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PAUL STILWELL McKIBBON
1886-1941
PAUL STILWELL McKIBBEN
(1886-1941)

Dr. Paul S. McKibben, Professor of Anatomy and Dean of the Medical School of The University of Southern California, died November 11th, 1941, after a brief illness. He was born in Granville, Ohio, March 14th, 1886. He was educated in Ohio taking his B.S. degree at Denison University. His graduate studies in Anatomy were in The University of Chicago. After receiving his Ph.D. from Chicago, he was instructor in the Department of Anatomy there. In 1913, at the invitation of the late Dean H. A. Macallum, he came to London as Professor of Anatomy in the Medical School at the early age of 28. His coming to London marked a turning point not only in the history of the Medical School but of the University as a whole.

McKibben was a real organizing genius. He was made Assistant Dean in 1919 and, later, Dean of the Medical Faculty, which position he held until he left London in 1927. From London he went to The University of Michigan, thence to The University of Southern California. His continuous service to this University was broken only by a short period of service as an officer in the American Army in 1918 and the early part of 1919.

The Medical School on Ottaway Avenue, built in 1921, stands as his monument. He planned the building carefully, actually drawing many of the plans and laying out the detail. The laboratory departments in the Medical School proceeded in an orderly and logical development under this guidance and, during his tenure of office, the clinical departments were reorganized. The building up of the Medical Library, one of the best in Canada, owes more to Dr. McKibben than to anyone else.

While a genius in arranging material things, it was, however, the human and personal side of Dr. McKibben that loomed so large. He was quiet, dignified and reserved, with the most delightful sense of humour, and facility of expression. He was absolutely unselfish and gained more genuine satisfaction from the accomplishment of others under his direction than from anything he did himself. Many a weary, discouraged student was inspired by him to greater effort. Faculty members of all grades came to him regarding their personal problems. He had tact and wisdom abounding. He was a great teacher and educator and, withal, a true gentleman. As a builder and creator his name will be engraved large in the history of this University.
SEEKING NEW FRONTIERS
Within Four Walls

The four walls of the laboratory do not a prison make for the vision of the inspired worker in search for new frontiers in medical progress. From his retorts and crucibles issue new ways and hitherto unknown measures for the alleviation of disease and for its prevention. To the earnest search for new and improved medicinal substances the Warner Institute for Therapeutic Research is dedicated. The results of its exploration in the field of purely scientific research are presented in the reports published from time to time. To make available its discoveries in therapeutic products to the physician and pharmacist, is the privilege of William R. Warner & Co., Inc., a worldwide organization, with laboratories and agencies in 75 countries.
AN editorial in the Canadian Medical Association Journal for June 1941 notes that much work has to be done before we have a perfect blood substitute or even before we have a suitable way of storing whole blood or plasma.

In order to restore and maintain the volume of circulating fluid, a transfusion fluid must answer the following requirements:

(a) The molecule must be of such a size that the fluid will not leave the vessels too freely.

(b) The osmotic pressure and viscosity must be equal to those of blood.

(c) It must be isotonic.

(d) It must be non-antigenic and innocuous.

(e) It should be cheap.

(f) It must be easily sterilizable.

A number of transfusion fluids have been used to keep the circulating volume within physiological limits. Some of these are:

1. Blood
2. Blood plasma and serum
3. Hæmoglobin—Ringer and hæmolyzed blood
4. Gum-saline
5. Saline
6. Gelatin saline
7. Casein hydrolysate
8. Pectin.

1. Blood:

Whole blood is theoretically the best substitute when the circulatory volume is decreased because of hæmorrhage (hæmatogenic shock). It has a number of practical drawbacks. It cannot be sterilized; it deteriorates fairly rapidly; it is expensive. In the German army men are assigned to detachments according to their blood groups so that
if any soldier needs a transfusion a suitable donor is available to give it to him. The use of cadaver blood has been recommended by certain Russian investigators. Placental blood has been used and should prove to be a valuable source for supplying blood for plasma banks in general hospitals.

Blood changes fairly rapidly once stored. The leucocytes, especially the granulocytes, disappear very rapidly. The prothrombin and complement titers fall off more slowly. Ordinary citrated blood is useless after being stored for ten days because of hæmolysis. The exclusion of air, dilution of the blood and addition of glucose increases the length of time it can be stored. A solution of 500 c.c. blood + 650 c.c. of 5.4% anhydrous dextrose + 100 c.c. of 3.2% dihydric sodium citrate can be kept for thirty days. The longer the blood is stored, the shorter the life of the red cells in the recipient’s body. All stored blood must be filtered when being given to remove the inevitable small clots from it.

2. Plasma and Serum:

In most cases of shock the signs and symptoms are produced by a loss of plasma protein and with it a loss of fluid into the tissues. The circulatory volume is reduced, the tissues and capillaries become anoxic, more proteins are lost through the damaged capillaries, etc. Finally the body ceases to compensate for this loss and the blood pressure falls. Shock is not due to lack of hæmoglobin. By repeated bleeding it has been shown that animals can get along quite well on a hæmoglobin of 20%. Almost every physician knows patients who live quite well with a red cell count of 1,000,000 and a hæmoglobin of 20%. It is the plasma proteins that are important in maintaining the circulatory volume. This idea was introduced by Martius in 1882 and upheld by Ringer in 1885. The technique of plasmapharesis introduced by Abel, Rowntree and Turner simplified the study of the plasma proteins. The modern concept of the protein theory was worked out by Whipple in 1920.

After hæmorrhage, the concentration of hæmoglobin is altered little at first but gradually falls as the blood volume is brought back to normal by production of plasma proteins from the patient’s own reserves. When the hæmoglobin falls below a certain level, this process of hæmodilution becomes incomplete. If the hæmoglobin is over 50%, plasma is a good substitute. If below 50%, whole blood should be used.

In crushing injuries, peritonitis, burns, etc., shock occurs from loss of plasma proteins out into the injured tissues. In such cases, plasma is a better replacement substance than whole blood. It does not need any cross-matching if pooled plasma is used. An hæmatocrit reading of more than 55% reveals a need for more plasma.

In England, mixed plasma is made by adding to two parts of
Group O plasma four parts of Group A plasma and one part of Group B plasma. This is given in quantities varying from 1,000 to 2,500 c.c. As much as 5,000 c.c. has been given.

Plasma is obtained from blood which has been stored too long to be used as whole blood. It is passed through a bacteriologic filter and stored. Frozen plasma keeps perfectly. When it is needed, it can be melted, brought to 37°C and given to the patient. Dried plasma keeps well for years. The best dried plasma yet produced is made from frozen plasma. Other methods are vacuum evaporation of the liquid plasma, evaporation of the liquid in cellophane bags, and removal of the water under oil in a special centrifuge apparatus. Wangensteen has introduced bovine plasma and has used it with very few ill effects.

