholecystectomy has several advantages over cholecystostomy including no danger of recurrence, no necessity for a second operation, no soiling by leakage and more rapid convalescence. However, cholecystostomy may have to be carried out in poor surgical risks, seriously ill patients demanding minimal surgery or those presenting technical difficulties for cholecystectomy.

MacDonald advises the two-stage operation, supported by the following argument: The first stage—drainage—is preventive for the immediately dangerous phase, while the second stage—cholecystectomy—is easier to carry out later. Drainage may be performed, using a small catheter inserted under local anesthesia. This allows the physiological changes to return to normal, before extensive surgery is performed. Before the second stage cholangiography may be done to determine the condition of the duct system as well as the size and location of calculi. This two-stage procedure has not been widely used, hence its value is still undecided.

The main principle in treatment is surgery when indicated and when the patient is properly prepared. This can be carried out only when pathology and symptomatology are correctly and rationally correlated.

SUMMARY

(1) Acute cholecystitis is discussed with special emphasis on the etiology and the pathology and the correlation of the pathology with the symptomatology.

(2) A short review of the newer methods of treatment is given.

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DIABETIC COMA

In diabetic coma
The skin is red and dry.
This thirsty, sated patient
Has softness of the eye.

His breath is deep and rapid,
And reeks of acetone;
A tremor is uncommon,
Convulsions quite unknown.

His tongue is parched and wrinkled,
His head and belly ache;
His rapid heart and restlessness
And illness he can't fake.

One may obtain a history
Of insulin withheld.
Infection, food, emotion
Have many doubts dispelled.

If sugar-laden urine,
If CO_2 is low,
He should improve with insulin
In seven hours or so.

These facts we should remember:
There's acidosis first,
Then loss of body sodium,
And lastly tissue thirst.

Give insulin heroically,
And give glucose as well
As fluids intravenously,
With some NaCl.

If still he fails to rally
(That is, if you're no fakir),
You must admit he'll make a date
With some sweet undertaker.

—O. E. OCKULY, M.D.

(From Tonics and Sedatives, J.A.M.A., Volume 122, page 30; July 3, 1943.)

UNIVERSITY OF WESTERN ONTARIO
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Articles and communications for publication in the JOURNAL should be addressed to the Editor, University of Western Ontario Medical School, London, Ontario.

The Editor will accept for publication articles by undergraduates, by alumni, and by staff members. The number of articles by graduates published in any one issue shall not exceed 50 per cent of the total.

All correspondence regarding subscriptions, advertisements, etc., should be addressed to the Business Manager, University of Western Ontario Medical School, London, Ontario.

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It is drawn to the attention of the readers of the JOURNAL that *Volume 13 was complete in one issue and that Volume 14 will be complete in three issues.*

The JOURNAL will be published in June and October of this year.

Editorial

This is the first issue of Volume 14 of the MEDICAL JOURNAL. Last year only one issue was published but the JOURNAL staff this year has pledged itself to renew and maintain the quarterly publication of the JOURNAL.

At various times interest in the MEDICAL JOURNAL ebbed so low that it seemed advisable to discontinue the publication. The JOURNAL had been launched on the sea of publication only for a short time when it was cast on the financial rocks. But in one year, an industrious staff, with careful planning, erased the large debt. The alumni were generous in their support and encouraged the undergraduates to continue the publication.

Finances were not the only problem. There came a war, and with it a speed-up in the medical curriculum. Doctors and students had new and increased responsibilities. Thus, no one found time to write articles for publication. It became increasingly difficult to publish four issues of the JOURNAL each year.

There are many reasons why the publication of the JOURNAL should not be discontinued for the duration. It would be very difficult for an untrained staff to be organized after the war. They would lose contact with our advertisers, whose support is indispensable. But most important is the fact that the JOURNAL fulfils a definite purpose in bringing to the practitioner and students valuable surveys of the literature on new and old subjects. What is more ideal than to have each issue of the JOURNAL as instructive and practical as a refresher course?

