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MEDICAL DISEASES OF THE WAR

BY

ARTHUR F. HURST, M.A., M.D.(Oxon.), F.R.C.P.

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SECOND EDITION
REVISED AND ENLARGED

LONDON
EDWARD ARNOLD
1918

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To

MY WIFE

TO WHOSE PATIENCE AND SKILL

MANY STAMMERING, APHASIC AND DEAF SOLDIERS

OWE SO MUCH

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PREFACE TO THE FIRST EDITION

I HAVE had constant opportunities of studying every phase of the various diseases occurring on active service, first as physician and neurologist to a number of military hospitals in London and to the New Zealand Hospital at Walton-on-Thames, then as a member of the Medical Advisory Committee for the Prevention of Epidemic Disease in the Mediterranean Expeditionary Force in Lemnos, and subsequently in Salonica, where I also acted as Consulting Physician to the British Forces.

This book is a record of my own observations, amplified by a study of the literature so far as time and opportunity have allowed. I have read most of the papers bearing on the subjects dealt with, which have been published in the English medical journals, together with a number of the more important French contributions and a small number of those which have appeared in the German language. The principal references are given at the end of each chapter.

I should like to add, for the benefit of those who are more familiar with my former name of Hertz than with my new name of Hurst, that I have made the change because under present conditions it is natural for one of English birth and English descent for several generations to be unwilling to retain a German name.

ARTHUR F. HURST.

November, 1916.

PREFACE TO THE SECOND EDITION

THE present edition has been revised throughout and brought up to date after reading the available literature in English, French, Italian, and German up to the end of 1917. In the first edition I stated that the difficulties in the way of scientific classification of functional nervous disorders in soldiers was so great that I would not attempt one. Increased experience as Neurologist to the 3rd Southern General Hospital at Oxford, and since December, 1916, to the Royal Victoria Hospital, Netley, has made me more venturesome, and the first eleven chapters of this edition have replaced the single chapter on war neuroses in the original book. A new chapter has been added on tetanus, and the chapters on spirochætal jaundice, soldier's heart, and war nephritis, have been largely rewritten.

I wish to thank Major G. W. Crile, Lieut.-Col. F. W. Mott, Lieut.-Col. C. M. Wenyon, Mr. Clifford Dobell, Surg.-Gen. Sir Bertrand Dawson and Captain W. E. Hume, and Dr. P. P. Laidlaw for the loan of drawings and microphotographs, and my colleague at Netley, Captain J. L. M. Symns, for his constant help in the investigation of neurological problems.

ARTHUR F. HURST.

March, 1918.

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PART I.—WAR NEUROSES

PART I

WAR NEUROSES

CHAPTER I

PREDISPOSING CAUSES

The chief predisposing causes of war neuroses are congenital nervousness, a previous nervous or mental breakdown, concussion and chronic alcoholism.

Some men are constitutionally brave; others are constitutionally timid. Among the former are a few lucky ones, who do not know what fear is; their bravery is so natural to them that it is hardly a virtue. "Hunger and thirst could not depress them," wrote the Student in Arms. "Rain could not damp them. Cold could not chill them. Every hardship became a joke. They did not endure hardship, they derided it. . . . As for death, it was, in a way, the greatest joke of all." But most men feel frightened when first exposed to the danger of the trenches; they perspire profusely, their hands shake, their hearts beat rapidly, and each time they hear a shell whistling by they duck their heads. Their bravery is a genuine virtue, as it consists in the complete suppression of their natural fear. As a Student in Arms wrote, "Your teeth may chatter and your knees quake, but as long as the real you disapproves and derides this absurdity of the flesh, the composite you can carry on." In the course of time such men generally become more or less accustomed to the sights and sounds which at first frightened them, and the necessity of suppressing the emotion of fear becomes more and more rare. The constitutionally timid, on the other hand, are more liable than the average soldier to all war neuroses, and if

blown up by a high explosive shell, they are almost certain to develop hysterical symptoms in addition to the symptoms due to concussion, which alone constitute pure shell-shock. They have not the strength of will required to suppress the fear they feel, often even before they reach the front, and they break down as soon as they hear the sound of distant guns. These men, well described by Buzzard as "martial misfits," are frequently able to live a normal life as civilians, but they are totally incapable of adapting themselves to a soldier's life. An officer, typical of many similar cases, obtained his commission in July, 1915; he had always been nervous and subject to palpitation and diarrhœa when excited. Directly he reached the front he became sick with terror, sweated profusely and had constant palpitation and diarrhœa. At the end of a month he was quite unable to carry on.