The advantages of plasma have been summarized well by Blalock. "Volume for volume plasma supplies approximately twice as much osmotically active protein as does whole blood, it is nutritionally more effective, it need not be typed, it is more easily administered, and finally, it is readily preservable in the liquid state for considerable periods, or, suitably prepared as a dry powder, it may be stored for any length of time desired. Whole blood, on the other hand, has a limited life of from two to three weeks."

3. \textit{Hæmoglobin—Ringer and Hæmolyzed Blood:}

According to some writers (Osborne, 1930, and Field, 1931) laked blood is quite toxic. Others (Herman, 1859, Ringer, 1882, and Bayliss, 1920) do not believe it is toxic. This discrepancy in the literature may be due to the fact that the stromata of the red cells are toxic and not the hæmoglobin itself. Amberson (1934) thinks the hæmoglobin is toxic. The high potassium content of human hæmoglobin may produce some of the deleterious effects. In massive doses, it damages the kidneys.

It has some advantages, being soluble in physiological saline, takes up oxygen faster than the red cells, has a molecule equal in size to serum albumin (68,000 according to Svedberg and Nicols, 1927) and stimulates the formation of red cells.

4. \textit{Gum-saline:}

Gum arabic, first used experimentally by Ludwig in 1863, had its first clinical trial in 1917. Crude gum arabic consists of the calcium, magnesium and potassium salts of arabic acid. These cations are replaced by sodium in commercial preparations. Arabic acid can be hydrolyzed into a number of pentose and hexose sugars and acids with a galactose-glucuronic base.

The gum tends to aggregate in higher concentrations and to split up into smaller molecules at lower concentrations. Therefore, the mole-
cular weight changes with the concentration. The viscosity increases more rapidly than the concentration due to the formation of aggregations of molecules. The osmotic pressure of a 6% solution is 9 to 60 mm. of mercury according to the investigators and methods used to calculate it.

Gum acacia greatly hastens the sedimentation of the red corpuscles. According to Christie (1935), it is supposed to decrease the oxygen carrying capacity of the red cells by 40%. It depresses the blood proteins, especially fibrinogen.

Many British writers, such as Watson-Jones (1941), believe it is dangerous. Others tolerate its use and still others, such as Keith, believe it is very good. The Mayo Clinic have used it in over 3,000 cases without a fatality. Stanbury et al (1936) reported very enthusiastically about its ability to maintain the blood pressure in dogs after plasmapharesis had reduced the plasma proteins to .1 to .2%. It disappears from the blood in about 120 hours and is only effective for 48 hours.

Some anaphylactic effects have been reported due to traces of protein in it. Even highly purified solutions of gum acacia contains some protein, but Amberson (1937) has never observed any toxic effects from these solutions.

The use of gum acacia may be summed up by saying that the objections are not so serious that they prohibit the use of it if whole blood or plasma are not available.

5. Saline:

This is the cheapest, the most easily prepared, and least valuable substitute yet produced. The only place where it is any good is in marked haemoconcentration where large quantities of water and salts have been lost and the patient is markedly dehydrated. Patients are made worse if it is used in haemotogenic or vasogenic shock because it helps to wash out any plasma protein in the blood and an accentuated hypoproteinemina results.

6. Gelatin-saline:

Ringer first used it in 1885 and found it was not as good as serum. It is non-antigenic, leaves the vessels in six to eight hours, favors intravascular clotting and occasionally produces tetanus and anthrax because of contamination with B. Tetanus and B. Anthracis.

Taylor and Waters introduced isinglass in June, 1941. This substance is a gelatin obtained from fish swim bladders. It is supposed to possess all the properties of a good replacement substance with no known drawbacks. Time will tell how it works out.
7. Casein Hydrolysate:

Casein hydrolysate has been used by Whipple and his co-workers as an extremely powerful stimulant to plasma protein production when given intravenously. It is used to get the body producing its own plasma proteins as fast as possible.

8. Pectin:

Hartman et al (1941) point out many objections to the use of gum acacia. They propose to use a purified type of pectin. It is supposed to be non-antigenic and non-toxic, cheap, and to have the correct physical properties in a 0.5% solution. It temporarily depresses liver function slightly.

SUMMARY

1. A short review of the literature is presented.
2. An ideal blood substitute has not yet been found.
3. Blood and plasma are the two best yet produced.
5. Plasma is best for vasogenic shock.
6. Gum saline is the best of the synthetic replacement substances that have been given a clinical trial.

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Involuntary Movements

By SHIRLEY M. FITCH, B.A.

UNDER this heading are included muscular contractions not brought about by the individual's volition. They can be conveniently divided into fine, medium, and coarse movements. Under such divisions, etiological types and, where possible, pathological nature will be discussed.

It is unfortunate that neuro-pathology is not complete; as yet electroencephalographic studies are of little help on this subject, except in the epilepsies when "seizure discharges" may be found. Otherwise the graphs rarely show abnormalities, since the record merely represents cortical function. Organic causes of involuntary movements are usually pathological lesions in the extrapyramidal system of the "old brain," i.e., the corpus striatum, red nucleus, substantia nigra, in the cerebellum, and in the associated tracts. Lesions resulting in complete interruption of the efferent path from cortex to muscle must produce paralysis, and if involuntary movements have been present they will disappear with the paralysis. Whether the movements to be discussed are due to irritation or are release phenomena cannot be decided, but probably both factors act in different cases.

Fine Movements

SHIVERING is a physiological reflex, producing warmth by increased muscular metabolism and peripheral vasodilatation, when the surface of the body is cooled. In toxic conditions as pneumonia, malaria, urinary infections, etc., a similar skin-vascular reflex occurs with a rising temperature, when the skin surface temperature is relatively lower because of vasoconstriction. In these RIGORS or chills, the amount of movement varies from contraction of the erectores pili to shaking of the whole body.

FIBRILLATIONS are slow vermicular contractions of individual muscle fibres or bundles, usually signifying slow degeneration of anterior horn cells. The origin may be from disordered muscular metabolism secondary to loss of the trophic influence of anterior horn cells, instead of central. In anterior poliomyelitis, syringomyelia, and other lesions which cause destruction of anterior horn cells or motor nuclei, as tumours or vascular occlusion, fibrillations and atrophy occur. The progressive muscular atrophies probably result from degeneration in the anterior cornua, but muscle atrophy and neuritis have been held responsible. The spinal form of Aran-Duchenne was described first as beginning in the small muscles of the hand. With the bulbar form the muscles supplied by the twelfth, tenth, ninth, seventh and sometimes fifth cranial nerves are involved and tongue fibrillations are prominent. Pseudobulbar palsy is differentiated from this by the absence of atrophy,
fibrillations, and changes in electrical reactions. Chronic progressive ophthalmoplegia of von Graefe involves the motor nuclei of cranial nerves three and four, while the smooth muscles of the iris and ciliary body are not affected. Werdnig-Hoffman's hereditary or familial form of progressive muscular atrophy occurs in siblings between the ages of six months and one year, involving the trunk and extremities in a symmetrical atrophy. Fazio-Lande's form is familial and occurs in infants but is a bulbar lesion. Charcot-Marie-Tooth peroneal atrophy is a familial disease in which fibrillations in the peroneal muscles with foot drop are early symptoms. Amyotrophic lateral sclerosis differs from the progressive muscular atrophies in that both upper and lower motor neurones are affected, and spasticity accompanies fibrillations and atrophy.