It is hoped that the teaching staff of the University will contribute an increasing number of articles. Their unselfish devotion to the University, to their practice and to research in these busy times is commendable. The JOURNAL still provides them with an opportunity to render a further service to the students and to those doctors whom they taught in Medical School, by serving as a medium for the publication of sound, practical advice.

The JOURNAL functions as a link between the graduate and the medical work at his University. The alumni are encouraged to contribute articles for they own a large share in this publication. Perhaps a graduate has some suggestions for the improvement of the MEDICAL JOURNAL. The staff will appreciate hearing from the alumni. Their support will encourage the staff in their effort to publish a more interesting and instructive JOURNAL.

Abstracts

CHOLESTEROL LYSIS IN ATHEROMA

By TIMOTHY LEARY, M.D., Bastan.

Archives of Pathology, Vol. 37, No. 1

January, 1944.

Experiments with cholesterol-fed rabbits have established that cholesterol is esterified in the liver and deposited in the liver cells. Phagocytosis of the cholesterol ester is accomplished by the Kupffer cells which are then called "foam cells". The foam cells are freed into the blood and lymphatics and pass through the right heart, lungs and left heart to invade the subendothelial layer of the aortic (arterial) intima, thus initiating atherosclerotic lesions.

Observations of the foam cells massed in the lymphatics showed the splitting of the cholesterol ester in the cells and the subsequent solution of the free cholesterol in fatty acids secreted or absorbed by the foam cells.

In man it is thought that atheroma is the invasion of the subendothelial layer of arterial intima by foam cells loaded with cholesterol ester, causing yellow spots or streaks. If the cholesterol ester is removed, the lesions disappear; if not, they progress to atherosclerosis.

In studies of atheroma it has been established that excess cholesterol is removed from the foam cells by the action of lipolytic fibroblasts and dissolved in fatty acids within the fibroblasts. The cholesterol disappears from the affected area as do the gross lesions and the foam cells disintegrate. The fibroblasts evidently deal with the cholesterol by carrying on cholesterol metabolism within themselves.

The defence mechanism presented here is the first specific example of cholesterol metabolism so far described. Nature's method of removing cholesterol

from the lesions may suggest a new approach to the treatment of atherosclerosis.

—KEN. WARD, '47.

THE PROGNOSIS OF ANGINA PECTORIS

By PAUL D. WHITE and
EDWARD F. BLAND

From J.A.M.A., 123:13, Nov. 27, 1943.

The average duration of life after the first attack of angina pectoris has classically been set at five years. Follow-up of White and Bland's old group of 500 cases has yielded 7.9 years as a more accurate prognosis. This does not necessarily mean the angina patient is living longer than formerly, but does indicate the need for careful search for the date of a first attack. It is possible, however, that practice of more careful living during the times of greater coronary insufficiency has had a beneficial effect. 76 per cent of deaths in the group were due to cardiac causes—myocardial infarction and congestive failure. Certain factors such as hypertension, myocardial infarction, cardiac enlargement, abnormal heart sounds, congestive failure and abnormal electrocardiogram were found more often in those that died in three years than in the small number still living. Nervous sensibility was more frequent in the longer than in the shorter living group for the steady, not easily agitated person usually does not heed the early symptoms of disease and tends to exceed his physical reserve. The time to death of those in the group afflicted at some period with angina pectoris debitees was not much less than that of the group as a whole. From study of 75 additional patients, however, it was found that once angina at rest appeared, life thereafter was relatively short—averaging 2.8 years. Angina pectoris indicates temporary coronary insufficiency.

ency and though the change in the arteries is that of chronic disease, the resultant myocardial pathology can be acute or subacute. With this fact weigh all treatment or prognosis of the disease.

POTASSIUM SULFOCYANATE THERAPY IN THE TREATMENT OF ESSENTIAL HYPERTENSION

By ETHEL FANSON, M.D., DERA KINSEY,
M.D., COMMANDER R. S. PALMER
(M.C.), U.S.N.R.