Some martial misfits are easy to recognise by their appearance and their conversation, and the majority themselves recognise their disability. The number of this type has greatly increased since the introduction of compulsory service. The artistic temperament is common among them, although it is of course far from being incompatible with bravery. In other cases the discovery that they are martial misfits comes as a surprise to themselves and their friends. A keen territorial officer of many years' standing, who had been fretting during the first eighteen months of the war because he had been kept at home for training purposes, discovered to his horror that he was totally incapable of facing the strain of active service at the front. He became so emotional and tremulous as soon as he approached the trenches that he had to be sent home.

Contrary to what might have been expected, soldiers from the Dominions appear to be no less liable to functional nervous disorders than English and Scottish troops. The French have probably suffered rather more than the British. Neuroses have been very common in Germany, but Professor L. R. Müller, who was consulting physician to the Turkish armies, states that he neither saw nor heard of any cases of hysteria or other functional nervous disorders among them.

Men who have previously suffered from neurasthenia or mental breakdown, whatever may have been the cause, and men with a

family history of mental disease are particularly liable to develop all forms of war neuroses. Concussion caused by an injury to the head even years before makes a man specially liable to develop nerve symptoms if exposed to shell-shock, unless it was treated by more prolonged rest than is usually the case.

A man with a good family history, who has never suffered from any nervous disability, only develops war neuroses, including shell-shock, under exceptional circumstances. A single explosion, for example, is unlikely to give rise to symptoms apart from those due to concussion, unless he is already weakened by a long period of physical fatigue combined with severe mental strain; more frequently serious symptoms only develop after exposure to a third or fourth explosion.

Chronic alcoholics often benefit greatly from the discipline and comparatively healthy life they lead during training. But even if they have taken little or no alcohol since enlisting, they remain particularly liable to nervous disorders when exposed to the stress and strain of active service. The rum ration has been abundantly justified by its stimulating effect when an attack has to be made at dawn, but many men suffering from the early stages of various war neuroses have precipitated their final breakdown by attempting to keep themselves going by means of alcohol, and cases of this sort are particularly resistent to treatment.

CLASSIFICATION OF WAR NEUROSES.

A. PREDISPOSING CAUSES.

(1) The congenitally nervous or "martial misfits." (2) A previous attack of neurasthenia.	(3) A previous mental breakdown. (4) Chronic alcoholism.	(5) Concussion, even years before.

B. EXCITING CAUSES.	RESULTING NEUROSES.	TREATMENT.
(1) Exhaustion, due to— (a) Mental and physical strain. (b) Toxæmia of acute and chronic infections. (c) Insufficient food. (d) Excessive heat. (e) Pain and toxæmia of wounds.	NEURASTHENIA (nervous, suprarenal, and occasionally thyroid exhaustion). SOLDIER'S HEART.	Rest followed by graduated exercise; sometimes adrenalin or thyroid.
(2) Emotion, due to— (a) A single horrible incident. (b) Prolonged and repeated horror, fear, etc. (c) Constant pain from a wound.	STUPOR and AMNESIA, PSYCHASTHENIA (nightmares and day-dreams with secondary headache, insomnia and tremor; obsessions; phobias; tics). HYSTERIA (convulsions, tremors, paraplegia, mutism). HYPERTHYROIDISM — HYPERADRENALISM, with hormonic form of SOLDIER'S HEART. EXAGGERATED DEFENSIVE REFLEXES (especially "auditory-jump" and "visual-flinch" reflexes).	Rest, hypnosis. Suggestion with or without hypnosis. Persuasion, re-education, hypnotic suggestion. Rest, opium, belladonna. Quiet and isolation.
(3) Explosion of high power shells— (a) Concussion. i. Aerial. ii. Contact (burial, sandbags, etc.) (b) CO poisoning.	SHELL-SHOCK: an organic basis (concussion symptoms), often with superadded hysteria (paralysis, deafness, mutism, etc.).	Rest for organic basis; persuasion, re-education, and rarely hypnotic suggestion for hysteria.