Twitchings are rapid contractions of muscle bundles, and may be seen in muscles whose motor nerves are regenerating as in the facial muscles following a Bell's palsy. Myokymia is characterized by transient quivers of a muscle, and may be observed in weak anaemic persons especially in the orbicularis oculi. Schultze's original case had brief painful contractions which were considered of toxic origin. Fibrillary chorea is used synonymously with myokymia.

Myoclonic twitchings are sudden regular or irregular muscular contractions usually not producing joint displacement. They are seen in the limbs, body or face, and are often the result of lenticular disease. Paramyoclonus multiplex, described by Friedreich, consists of generalized, irregular, asynchronous twitchings. The pathogenesis is unknown and there are no other neurological findings. Unverricht's myoclonus epilepsy is a familial disease combined with epilepsy which may gradually disappear, and myoclonia which may become progressively more violent. In Ramsay Hunt's dys-synergia myoclonica, progressive cerebellar ataxia, convulsions, and myoclonus occur, but Hunt points out that this may be a combination of several independent degenerative disorders. Nystagmus-myoclonia of Lenoble and Aubineau, a familial disease, consists of congenital nystagmus which may be an ocular myoclonia, tremors, and myoclonic twitchings. Encephalitis may have as a symptom, or as a residual, localized twitchings especially of the abdominal muscles. Dubini's chorea, a meningo-radiculare variety of epidemic encephalitis has myoclonus, progressing to convulsions, with insomnia and delirium, and then death. The movements of Electric Chorea of Bergeron and Henoch are also twitchings, but last only a few weeks. As a preconvulsive symptom in grand-mal epilepsy, Adams-Stokes syndrome, azotemia, hyperinsulinism, and metrazol shock, myoclonic twitchings occur, and they may be the only involuntary movements in petit-mal epilepsy. Perioral fibrillary twitchings are part of the classical picture of general paresis, and the convulsions may be accompanied by twitchings. In tetany, stimulation causes twitching,
perhaps the result of increased irritability of the motor nerves, *e.g.*, Chvostek's sign.

**Medium Movements**

**TREMORS** are oscillatory movements described as coarse, medium, or fine, depending on the range of movement; slow, moderate, or rapid in rate, and regular or irregular in rhythm. With strong emotions as in stagefright, fine or coarse tremors occur when part of the body is held rigid, and in the anxiety type of psychoneuroses, emotional upset and tremor are related. In hysteria, tremors are a conversion symptom and may be bizarre in type. Fatigue tremors occur after over-exertion of a muscle, the explanation being that normal movements are a series of fused contractions but with fatigue the rate of contractions is slower and fusion less perfect.

Toxic tremors occur with mercury, lead, or nicotine poisoning, and in the alcoholic's lips, tongue, and hands (or can be demonstrated by Quinquaud's finger-crepitation test); this tremor may, however, temporarily disappear with the ingestion of alcohol. Acute infections may produce toxic tremors—the fine tremor of hyperthyroidism, most commonly noted in the hands, and often associated with respirations, is considered toxic in origin.

Tremors of organic origin occur with lesions in the cerebellum or in the basal ganglia. The cerebellar syndrome may show head tremors or tremors in the arms or legs, oscillatory and ataxic in nature and brought on by involuntary movement. With multiple sclerosis intention tremor is one-third of Charcot's triad, but violent tremors of the limbs or constant head "waggling" may occur, usually disappearing with rest. The pathological process may be either in the cerebellum or the red nucleus. Intention tremor occurs also in Marie's cerebellar heredo-ataxia, a variant of Friedreich's ataxia which affects the cerebellum instead of the cord, and occurs later in life. Merzbacher-Pelizaeus's disease occurs in children as a rapidly progressive, degenerative disease with tremor, nystagmus, and slow speech. Spasmus nutans is seen in babies between six and twelve months as a rotatory tremor or rhythmic nodding of the head, often accompanied by nystagmus. Closing the eyes stops the tremor, while forcibly controlling the tremor increases the nystagmus. Babies with rickets may have an involuntary rhythmic head rolling.

If the basal ganglia are involved in an hemiplegia, tremor sets in as the paralysis recedes. In paralysis agitans, either the degenerative type or that following encephalitis, tremor is an early symptom, appearing first in the distal parts of the extremities. It occurs at rest and ceases with voluntary movement, is slow, stereotyped, and frequently described as pill-rolling. Eventually it spreads to involve the jaw, tongue, and the whole body. Senile head tremor simulates that of
Parkinsonism but is not accompanied by rigidity. The first symptom of Wilson’s disease, hepato-lenticular degeneration, may be a tremor of the extremities which has a constant rhythm. The trunk and head are progressively involved, as in the pseudosclerosis of Westphal, which is probably a variant of Wilson’s disease, the degenerative changes affecting the nervous system more diffusely. When an unilateral lesion of one crus cerebri produces oculomotor paralysis of one side and tremor of the opposite arm and leg, it is known as Benedikt’s syndrome.

**Coarse Movements**

Tics are pattern movements which are performed rapidly, repeated frequently, and seem to have a purposive content. They occur in the compulsive group of psychoneuroses. “Maladie des tics impulsifs” is an uncommon disease which begins between the ages of seven and fifteen years, and with compulsive movements are associated echolalia and coprolalia.

Spasms are slow, often prolonged muscular contractions which may occur anywhere in the body. Singultus or hiccup is a clonic spasm of the diaphragm, the contraction being so sudden that the glottis is closed by suction, and the sound produced by air rushing in. Hiccup may be an hysterical manifestation, due to irritation of the sensory part of the phrenic nerve, or irritation of the inspiratory centre by toxins as in uremia, in epidemic encephalitis, brain tumors, etc. Yawning is a subcortical basal ganglion reflex, a slow tonic contraction of a wide group of muscles resulting in increased oxygen supply to the brain, and assists in emptying the veins. Habit spasms may be psychogenic in origin, reflex from irritation, or occur with disease of the lenticular nucleus; the common forms are spasmodic torticollis, masticatory spasm, glossospasm, pharyngeal and laryngeal spasms, and saltatory spasms—a hopping which occurs whenever the individual puts his feet on the ground. Spasms due to irritation are illustrated by blepharospasm with conjunctivitis, opisthotonus with meningitis, facial spasm with tic do loureoux. Fatigue, heat, and cold may precipitate spasms, probably by upsetting the electrolyte balance. Heat spasms, occurring in persons who work where the temperature is very high, are intensely painful and may last for hours or days, but may be prevented by an adequate salt intake as by drinking Ringer’s Solution. Tetany, a symptom of increased irritability of the nervous system, whether due to low blood calcium or alkalosis, is characterized by increase of muscle tonus and spasmodic contractions, especially in the muscles of the extremities, usually bilateral and painful. In the upper extremity they result in the obstetric hand, and in the lower extremity with the foot in the equinovarus position. With tetanus, spasms are brought on by the slightest stimulation and are often limited to the muscles supplied by the cranial nerves. Spasm of the vertebral muscles produces opisthotonus, but spasm of the abdominal muscles may be more powerful and empros-
thotonus results. In rabies, mild spasms of the larynx and pharynx may occur in the premonitory stage, but with the stage of excitement they become more intense and spread to involve the whole body. Spasms of opisthotonus occur in strychnine poisoning. Salaam spasms occurs in babies as a type of head nodding which may be preceded by pallor accompanied by dilated pupils or unconsciousness, or by true convulsions. Reflex involuntary spasms occur in spastic paraplegia.