From the Medical Clinic of the Massachusetts General Hospital.

In this paper the authors have studied the use of KCNS in the treatment of essential hypertension. Previous to 1939 the use of this drug was particularly dangerous because of the severe toxic symptoms. In 1939, Barker and Wald showed the relation between the toxic and the therapeutic effects by following the blood cyanate level. The authors then proceeded to use KCNS more extensively in the treatment of essential hypertension, the results of their work being recorded in this paper. They studied the drug on 100 patients: 62 females and 38 males. The age range was 13-68, distributed as follows:

- 7% were over 60
- 20% were over 50-60
- 61% were over 30-50
- 12% were under 30.

Grades of Hypertension: The authors grade hypertension as early, moderate, late benign and malignant on the basis of changes in the eye-ground, the heart and the kidneys.

First 3 months—Rest; mild sedation ($\frac{1}{4}$ grain of Phenobarbital QID); low fat and salt diet; simple psychotherapy which they consider very important.

The results are as follows: In 40% of cases there was a fall of 10-50 points in systolic and 10-30 points in the diastolic pressure.

Then KCNS therapy was instituted. The dose used was 5 grains once to TID. The blood cyanate level was checked weekly until the dose found necessary to keep the blood level constant and at therapeutic levels. The drug was dis-

continued at the sign of the first toxic symptom. The drug was started again as soon as the toxic symptom disappeared. Thus the toxic symptoms were kept at a minimum only occurring in 20% of cases. In two of the cases there were rather more severe toxic effects, namely the enlargement of the thyroid. The gland promptly receded to normal size as soon as the drug was stopped. Then the patient was put on thyroid extract and the KCNS started again and there was no further trouble.

The results were as follows:

A fall to below 150/110 occurred in 12% of cases.

A fall of 30-50 systolic and 20-30 diastolic occurred in an additional 16% of cases. Thus a total of 28% of the patients showed a response to the drug in addition to the usual rest and psychotherapy methods which were used for three months before using KCNS. Thus the effects on the blood pressure due to the drug could be seen separated from that due to the usual method of treatment of essential hypertension.

The fall was distributed over the various groups as follows: Grade 1, 34%; Grade 2, 21%; Grade 3, 21%; Grade 4, 38%.

Remarks: The most notable observation was the additional symptomatic relief obtained with the use of KCNS in addition to the fall in B.P. already noted. The symptomatic relief was also in addition to the fall in B.P. obtained with the usual regime of rest and sedation used in treatment of hypertension. The commonest symptom relieved was an intractable migraine-like headache. This was completely relieved in 53% of all cases and was greatest in Grade 1 hypertension. Other symptoms relieved were dizziness, faintness and lightheadedness. The blood KCNS level varied from 8-12 mgms. per cent in the cases that responded and up to 18 mgms. per cent in the cases that did not respond. In many of the cases that responded the blood KCNS level was less than 8 mgms. per cent, as for example in the case report included the level was 4 mgms. per cent. This patient showed a fall from 190/120 to 140/100 with complete symptomatic relief. He is now working at moderately heavy work. His previous heart failure was cleared up

with digitalis therapy at the same time as the KCNS therapy. He now has no signs of his previous heart failure.

Summary: A fall to normal occurred in 12% of cases. A sustained fall, although not to normal values occurred in 16% more of the cases, making a total of 28% of cases showing hypotensive effects. Symptomatic relief occurred in 53% of the cases, the chief symptom relieved being headache. In the opinion of the authors, this is the chief benefit of the drug.

KCNS is a potentially dangerous drug because its pharmacology is still incompletely understood and because the therapeutic and toxic blood levels are too close and vary widely in different patients. The greatest usefulness of KCNS therapy in essential hypertension is in the relief of the severe migraine-like headache which was the most common complaint of the patients.

—BRUCE COLWELL.