C.—CONDITIONS PREDISPOSING TO THE DEVELOPMENT OF SPECIAL SYMPTOMS.	RESULTING NEUROSES.	TREATMENT.
(1) Family or personal history of epilepsy.	EPILEPSY ... ... HYSTERICAL convulsions	Bromide. Hypnotism.
(2) Mental deficiency (3) Mental disease	INSANITY.	
(4) Syphilis	CEREBRAL SYPHILIS: TABES: G.P.I. ...	Hg.: K.I.: salvarsan.
(5) Ocular defects, already present or produced simultaneously.	ASTHENOPIA and headaches ... ... HYSTERICAL blindness	Glasses. Hypnotic suggestion.
(6) Aural defects, already present or produced simultaneously.	HYSTERICAL deafness; tinnitus	Hypnotic suggestion for incomplete deafness; pseudo-operation for complete deafness without mutism; re-education.
(7) Previous or simultaneous disease, injury or wound of a limb.	HYSTERICAL paralysis, spasm or tremor of the limb.	Persuasion and re-education.
(8) Previous speech defect (mutism, aphonia, stammering).	HYSTERICAL mutism, aphonia, stammering.	Persuasion, suggestion and re-education.
(9) Excessive smoking	TREMOR; SOLDIER'S HEART.	
(10) Passed emotional disturbances or illnesses, which may since have been forgotten.		Hypnotic suggestion. *Not* psycho-analysis.

CHAPTER II

EXHAUSTION RESULTING IN NEURASTHENIA AND SOLDIER'S HEART

Etiology.—Exhaustion may result from physical and mental strain, the toxæmia of acute and chronic infections, insufficient food, excessive heat, and the pain and toxæmia of wounds.

Although it would require a very exceptional nervous system to pass unscathed through twelve months or more of war under modern conditions, uncomplicated neurasthenia due to prolonged physical strain, the utter nervous exhaustion caused by pure physical fatigue, has been comparatively rare since the battle of the Marne. But there were many cases after the retreat from Mons, and it says much for the spirit and discipline of the original Expeditionary Force that there were no more.

Major A. Corbett-Smith gives a vivid picture of what the Army endured. "Oh, the inexpressible weariness of it! No torture is more refined than that of preventing a worn-out human being from sleeping; and here it was experienced to the full. Hunger was long since forgotten, but a red-hot thirst remained. . . . On, ever on, for hour after eternal hour, riding or trudging through the inky darkness, never a halt. . . . How the troops did it I cannot tell. It was not the triumph of will over the exhausted body, for the sense of volition had fled, and men were mere automata in their movements. The legs jerked forwards as those of a clockwork toy. Had the men halted they would never have got moving again; the clockwork would have run down. . . . Every muscle of the body ached with an intolerable dull throbbing; a deadly coma crept through the brain and dragged at the eyelids." And this was only the fourth night of the fourteen which elapsed before the exhausted Army turned and routed the Germans at the battle of the Marne.

Life in the trenches is always accompanied by a state of nervous tension. Danger is ever present, and during periods of

activity a man can only sleep at odd moments, and the night may only bring increased responsibility and anxiety instead of rest, especially to young officers. Although most men become gradually accustomed to the sights and sounds, which at first frighten all but the few who do not know what fear is, the nervous system does not wear well, and unless regular periods of rest from the mental strain are given, the majority of men gradually become irritable, restless, and unable to perform their duties satisfactorily.

The continuous and monotonous strain of active service in the Navy might have been expected to result in functional nervous disorders, but the fine spirit of confident superiority in the men and the excellent hygienic conditions have, according to Surgeon-General H. D. Rolleston, prevented the development of mental, neurasthenic, or hysterical symptoms in any but exceptional cases.