**Convulsive States** consist of recurring attacks of involuntary movements with disturbance of consciousness, usually unconsciousness. The epilepsies of unknown origin are of three main types: (1) Petit mal attacks in which twitching may occur with a momentary loss of consciousness; (2) Grand mal attacks in which an aura may precede the loss of consciousness and the convulsion, which has a period of generalized muscle spasm—the tonic phase, followed by a period of jerking movements of the extremities, head and trunk—the clonic phase; (3) Psychomotor epilepsy, characterized by automatic purposeful movements during the attack, with no apparent disturbance of consciousness, but amnesia for the actions. Status epilepticus is a state of unconsciousness in which rapidly repeated convulsions occur; pyknolepsy is a mild epilepsy of children which disappears at puberty. The term spasmophilia is used to describe infantile convulsions of unknown cause.

Jacksonian epilepsy is a convulsion localized to one part of the body and is considered a focal symptom, in many cases of dural adhesions resulting from trauma. Neonatal convulsions often have birth trauma as a cause.

With acute infectious diseases in children and other generalized upsets as in tetany or alkalosis, convulsions occur. The convulsant drugs, strychnine, metrazol, and camphor, and such toxic states as chronic alcoholism, absinthe poisoning, lead encephalopathy cause convulsions; endogenous toxins are the cause of convulsions in uræmia and eclampsia. The meningitides, encephalitides, tetanus, rabies, syphilis, and other infections of the central nervous system have convulsions as a symptom, that of tetanus having only a tonic phase. Another cause is cerebral anoxemia as in the Adams-Stokes syndrome if the period of asystole lasts long enough, in cerebral arteriosclerosis, and in migraine which is probably the result of vasospasm. The cause of the convulsions of hyperinsulinism is related to poor cerebral nutrition.

Cerebral neoplasms may be symptomless for years except for convulsions, which may or may not be of the Jacksonian type—convulsions onsetting after the age of twenty, should be considered due to neoplasm until proved otherwise. Tumours and other cerebellar lesions may produce “cerebellar fits,” tonic spasms in which the head is retracted, the
back arched, arms extended and adducted with the hands flexed, and complete extension of the lower limb, the picture of decerebrate rigidity. With Friedreich's ataxia, the cerebral diplegias, tuberous sclerosis, and the mental deficiencies, convulsions are often associated. Hysteria should never be omitted as a cause of convulsive states.

Chorea, one of the hyperkinetic syndromes associated with lesions of the basal ganglia, is characterized by fairly rapid, irregular, purposeless movements which are usually aggravated by voluntary effort. The poor coördination which is so often present would suggest a cerebellar component to the symptoms. Huntington's chorea is a progressive, familial disease beginning in middle life, and accompanied by mental deterioration. The earliest movements are in the face or upper extremities, while the advanced case has a typical clownish, dancing walk. Sydenham's chorea or St. Vitus Dance has been shown to be a nervous manifestation of a rheumatic infection, probably an encephalitis, which affects the caudate nucleus, the cortex, and also the pia-arachnoid, although some writers think that the symptoms are due to minute emboli from the endocarditis. The onset is usually gradual, with change of personality, and the development of involuntary movements, usually to involve the whole body, but hemichorea may occur. Paralytic chorea involves one or more limbs, and twitches may occur in the paralytic limb, especially with improvement. Chorea may occur in the course of acute infectious diseases as scarlet fever, diphtheria, measles, influenza, etc. Chorea gravidarum may be a toxemia of pregnancy or a Sydenham's chorea occurring during pregnancy. Mild forms are not uncommon, but severe cases frequently go on to death from exhaustion unless the pregnancy is terminated. The mortality rate is about 20% for all cases in which the uterus is not emptied.

In athetosis or "mobile spasm" the movements are slow, and affect mainly the small muscles of the hands and feet with irregular flexion and extension, accompanied by excessive associated movements or synkinesias. Pseudoathetosis which occurs with impairment of deep sensibility as in tabes, combined sclerosis, paresis, or disturbed cerebellar innervation, is not a state of spontaneous movements, but is elicited when the hands are held outstretched with the eyes closed. Double athetosis is a congenital disease, while symptomatic athetoses follow hemiplegias, encephalitis, or may be seen with Wilson's disease, or Little's disease. The pathological lesion apparently is in the thalamus, interrupting the radiations to the cortex; the pallidal centres are perhaps released from neo-striatal inhibition in cases where atrophy of the corpus striatum is found. In the congenital group, the Vogts have described the status marmoratus with atrophy of the striatum and status dysmyelinisatus where the striopallidal connections have disappeared.
Dystonic movements are slow, purposeless movements, often following a definite pattern, involving the limbs, neck, and body, and probably another "striatal syndrome." Dystonia muscularum deformans is a chronic progressive disease, not familial but occurring more often in Jews of Russian extraction, beginning in childhood. The slow twisting of the neck or limbs is usually called hysteria for a long time, but the whole body becomes involved and such contractures as tilting of the pelvis, scoliosis, and foot deformities result. At necropsy degenerative changes have been found in practically every part of the body, and the occasional involvement of the liver has linked it with progressive lenticular degeneration and pseudosclerosis. Acquired dystonic movements are uncommon but they may follow encephalitis.

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The Psychosomatic Aspect of Essential Hypertension

By ALAN DOUGLAS, '42

WHILE our technique in the management of essential hypertension during recent years has remained relatively stationary, our knowledge of its pathogenesis has progressed rapidly. But even so, our concept of the symptom-complex is far from complete. Modern textbooks of physiology contain detailed discussions of the mechanism of the physiological control of the blood pressure, and recently Goldblatt's classical experiments have opened up new vistas in this regard. In addition to the old-established factors of hereditary and constitutional predisposition, we can fairly definitely say that the actual mechanism of essential hypertension is primarily humoral, and of renal origin, secondary to vascular changes, not necessarily organic.