INFLUENCE OF ENVIRONMENTAL TEMPERATURE AND VITAMIN-DEFICIENCY UPON PHAGOCYtic FUNCTIONS

By E. COTTINGHAM and C. A. MILLS

J. Immunology, 47:6, pp. 493-502; December, 1943.

Since recent studies in the Laboratories for Experimental Medicine, University of Cincinnati, have shown (1) that tropical moist heat lowers resistance to infection in experimental animals, although this is not accompanied by any appreciable interference with normal antibody production, and (2) that there is an increase in thiamin—and choline—requirements in heat, it was thought profitable to determine the possible changes in the activity of the body's phagocytes at different levels of environmental temperature and to investigate the part possibly played by vitamin deficiency.

Two methods of investigation were used: (1) Intraabdominal phagocytosis in mice with graded vitamin deficiencies was determined by recording for thiamin- and choline-deficient mice kept in rooms at 68° F. and at 90-91° F. and 60-70% relative humidity, on diets for

three weeks; (a) average weight gain or loss, (b) percentage of polymorphonuclears seen in smears taken from the peritoneal surface of the intestine after injection of pneumococcus culture, (c) percentage of polys with phagocytosis, (d) percentage of large mononuclears seen in smears, (e) percentage of monos with phagocytosis, and (f) average number of bacteria per monocyte.

(2) Rat white cell phagocytosis in graded vitamin-deficiencies was determined by recording for thiamin- and choline-deficient rats in cold and hot rooms on diets for three weeks; (a) average weight at end of period, (b) amount of vitamin per kilo of diet, (c) number of bacteria ingested after shaking up with non-coagulable blood for four minutes, (d) percentage of cells showing digestion after one hour.

Important findings were: (1) any factor altering the nutritional state of an animal produces a like effect upon that animal's phagocytic cells. This is shown to be true for thiamin, riboflavin, pyridoxine, pantothenic acid, choline and ascorbic acid; (2) Even mild vitamin-deficiencies seem to bring on corresponding reductions in phagocytic functions while highest activity occurs at levels of vitamin-intake above those considered necessary for good growth.

It is suggested that sub-clinical vitamin-deficiencies in man may be detectable by phagocytic determinations before and after test period administration of single vitamins and that while such determinations might be a valuable diagnostic technic, they are far from simple and may be restricted to research conditions only.

A very complete description of technics used and a list of thirteen references are incorporated in this article.

—RAY BAINBOROUGH.

PRIMARY ATYPICAL PNEUMONIA OF UNKNOWN ETIOLOGY

By S/LDR. J. F. MEAKINS, M.A., M.D., R.C.A.F.

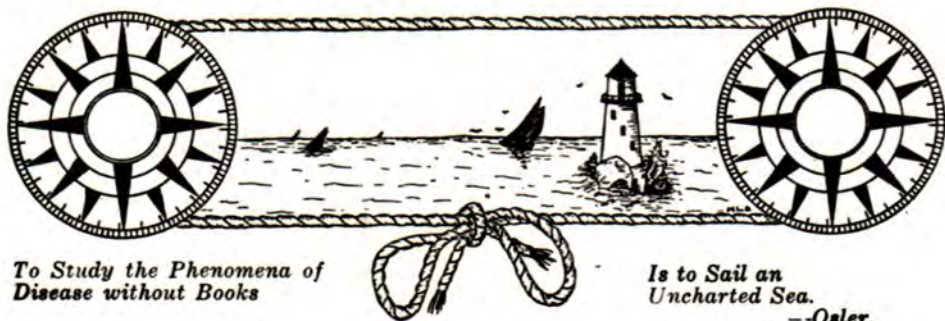
From C.M.A.J., April 1943, vol. 48.