The sanitation of the Army in France has been so wonderfully efficient that the second great cause of neurasthenia—acute and chronic infections—has been less prominent than in any previous campaign. But comparatively slight toxæmia, such as that due to diarrhœa, trench fever, influenza or oral sepsis, which is not sufficiently severe to make a man go sick, may cause nervous exhaustion, which would have been prevented if sufficient rest had been taken. A subaltern in the Flying Corps, who was actively engaged almost daily from September, 1914, passed unscathed through all the fatigue and excitement until January, 1915, when he had an attack of acute tonsillitis. He was taken to a hospital, where there was a shortage of food, and he was then conveyed in a half-starved condition to England, the journey occupying over thirty hours. The fatigue and insufficient food were not new experiences, but the addition of the intoxication caused by the tonsillitis, which by itself would have only led to a few days of illness, was sufficient to cause a condition of severe nervous exhaustion, the recovery from which was, however, remarkably rapid. In the early months of the war I saw several cases of neurasthenia following anti-typhoid inoculation, when the patient had to march a long distance immediately afterwards, but no more occurred after the authorities wisely issued an order that all inoculated men should be off duty for forty-eight hours.

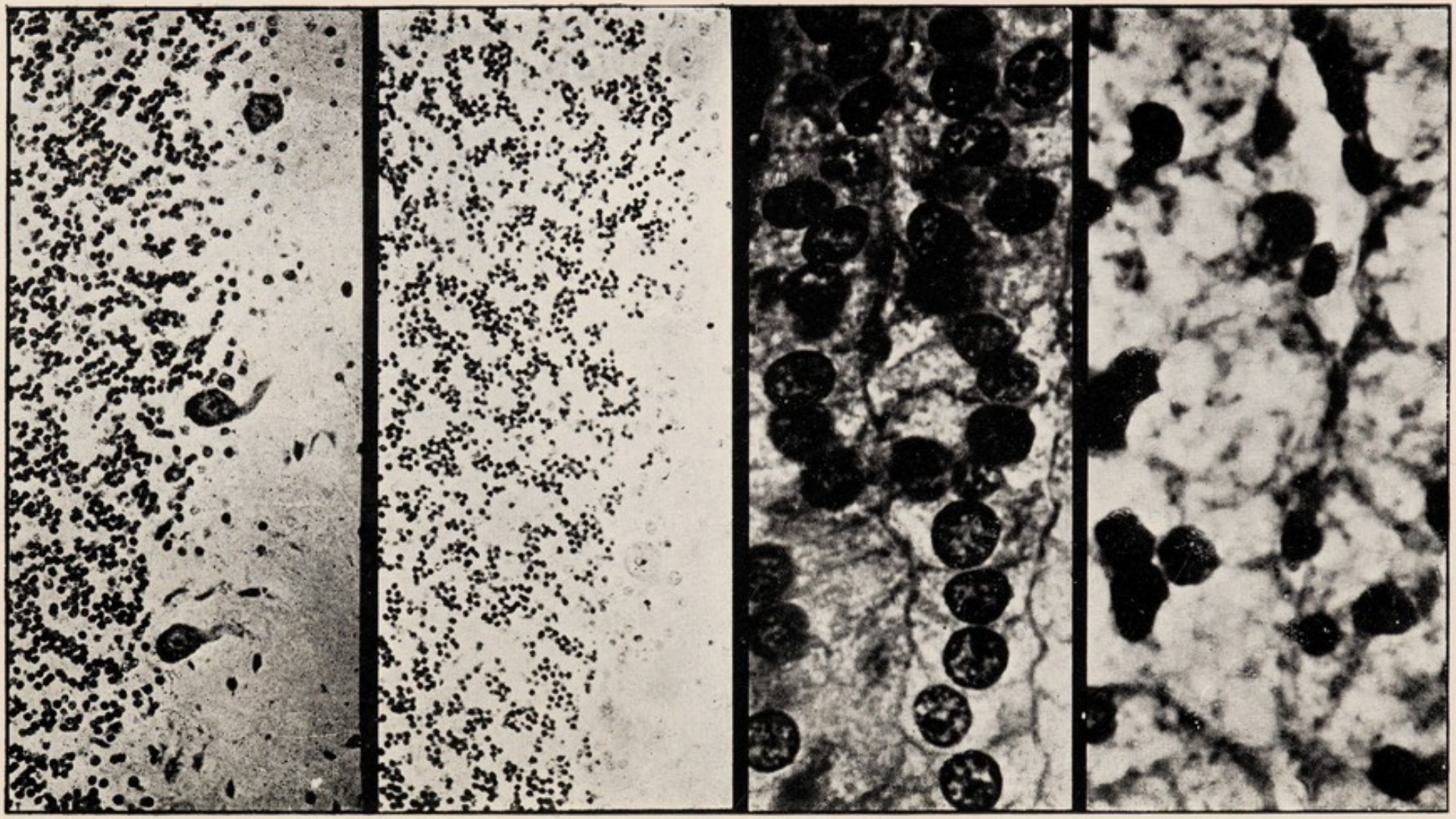
The Armies in other parts of the world have, unfortunately, not shared the immunity from serious epidemics with the Army