Since Goldblatt's work in 1937, the literature has been flooded with reports and papers dealing with "renin," "hypertensin," "ischemin," "angiotonin activators and inhibitors," "angiotonin tachyphylaxis" and various other humoral agents and their effects produced, it is said, by ischaemic kidneys. But it will be some time before the maze is charted and we can be sure of our ground in this regard. Nor can we say how many new humoral factors will be postulated this year or next, nor how much more complicated the maze will grow. We must be content to leave that in the hands of investigators. As yet we cannot be absolutely sure that the factors producing renal ischaemia are the cause, and not the result, of arteriolar sclerosis.

Every discussion regarding the pathogenesis of essential hypertension must, then, include three general factors—the renal, the endocrine and the environmental. The "psychosomaticists" are those who now call upon us to consider the third factor more than it has been our custom in the past. "Psychosomatic medicine" is a new term. Like all new terms, unless it is adequately defined, misconceptions as to its true nature will arise. Psychosomatic medicine covers a much wider field than psychiatry. It is not restricted to any special branch of pathology; its object is to study in their inter-relationships the psychological and the physiological aspects of all normal and abnormal bodily functions and thus to integrate somatic therapy with psychotherapy. It is not another new medical specialty, but rather a method of approach to problems of etiology and treatment. Its great object is to study together that unity which is so often facilely separated into the pseudo-entities, "mind" and "body."

To revert to the question of environment—there are two "environments" which may be, and probably are, of importance in the patho-
genesis of essential hypertension. One is external — the urgency and intensity of modern living. This is brought out strongly by the prevalence, and greater malignancy, of hypertension in the negro in the great industrial cities of the United States, as compared to its rarity in the people of the same race in Africa—or even in the British West Indies. Likewise, the recently westernized Japanese show a far higher incidence of hypertension than the more calm and philosophical Chinese, presumably of the same racial stock. The second environmental factor is internal—the patient’s “psychic environment.” Both these factors are closely related.

Although it is difficult at present to establish definitely any concrete relationship between civilization and hypertension, it is interesting to speculate on such a cause-and-effect basis. Tobacco, alcohol, climate, clothing or housing do not seem to be the primary variables involved. The most probable factor would seem to be the effect of Western civilization on the mental conflicts of man. Our data on such a relationship is as yet meagre, but a search of the literature reveals many points of interest. Detailed studies of the emotional lives of hypertensives show that many of these individuals are characterized by increased psychomotor activity. Often they are “hyperkinetic” in all ordinary activities and show the increased drive which we popularly associate with the excitable and harassed Hollywood motion picture producer. Many have hypertrophied consciences which strive for perfection. Many have experienced emotional disturbances resulting from faulty adaptation to marked psychic and environmental difficulties. These emotional burdens often extend back into youth and seem to have been of a magnitude surpassing those experienced by most normal persons. Most of these patients, in fact, have always responded with marked emotional reactions to the usual incidents of life. Often the symptoms started with a great emotional upset, and in later life, recurrences were often found to be associated with periods of definite mental agitation. The possibility of vasomotor disturbances originating as a neurotic reaction and eventuating in *hypertension as a habit* must be considered seriously.

Fishberg states that “in all probability emotional and mental strains play an accessory rôle in the genesis of essential hypertension, serving to precipitate or aggravate the increase in blood-pressure in those individuals who have the inherited constitutional predisposition which is so important.” Admittedly this “constitutional predisposition” is one of the most constant factors met with in hypertensives. But why may not a seemingly constitutional factor such as the “spasmogenic aptitude” be but the bodily expression of psychic forces, and that in consequence the psychic make-up of the whole personality may be a factor? The studies of some psychoanalysts have called attention to the hostile aggressive impulses existing in some of these people—impulses which are neither thoroughly repressed nor yet adequately
expressed, and which, therefore, serve as a constant stimulus to the circulatory system. It has been shown often that pent-up unexpressed aggressive emotional tension seems to react on the cardiovascular system, just as do the more evanescent emotional experiences, such as fright, in the "normal."

The principles of treatment of essential hypertension have changed from time to time with changing emphasis on various possible etiological factors. During the period of the theory of the organic renal origin of hypertension, dietary treatment was in vogue. Then as the constitutional and hereditary viewpoints were popularized, general hygienic measures were emphasized. Frenzied search for foci of infection has had its day. Since Goldblatt's work, denervation of the kidneys by splanchnicectomy or thoracolumbar sympathectomy has been done in attempts to produce renal vasodilatation. These operations are as yet too new to provide any reliable data.

If hypertension is believed to be due to, or closely associated with, unresolved emotional conflicts of one kind or another, thorough analysis and psychotherapy would seem to be of prime importance. This, of course, is time-consuming. But psychoanalytic observations on hypertensive problems indicate a more adequate approach in their study than "Are you worried about anything?" and a more adequate treatment than "Go home and take it easy." In the treatment of hypertension, then, we should re-define our objectives and realize the necessity for a total evaluation of the patient. This will help us to realize that many of the hypertensive's problems are of emotional rather than physical origin, and that his incapacity, at least in the early stages, is out of proportion to his disease. Invalids may be made by their knowledge of their "high blood pressure" rather than the disease itself, and re-education along the lines of "carrying on" might be more effective than rest and more rest.

Every psychic tendency seeks bodily expression. Explanations to this effect and how inner tension which cannot be released through ordinary channels may manifest itself through the circulatory system by hypertension often leads to a discussion of problems which are of considerable importance and concern from the standpoint of the patient's illness. He talks about himself, and insight into the complex situations arises, often with relief of the hidden anxiety so closely related to the high blood pressure. This approach does not offer a complete solution of the hypertensive problem, nor does it apply to all patients. It does deal in a practical way with a set of important factors which may be modified, whereas the individual's "constitution" cannot be touched.

To sum up, Goldblatt's work and that of his followers furnishes us with a convenient hypothesis which has not yet been capable of experi-
mental proof in all aspects. And further, it is not certain that human hypertension is entirely comparable with that of experimental animals. Regardless of this, it would seem that for the time being at any rate, one of the most successful approaches to the hypertensive problem and its therapy has been the psychosomatic one. We should therefore be mindful of its possibilities and not quickly discard its method because they lie in a field somewhat more intangible than that in which we are accustomed to work.

REFERENCES

Extensive use has been made of a collection of papers on this subject found in the journal "Psychosomatic Medicine"—Vol. I, 1939—especially the paper of Edward Weiss and its bibliography. Also referred to were:
WHY READ JOURNLALS?

It is perhaps an exaggeration to say that the majority of students do not read the current literature, though undoubtedly many do not. The normal student says, and with perfect justification, that he has yet to learn all that is in the textbooks and indignantly wonders why he should be urged to go dabbling in the controversial stuff in journals. Are we to admit, then, that the current literature is only for the super-academic student? Decidedly not!

It is our conviction that a reading acquaintance with several of the more prominent journals will reward us well, even though we do not, and probably never will, know everything that is to be found on the multitudinous pages of the standard texts.