This article presents 100 cases of pneumonia which were atypical throughout their entire course. The etiology as

yet is unknown. However, streptococcus viridans has been isolated as the probable cause. The cases at onset could easily have been confused with severe coryza, "grippe" or bronchitis. Later the diagnosis of pneumonia was made in each case by the X-ray, which showed a patchy distribution. The main symptom at onset was a dry, hacky cough. Associated with this were malaise, headache, chilliness and a head or chest cold. The toxicity during the disease was very slight in most cases. The pulse was slow considering the febrile state which fluctuated between 100 and 103. In the

majority of patients the leucocytic count was below ten thousand and never more than fifteen thousand. The duration of hospitalization was on an average 27 days. The treatment given to 68 cases was entirely symptomatic. These patients did equally as well as those who received sulpha drugs. Sulpha drugs seemed to have no effect on these atypical cases. This disease has therefore an unknown etiology, runs an atypical mild course which is not influenced by any drug and presents very few complications.

—DOUG CRAM, 45.



*To Study the Phenomena of
Disease without Books*

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

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BORDERLANDS OF PSYCHIATRY

By STANLEY COBB, M.D.

(pp. 166, 27 fig. Cambridge, Mass: Howard University Press, 1943.
Price \$2.50, U.S.A.)

In this fourth Howard University monogram, the author is concerned with a somewhat neglected six and one-half million persons who are partially incapacitated. The author stresses the body-mind aspect of disease and health rather than the sound mind in a sound body concept which is being discarded.

Every symptom of disease is both functional and organic. In the section on parallel evolution of speech, vision and intellect, it is asserted that at birth both cerebral hemispheres appear equipotential but through inheritance the learning process tends to be unilateral. "Functions of the Frontal Area" suggest the undesirability of removing a portion of the brain, thereby rendering the patient lazy and indiscriminating, in order to make the patient happier.

"Psychosomatics" is a relatively new term, having been introduced to stress the body-mind aspect of modern psychiatry. The close relationship between body and mind was startlingly demonstrated in 1907, when Kreihick, a German, induced a blister on the skin by hypnosis. More extensive lesions have been induced and it is argued that such phenomena may occur in tissues less accessible to scrutiny.

This short monogram of 166 pages would well repay further study.
—M. EDWORTHY.

THE COMPLEAT PEDIATRICIAN

4th Edition

By WILBURT C. DAVISON, M.D.

(Published by Durham, N.C., Duke University Press, 1943)

Since its origin several years ago as a notebook on facts and methods of pediatric practice for the author's personal reference and reminder, this book has become very popular and widely distributed.

It is unique in that a mass of information is presented with emphasis on signs and symptoms rather than on diseases themselves. The different systems of the body are discussed in separate chapters. Included in the latest edition are disease and prevention, nutrition, general treatment, nursing care and laboratory procedure, drugs and prescriptions with the new advances in chemotherapy. There is also a section on tropical medicine.

The book is up-to-date and complete, although it is condensed and convenient in size. The Compleat Pediatrician should serve a useful purpose in the reference library of both students and practitioners.

—SHIRLEY E. DRESSER.

NEUROLOGY

By RAY GRINKLER, M.D.

(3rd edition, 1,070 pp., 416 illus. plates, 35 tables. C. C. Thomas, Springfield, Ill.)

The third edition was brought out to fulfill the demands of recent advances in the field of neurology.

As stated in the author's preface to the 3rd edition, this book is "an attempt at a correlation of certain biological data which are considered of importance to the study of the human nervous system in health and in disease." The book is not a tabulation of signs and symptoms but a well-planned and clearly described treatise on neurology. All outmoded concepts have been eliminated.

Diagnosis and treatment of such important subjects as brain tumor, epilepsy, migraine, and histamine headaches, neurosyphilis and factual information on roentgen neurology are among the new additions.

This book of a thousand pages provides instructive reading for both the medical student and the general practitioner.

—R. B. MARCHILDON.

NERVOUSNESS, INDIGESTION AND PAIN

By N. C. ALVAREZ, M.D.,

Professor of Medicine, University of Minnesota, U.S.A.