in France. Few men remained on Gallipoli for more than a month after August, without suffering from chronic diarrhœa or falling a victim to dysentery, paratyphoid fever, or epidemic jaundice, and often to more than one of these infections at the same time. Added to this was the impossibility of withdrawing the troops from the shell-swept area, as is done when necessary in France, for every corner on the Peninsula was exposed to hostile fire. Thus it came about that there was hardly a man at the end of November, 1915, who was not suffering more or less severely from nervous exhaustion due to toxæmia and prolonged mental strain. I have seen several men, who had carried on to the end without going sick at all, but found even a year after the evacuation that they were unable to stand the strain of active service in some other war area, although they had had many months of comparative rest after they left Gallipoli.

In Salonica conditions were more favourable, except during the summer of 1916, when the hot weather and the virulent form of malaria, for which the Struma Valley has always borne an unenviable reputation, were the chief causes of many cases of neurasthenia. The effect of the numerous infections rife in Mesopotamia and East Africa has been aggravated by the great heat, and at times by the insufficient supply of food. During the autumn of 1916, the majority of uncomplicated cases of neurasthenia reaching England came from Mesopotamia ; in several cases there was a history of actual heat-stroke.

The pain, toxæmia, and loss of blood which may result from severe septic wounds often cause neurasthenia, especially if the soldier is already over-fatigued from mental and physical strain. The frequency and importance of these cases are too often neglected by those who confine their interest to the surgical treatment of their patients.

Pathogenesis.—Mental and physical strain and severe pain lead to exhaustion by their direct action upon the nervous system, and indirectly by their action upon the suprarenal glands, together with the liver and probably the thyroid. Prolonged muscular exertion and great emotions, especially fear and anger, have been shown to produce chromatolysis of the brain cells. This is well seen in Crile's microphotographs of the brains of rabbits and cats, which have been subjected to great exertion,



Normal Cerebellum. | Cerebellum of Exhausted Soldier. | Normal Suprarenal Cortex. | Suprarenal Cortex of Exhausted Soldier.

Fig. I. Microphotographs of brain and suprarenal cortex of a soldier who died from exhaustion in the retreat from Mons. (After Major G. W. Crile, M.O R.C., U.S. Army).

acute and chronic fear, and prolonged insomnia, and of a soldier, who died from exhaustion caused by excessive physical and mental strain, hunger and thirst during the retreat from Mons (Fig. I).

At the same time the overaction of the suprarenal glands, which, as I shall presently describe, is a constant feature of excessive muscular activity, fear, anger, and pain, leads to disintegration of their cells, which show loss of cytoplasm and misshapen eccentric nuclei, especially in the cortex, in which perhaps a precursor of the adrenalin of the medulla is formed (Fig. I). Similar changes, together with loss of glycogen, are found in the liver. No other organs show any changes, but in the case of the thyroid gland this may be due to its peculiar structure, which makes it difficult to recognise the histological changes caused by exhaustion.

The physical signs of exhaustion disappear after sleep, and the cells of the central nervous system, suprarenal glands and liver are simultaneously restored to their natural condition, unless the stimulus is very excessive, when a considerable period of rest may be necessary before complete restoration occurs, and some nerve cells may be permanently destroyed. This explains how the apparently exhausted Army, which had hardly slept during the retreat from Mons, was yet able after two or three days' rest to turn and defeat the Germans in the battle of the Marne.