To a text-book reader, Anatomy seems a rather static science. Yet continually new conceptions are being added. It is becoming a dynamic interrelating of form and function. It is a living, instead of a dead, thing. These changes are being reflected time and again in the current literature, where they are unhindered by the shackles of tradition and form, inherent in the composition of a book.

The frontiers of physiological science are continually being pushed back. In the current literature we can all be frontiersmen. The whole medical world is literally waiting on the biochemist's researches in cellular metabolism, and the first trumpetings of these discoveries will, of course, be from the Biochemical and Physiological journals.

New correlations between physical and technical diagnostic methods and the pathological processes they represent are first heralded in the journals. Most prominent, perhaps, in the subject matter of current articles is therapy, both medical and surgical—new methods and their results—and this gets very close to the core of the practitioner's toughest problems.

Above and beyond all that has been said regarding the value of reading current literature for its own sake, there is another reason. If we have ever stopped to wonder where the present medical practitioners got their first information about the sulphonamide drugs, we will understand. It was in the current literature. It is of extreme importance, then, that we come to have a reading acquaintance with the outstanding journals in our undergraduate years so that when, at graduation, we are weaned from lectures and clinics, there shall be no break in our quest for new knowledge, and we shall escape from the indictment of practising "Rip van Winkle" medicine.

Much that is written in journals is contradictory and premature and as such does not deserve our attention. There are, however, many articles that are terse, thought-provoking jabs at the unknown or at the erroneous. It is these grains of gold which this section of the journal will endeavour to sift from the dross and present in quickly readable form, for the criticism of the students.

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EDITOR'S NOTE: The articles of this issue are devoted entirely to War Surgery and Wound Healing.

WAR SURGERY IN SPAIN
By A. T. HART, B. M. J., 1:1099 (May 27); 1146 (June 3) 1939.

It is Hart's purpose herein to present some of the most recent experiences in war surgery. He states that wound excision has completely replaced all attempts at local disinfection. Success after primary wound excision lies in the avoidance of sepsis, especially of gangrene. The chief factor in failure is persistence in the wound of non-viable tissue or gross foreign material. Hema-
Tomatoes are a very common danger and must be avoided. Once excision is done as completely as possible and, providing the patient has come to operation within twelve hours, or in certain cases within twenty-four hours of the time of injury, there is no contraindication to primary suture if certain conditions are fulfilled. The first of these is that the edges of the skin be brought together without tension. If the suture cannot be made without tension it is useless and will invariably break down. However, in a few situations a primary suture is the only method of saving life; these are open pneumothorax, penetrating head wounds, and penetrating wounds of joints. The author states the opinion that no internal suturing of nerves, tendons or of fascial layers should be performed under any circumstances. He never sutured the capsule of any joint and his results were good. Only the skin is sutured and this is done carefully to insure exact apposition of the edges.

It is agreed that in certain situations, drainage after primary suture should be avoided. These are—peritonitis, the skull and brain, joints and the sites of compound fractures where primary suture is attempted. Drainage using gauze strips is condemned; the gauze acts merely as a plug and if left for more than 48 hours makes sepsis a certainty. Dependent drainage is usually advisable if it is thought that hemostasis is imperfect. The author advises firm bandaging for about 6 hours to stop oozing.

The conditions which are considered essential for success in wound treatment are clearly stated. Success depends on providing optimal conditions for the injured tissues to undergo spontaneous repair. The first part of the treatment consists in the apposition of live tissue to live tissue, after the removal of all foreign, dead and dying matter. The second part is the provision of rest to promote the processes of repair and to overcome the infection which is almost invariably present. Necessary conditions include the immobilization of the injured parts in relation to one another, a good blood supply and the avoidance of all that hinders rapid tissue metabolism. Besides insuring adequate blood supply, one must reduce or avoid oedema which is always present at first, the onset being inevitable and fairly rapid after injury. Elevation of the wound is therefore essential. The disappearance of this initial oedema is usually a good sign, indicating successful treatment, while its persistence is invariably a sign of at least relative failure.

The next most important consideration is non-intervention. In this, the Spanish war makes a definite advance. The Winnett-Orr operation, the Dickson-Wright treatment of varicose ulcers and the occlusive treatment of septic burns are only a few examples of the general realization that the surgeon's task is to assist tissue repair that is trying to develop independently of any efforts on his part. Two alternative methods are available. One is the open wound treatment with no dressing whatever, whether it has been sutured or not. Results in this type have been excellent. The second type which also gave such excellent results in Barcelona is the application of a closed plaster cast to a fresh wound with or without petrolatum. It is evident that if primary suture has been performed there is little advantage and considerable danger in a closed cast. In such circumstances a large window should always be cut immediately, with open treatment of the sutured wound. A number of wounds remain in which suture cannot be performed and for these the closed plaster method is ideal. The conditions for its application are: (1) that a complete wound excision must have been performed, and (2) that the conditions are the same as for wound suture, i.e., the wound must be fresh (generally under 12 hours old) and must show no signs of infection.

—J. B. Moore, '43.

SYSTEMIC FACTORS INFLUENCING WOUND HEALING

By W. Grant Waugh, M.D., F.R.C.S., B. M. J., 236, 16/8/41.

The author is of the opinion that since 1914-18 the great advances in prevention and reduction of sepsis have been paralleled by few, if any, coincident improvements in the handling of established suppuration.

The article deals with two aspects of
wound healing upon which the author contends there has been too little physiological thinking. These two aspects are: (1) the influence of vitamins, and (2) the effect of controlled electrolyte and protein balance.

Regarding vitamin therapy and the effect of avitaminosis he states that:

(a) No locally applied vitamin has been shown to have any effect on repair. As an example of this, he cites cod liver oil dressings which do not accelerate healing and whose only virtue lies in their atraumatic properties.

(b) Only vitamins C and K, in the diet, can be shown to directly influence healing. Lack of vitamin C results in fibroblastic aplasia affecting collagenation of proliferated fibroblasts and their maturation to fibrous tissue. The end result is a weak, irregular scar. He estimates the patient’s daily requirement of vitamin C at 75 to 100 mg. of ascorbic acid.

In vitamin K deficiency the production of prothrombin and capillary clotting are retarded.

Upon electrolyte and protein balance the author advances the ideas that:

(a) All wounds, especially fractures and suppuration, show an initial protein loss and the normal “lag” period of 4 days is prolonged by a low protein or high fat diet. In addition, edema, the most powerful enemy of repair, is largely due to electrolyte disbalance resulting from hypoproteinemia.

Patients should at once be put on a high protein and low fat diet, augmented if necessary by parenteral injections of plasma or a solution of amino acids and dextrose.

(b) Local edema may be prevented by: maintenance of tissue support, obviating of gravitational fluids, and maintenance of movement. Indiscriminate dextrose-saline administration, however, disrupts the fluid balance and it is wise to use “No more than one and a half times the amount of urine, except with extensive burns.”

The writer recommends various high protein diets which make liberal use of vegetable proteins.

—DOUG LAKE, 44.