(Paul B. Hoeber, Inc., New York. \$5.00)

This is an exceptionally interesting book, dealing not only with the diseases of the digestive tract but also with the many disturbances of nervous, arthritic or endocrine origin. There is also a great deal of psychiatry presented in a readable style.

In writing the book the author shows how the mind of the internist works as he makes a diagnosis. The sections on history taking are extremely good. A much-needed discussion of pseudo-appendicitis, pseudo-ulceration and pseudo-cholecystitis is included in this work.

The entire book is enlivened with pointed medical anecdotes and is as enjoyable as it is instructive.

—JOHN EYDT.

AN ATLAS OF ANATOMY

By J. C. BOILEAU GRANT, M.C., M.B., Ch.B., F.R.C.S. (Edin.)

(The Williams and Wilkins Company, Baltimore, 1943)

This new atlas, in two volumes, is a collection of illustrations depicting the structures of the human body; region by region, rather than system by system. Students will find this to be an advantage when they are using the atlas during their course of dissection. Volume I covers the Upper Limb, Abdomen, Perineum, Pelvis and Lower Limb, and Volume II covers the Vertebrae and Vertebral Column, Thorax, Head and Neck.

The illustrations were prepared from specimens in the Anatomy Museum of the University of Toronto, and they have been accurately produced, but it is felt that they would have been improved by full colouring. There are accompanying observations and comments that are designed to attract attention to salient points and to points of significance that might otherwise escape notice. The purpose of these brief remarks is only to aid in the interpretation of the illustrations, for they are not exhaustive descriptions. Occasional diagrammatic representations are included, where it is desirable to clarify certain features of the anatomy in Dr. Grant's own skilful manner.

The atlas is authentic and inexpensive and it will undoubtedly be widely used.

—K. W. RUNNALLS.

Royal College of Physicians and Surgeons of Canada

Examinations — 1944

The examination for the Fellowship is divided into two parts, viz., the first examination or Primary, and the second examination or Final.

The subjects of the Primary examination are:—Anatomy, including Histology and Embryology; Physiology, including Biochemistry.

The Primary examination is partly written and partly oral and must be passed as a whole.

The Primary examination may be taken at any time after the candidate has completed a course of study and passed the examinations in Anatomy, Histology, Embryology, Physiology, and Biochemistry, in a Medical School or University approved by Council. The candidate must submit a certificate thereof with his application.

LANGUAGES OF EXAMINATION

Candidates at the time of making application for either the Primary or the Final examination shall indicate whether they desire to be examined in the French or English language.

STANDARDS OF QUALIFICATION

No particular list of text books or syllabus is recommended to cover any subject. All candidates are expected to demonstrate a thorough knowledge of the subjects in which they are to be examined and to be familiar with the current literature relating thereto.

In their answers, written or oral, candidates in the Final examination must show evidence of critical judgment.

PLACES AND DATES OF EXAMINATIONS — 1944

The centres selected for the Written Examinations are Vancouver, Edmonton, Saskatoon, Winnipeg, London, Toronto, Kingston, Montreal, Quebec and Halifax — October 2nd, 3rd and 4th.

Oral Examinations in the Primary subjects and Oral and Clinical Examinations in the Final subjects will be held as follows: At Montreal, October 23rd and 24th.

THE FELLOWSHIP FEE

Candidates taking the Final Examinations will please note that the Fellowship Fee (\$150.00) must be paid in advance. This will be returned if the candidate is unsuccessful in passing the examination.

Candidates who are graduates of 1930 or prior thereto of a Medical School or University approved by Council shall not be required to take the Primary Examination, but shall in the Final Examination demonstrate a general and practical knowledge of the clinical application of Anatomy and Physiology.

This special examination shall be conducted by Clinicians.

Address all communications to—

WARREN S. LYMAN, M.D., F.R.C.P.(C),
Honorary Secretary,
Rm. 3018, National Research Laboratories,
Sussex Street, Ottawa, Ontario.