Similar changes occur in the brain as a result of acute and chronic infections. These are seen in Crile's microphotographs of men who have died from paratyphoid fever and streptococcal septicæmia. The observations of Sergent and others in France and of Elliott in England have made it probable that the muscular weakness and feeble circulation in certain acute infections, such as typhoid and paratyphoid fever, bacillary dysentery and malaria, are not so much due to the effect of the toxins on the skeletal muscles and myocardium as to their effect upon the suprarenal glands, the neurasthenia, which is a common sequel of these infections, being largely due to suprarenal exhaustion.

Symptoms.—The symptoms of neurasthenia in soldiers do not differ from those of the neurasthenia of civil life. Headache, rapid fatigue on mental and physical effort, and difficulty in concentration are always present. The appetite often remains excellent and the digestion good, but constipation is common.

A fine tremor of the hand is very common in men suffering

from neurasthenia. I have noticed it in officers who have been home on leave for a few days and have regarded themselves as perfectly fit. The symptom tends to be very persistent and is often a source of great worry to the patient, who regards it as an indication of the presence of some serious nervous disease.

The symptoms described under the name of "soldier's heart" may be prominent (*vide*, p. 287). In most cases the blood-pressure is low, corresponding with the fact that the blood-pressure of the average tired soldier in front-line trenches is slightly subnormal. In some cases, especially when the neurasthenia has developed after paratyphoid fever or malaria, the symptoms have been exactly what might be expected to result from hypo-adrenalism; the extreme asthenia, low blood-pressure, and feeble digestion characteristic of Addison's disease have been present, but instead of becoming progressively lower, the blood-pressure slowly rises as the general condition improves.

In a much smaller group of cases, all of which have followed prolonged mental strain, the patient's mental and physical condition has resembled very early myxœdema, and treatment of the hypothyroidism by small doses of dried thyroid gland has led to rapid improvement, when the condition had become stationary after the initial progress which had resulted from rest. Most of the cases have been sent home in company with congenital mental defectives, on account of being too slow and stupid to be of any value as soldiers, but further inquiry has shown that they may have been well educated and that they had been quite normal in civil life and during the first few months at the front.

Treatment.—Complete physical and mental rest are at first essential. The patient must be kept in bed until he no longer feels tired. In slight cases a week or fortnight may be enough, and it is rarely necessary to continue the complete rest for more than three weeks, though in severe cases two or three months may be required, especially after severe infections. Recovery is sometimes remarkably rapid when the symptoms are due to pure physical exhaustion. A private, who had fought in almost every battle since the retreat from Mons without being wounded and without having any leave, was admitted in March, 1917, in a condition of complete exhaustion. He was dazed and had a severe headache. He lay motionless in bed and would not answer

questions for four days. He then got up, said he was quite fit again, and asked to be sent back to duty.

For the first few days the patient should not read or have visitors, but in most cases these rules can soon be relaxed, and cheerful companionship with plenty of amusement and interest will hasten the cure.

Recovery is often greatly accelerated if a small dose of bromide, such as gr. v, is given two or three times a day. Opium must be avoided, as it often aggravates the symptoms and increases the excitability and restlessness of the patient. No alcohol should be allowed; it is very badly tolerated by almost all patients suffering from war neuroses, and especially from true shell-shock. A quantity which formerly produced no ill-effect is often sufficient to intoxicate. Progress is much hastened by combating insomnia, as exhaustion cannot disappear so long as the patient sleeps no more than three or four out of the twenty-four hours. I have found sodium diethyl-barbiturate (medinal) with acetyl-salicylic acid (aspirin) gr. xv the best combination. The dose of the former should be between 10 and 15 grains the first two nights, after which it should be reduced by gr. i every other night until only the latter is given; the dose of acetyl-salicylic can then be gradually reduced. In intractable cases, especially if nightmares are present, suggestion under hypnosis is very effective.

As soon as the patient is well enough to get up he should begin to help in the ward, and a few days later he should be given light employment out of doors. For—

> "The cure for this ill is not to sit still
> And frowst with a book by the fire;
> But to take a large hoe and a shovel also,
> And dig till you gently perspire."