TREATMENT OF 100 WAR WOUNDS AND BURNS


This is an account of experiences gained in the treatment of wounds sustained for the most part by airmen shot down over England.

1. The majority of the wounded were admitted to hospital within 6 hours of injury. Wounds of limbs constituted the greatest number of cases, the majority being compound fractures. Excision in early cases, debridement in later cases, followed by adequate immobilization in plaster of paris, have been successfully employed.

2. Removal of retained foreign bodies was incidental to general cleansing of the wound. In early cases in good condition a careful search was undertaken, especially in neighborhood of joints. Preliminary radiographs were essential. The multiple minute fragments of aircraft-cannon shell were left alone. Puncture holes were painted with iodine or gentian violet without applying dressings. Through-and-through bullet tracks, if not involving bone or large vessels, were left alone.

3. No wound was closed until completely excised. Time of primary suture was 12-14 hours after injury for upper half of body and 6 hours for lower limbs. No muscle, tendon or nerve was sutured. Such cases were sent to a neurological centre for secondary suture.

4. Wounds were lightly packed with acroflavine-paraffin gauze. Wounded limbs were then immobilized in plaster of paris. No case was treated by Carrel-Dakin irrigation. Plasters were skin tight except in wounds left open and expected to ooze considerably. In these, much cotton wool was packed over the wound. No “windows” were employed. Plasters were removed and stitches taken out in 7-10 days in soft tissue injuries and 14-21 days in fractures. Wounds were then re-dressed with thin strips of wick. Plaster immobilization was continued until the wound had healed completely.

5. For burns, preliminary cleansing under anaesthesia was carried out, followed by application of the triple tan
solutions. Coagulation was accelerated by the use of an electric hair drier. The tan was applied without any break in the continuity over the flexor surfaces of joints. A degree of splinting was therefore produced which was not considered unbeneﬁcial.

6. Chemotherapy is not necessary as a routine in the treatment of early wounds. Its best use as a prophylactic agent in large wounds in which it has not been possible to clean completely.

7. Pentothal intravenously has proved a safe and satisfactory agent for routine use in war surgery. The use of local anaesthesia in treatment of war wounds is strongly deprecated.

—A. D. Tompkins, '43.

THE TREATMENT OF WAR WOUNDS OF THE KNEE-JOINT

By A. G. Timbrell Fisher, B.C.M.B., Ch.B., F.R.C.S., St. Mark's Hospital;

Dr. Fisher stresses the seriousness of wounds involving a joint and the need for prompt and efﬁcient treatment. The following classiﬁcation of wounds of the knee-joint is given:

1. Small perforating wounds of entrance and exit—
   (a) Uncomplicated
   (b) With minor injury to bone and/or articular cartilage.

2. Small penetrating wounds with no point of exit and with retention of foreign body—
   (a) No damage to articular extremities.
   (b) Associated injury to bone and/or articular cartilage.

3. Lacerated wounds.
   Under general principles of treatment the author points out the need for:
   1. Immediate evacuation to the operating centre,
   2. Treatment of shock,
   3. X-ray examination of the affected knee in two planes.
   The use of local and general sulphanilamide therapy in preventing infections of wounds is discussed. Dr. Fisher concludes with an outline of the operative treatment of wounds of the knee-joint under the classiﬁcation presented above.

—S. Shapiro, '43.

HOSPITAL INFECTION IN WAR WOUNDS

By A. A. Miles and Collaborators,
B. M. J., 2, 895; 28/11/40.

Miles states that this type of infection is more prevalent than commonly sup­posed, and that it accounts for longer hospitalization than should be needed. It can be avoided by the most rigid aseptic and antiseptic techniques of manage­ment. Exacerbations of the wounds may result from “pocketing” of the septic tissue, or from outside contamination. Patients can be protected from these “protracted” infections and from “added” infections by: (1) Aseptic and antiseptic manipulation of wounds in and out of the operating room; (2) Recognition of potential reservoirs of infection in hospital environment and reduction so far as is possible, of such reservoirs. As examples of in­fection reservoirs the author cites: bed clothes, dust, baths, bed pans, ﬁngers, plasters and dressings contaminated with discharge, non-sterile instruments, and too frequent examinations by both authorized and unauthorized persons.

—F. Ross Howson, '43.

THE CRITICAL LATENT OR LAG PERIOD IN THE HEALING OF WOUNDS

Presidential Address to American Surgical Association

By Allen O. Whipple, M.D.

In more than half a century this was the ﬁrst time that a presidential address to this association had dealt with the subject of wound healing.

In the lag period or adjustment phase of wound healing there is initiated a sequence of deﬁnite processes giving rise to ﬁbroblastic fusion of wound surfaces. It is a composite biologic phe­nomenon of growth in which are recognized the important processes of: (1)
ameboid movement; (2) mitotic proliferation; (3) maturation of cells engaged in fusion of wound surfaces.

The normal lag period is four days, conditioned by: (1) local factors; (2) systemic factors.

Local factors—
1. Amount of killed or damaged tissue.
2. Vascularity of tissues involved.
3. Integrity of blood flow to damaged tissues.
4. Amount and character of exudate; embryonic tissue juices, products of cell destruction, glutathione and haemoglobin increase ameboid movements.
5. Number and character of infectious organisms.
6. Number and character of foreign bodies to be extruded or encapsulated.

Systemic factors—
1. Age of tissues.
2. State of hydration; dependent on fluid electrolyte and protein balance.
3. Nutritional balance; high protein diet accelerates healing since acid media are poor for bacteria and a fat diet retards the lag period.
4. Vitamin balance; Vitamin C is necessary for the production and maintenance of the intercellular cement substance; its deficiency, therefore, prolongs the latent period.
5. State of the circulation and normality of the blood picture. The problems presented in the repair and management of the wound call for both a surgical and physiological consideration, especially in relation to the critical lag period of wound healing.

—E. G. Butt, '43.
Diplomacy! Does this mean being civil in the face of provocation, or hypocritical to somebody’s face who is slightly more influential than we are?

Originally it carried the former meaning and was employed for purposes of peace-making. The term is now too often employed for a deceitful racket whereby one hopes to gain by his false “politesse.” The bald facts often stir up a bit of unrighteous indignation in venerable doctor-saints but, surprisingly enough, they often react most generously when one of their time-polished methods is dropped naked at their feet—providing “superior” petrification is not too far advanced.

What do you think of the conglomeration of over-ripe brevities that drop from the lecturer’s brief case on to the student’s notebook and hence, as a visual image, to the examination page in order to get a mighty “A”? What do you think of some of our courses, lecturers, clinicians, etc.?

THE JOURNAL will publish a list of “suggestions to sages” in the next issue—if you submit them.

We might make use of the enema Hitler and the wash-out Mussolini to relieve some future medical student’s chronic griping.

What say?
THE PHARMACOLOGICAL BASIS OF THERAPEUTICS

By L. S. GOODMAN, M.A., M.D., and A. GILMAN, Ph.D.

(1383 pp., 126 diagrs., 67 tables. Macmillan, New York, 1941)

The subtitle “A Textbook of Pharmacology, Toxicology and Therapeutics for Physicians and Medical Students” describes the scope of this new book that has as its purpose the correlation of pharmacodynamics and therapeutics.

The pharmacology of the drugs affecting the different organs and systems of the body is discussed as in the conventional textbook. A feature which appeals to the student who is just beginning his study of the *Materia Medica* is the description of the common preparations of the drugs. The distinctive characteristic of this book, however, is the discussion of “Therapeutic Uses,” including indications and details concerning dosage and administration. The new chemotherapeutic group of drugs is comprehensively considered and most of the recent contributions to the literature are examined. Sections of the book are devoted also to the drugs of endocrine origin and to the vitamins.

The authors present much data that is still controversial and abundant bibliographic references are given. This book has the disadvantage of being longer than the student beginning the study of pharmacology might desire, but it is excellently written and should find wide acclaim among senior students and physicians.

—F. C. HEAGY, '43.

FRACTURES AND OTHER BONE AND JOINT INJURIES

By R. WATSON-JONES, M. Ch. Orth., F.R.C.S.

(2nd Edit., 724 pp., 1034 illus. plates. E. & S. Livingstone, Edinburgh, 1941)

This second edition of a popular book was brought out by the demand of the war for a section on casualty surgery. This new section contains an authoritative review of general techniques and recent
advances. The indications for amputation are curtailed to a minimum while a prominent place is given to the closed plaster technique.

The author discusses bone and joint pathology of each region in detail, both as regards diagnosis and treatment. Recent literature on the subject is carefully considered and references are given in footnotes. It is interesting to see the mention of Penicillin in wound infection since it promises even more than the sulphonamide drugs as a chemo-therapeutic agent. The book is profusely illustrated with excellent photographs.

This volume certainly will take its place as a reliable orthopædic text and should prove a valuable reference work for the student and general practitioner.

—R. W. Hewson, '43.

A HANDBOOK OF FIRST AID AND BANDAGING


(629 pp., 239 diagrs. Ballière, Tindall and Cox, London, 1941)

The authors are to be congratulated for having produced a volume which so thoroughly encompasses all phases of first aid. The book is clearly written, well illustrated, practical, up to date and remarkably complete—a first rate book.

Although the book repeatedly stresses the importance of obtaining qualified medical aid as soon as possible in cases of emergency, it deals so thoroughly with signs, symptoms and treatment that, should medical help not be available, the first-aider would have the most accurate guidance available. Of special interest is the chapter on first aid in maternity cases. This is a unique subject to be covered in a first aid book and is especially appropriate in these times of air raids and similar emergencies where child birth is apt to be precipitated.

It is perhaps somewhat too advanced for the ordinary layman but this volume should be of special value as a text for the instruction of A. R. P. workers and for all those engaged in war-time medical and first aid work.

—G. A. Clark, '43.
To Study the Phenomena of Disease without Books

Is to Sail an Uncharted Sea.

— Osler.

RECENT ACCESSIONS TO THE MEDICAL SCHOOL LIBRARY

OCTOBER 27th, 1941

Adair, ed.—Obstetrics and Gynecology, in 2 volumes. 1940.
American Association for the Advancement of Science. A Symposium on Human Malaria. 1941.
American Congress on Obstetrics and Gynecology. Proceedings. 1939.
Bailey, Hamilton, ed.—Surgery of Modern Warfare, complete in five parts. 1940-41.
Ballenger and Ballenger—Diseases of the Nose, Throat and Ear; 7th ed. 1938.
Baur—Human Heredity. 1931.
Belilios—Handbook of First Aid and Bandaging. 1941.
Bing—Textbook of Nervous Diseases—from the 5th German ed. 1939.
Campbell—Operative Orthopedics. 1939.
Cobb—Foundations of Neuropsychiatry; 2nd ed. 1941.
Eliason—First Aid in Emergencies; 9th ed. 1938.
Fishberg—Heart Failure; 2nd ed. 1940.
Flexner and Flexner—William Henry Welch and the Heroic Age of American Medicine. 1941.
French and Alexander—Psychogenic Factors in Bronchial Asthma. 1941 (Psychosomatic Medicine Monograph No. 4.)
Green—Mechanisms of Biological Oxidations. 1940.
Hall—Life and Confessions of a Psychologist. 1923.
Heagerty—The Romance of Medicine in Canada. 1940.
Hess and Lundeen—The Premature Infant. 1941.
Holmes and Ruggles—Roentgen Interpretation; 6th ed. 1941.
Jones—Digestive Tract Pain. 1938.
Kardiner—The Traumatic Neuroses of War. 1941 (Psychosomatic Monographs Nos. 2 and 3.)
Kerr—Forensic Medicine; 3rd ed. 1939.
Kessler—Accidental Injuries; 2nd ed. 1940.
Kling—The Synovial Membrane and the Synovial Fluid. 1938.
Kolmer—Approved Laboratory Technic; 3rd ed. 1941.
Krusen—Physical Medicine. 1941.
Ladd and Gross—Abdominal Surgery in Infancy and Childhood. 1941.
Lawrence—The Scientific Photographer. 1941.
McCarty—Diagnosis and Treatment of Diseases of the Hair. 1940.
MacKee—X-Rays and Radium in the Treatment of the Skin; 2nd ed. 1938.
Meigs—Tumours of the Female Pelvic Organs. 1934.
Miall, ed.—A New Dictionary of Chemistry. 1940.
Miller—The Neuroses in War. 1940.
Novak—Gynecological and Obstetrical Pathology. 1940.
Nygaard—Hæmorrhagic Diseases. 1941.
Pardee—Clinical Aspects of the Electrocardiogram; 4th ed. 1941.
Pelouze—Gonorrhea in the Male and Female; 3rd ed. 1941.
Pohle—Clinical Röntgen Therapy. 1938.
Pohle—Theoretical Principles of Röntgen Therapy. 1938.
Ross—Lectures in War Neuroses. 1941.
Smith—Plague on Us. 1941.
Thoma—Oral Pathology. 1941.
Todd and Sandford—Clinical Diagnosis; 9th ed. 1941.
Watson-Jones—Fractures and Other Bone and Joint Injuries; 2nd ed. 1941.
Wilder—Clinical Diabetes Mellitus and Hyperinsulinism. 1940.
Williws and Keys—Cardiac Classics. 1941.
Wilson—Neurology, in 2 volumes. 1940.

Yearbooks for 1940—

- Eye, Ear, Nose and Throat
- General Medicine
- General Surgery

General Therapeutics
Obstetrics and Gynecology
Pediatrics

Physical Therapy
Radiology
Urology

A file on “War Information” is maintained in the Library for the ready use of those interested in the latest work on “Wound Surgery,” “Treatment of Burns” and “General Medicine and Surgery” as related to modern militaristic needs.