The amount of exercise should be gradually increased, and the patient should not be discharged from hospital until he can do moderately heavy work, such as digging, without undue fatigue. I have found the best results are obtained if the patient is kept under a certain amount of discipline, and for this reason I prefer to send him direct from hospital to duty or to employment at home instead of to a convalescent or auxiliary hospital, where the comparative freedom from supervision and the lazy life too often make him unfit for any kind of military service.

CHAPTER III

NEUROSES RESULTING FROM EMOTIONS

The emotion produced by a very horrible incident, such as seeing a friend killed whilst talking with him, is sometimes sufficient to give rise to nervous symptoms. More commonly, however, such an event is only the culmination of a long series of emotional storms. In most cases, indeed, the final breakdown is not due to a definite incident, but is the result of the cumulative effect of fear, which gives rise to symptoms sooner or later or not at all, according to the temperament and the physical condition of the individual. It is often impossible to obtain an accurate history of the onset until the patient is convalescent, as in the early stages of his illness he is generally unwilling to talk about his experiences and becomes emotional when he does so. Even after complete recovery a horrible incident, which was the exciting cause, may remain obliterated from the memory, although it may have returned in dreams or its nature may have been elicited from the patient during hypnosis.

(i) Stupor and Amnesia

The stress and strain of active service not infrequently result in a condition of mild confusion, which may merge into deep stupor. The onset is often gradual; a man who has hitherto been alert and efficient becomes more and more dull, slow and silent, until finally—often after an incident which has caused unusually great terror or horror—he becomes too confused to carry on, and he may wander away from his unit in a condition of stupor with more or less complete amnesia. Exactly the same condition may follow actual shell-shock, except that the symptoms

develop acutely after the explosion of the shell and not gradually, and signs of organic injury are often present, at any rate during the first few hours after the onset. The symptoms may also closely resemble the automatism of epilepsy. They are more fully considered in the description of shell-shock.

The following case is the worst one I have seen or heard of, and is the only instance I have known in which progressive deterioration occurred for many months after the onset of symptoms.

Total Amnesia with Hysterical Paralysis, Contractures, Analgesia and Mutism due to Emotional Strain; Recovery of Memory after 22 months.—Pte. M., 23, with no personal or family nervous history, joined the Army in 1913. He was slightly wounded in the thigh in May, 1915, after serving from the beginning of the war. He returned to the front in October, 1915. He was quite fit until February 19th, 1916, when he had to be forcibly prevented from going over the parapet to attack some German mortars which were firing at his trench. He then became dazed, and on reaching the aid post he could not answer questions, but he obeyed simple commands such as to put out his tongue. He believed he was still in the trenches which were being heavily shelled; his pupils were widely dilated and he sweated profusely. His pulse was 140. Convulsive tremors of the head, trunk, and limbs constantly occurred. The bowels and bladder remained under control. When he reached a hospital in England on March 2nd, his expression was apprehensive, he started at all sounds both when awake and asleep. In his dreams he saw the ghosts of Germans he had bayoneted come to take revenge on him and he heard them fire at him. He was still unable to speak, but he answered questions by nods and signs and in writing. He was able to walk with assistance. He was treated by hypnotism, but his condition steadily deteriorated, except that the hallucinations disappeared.

I saw him first in December, 1916, eleven months after the onset. He was still unable to speak; all four limbs were now completely paralysed, except that he was able with a great effort to make slight movements at his left elbow joint. An extreme degree of contracture was present: the legs were rigidly extended with the feet plantar flexed; the arms were

extended and the fingers tightly clenched. It was almost impossible to produce any passive movements, but the contractures were entirely hysterical, as they relaxed completely under an anæsthetic and during sleep. Total anæsthesia and analgesia of the whole body, including the conjunctiva and cornea, were present, except that passive movements at the elbow were painful, and he occasionally suffered from toothache. The anæsthesia disappeared, at any rate to some extent, during sleep. Although deep pressure over the abdomen produced no sensation, the sensibility of the bladder and rectum appeared to be present, as he retained perfect control over them.

On December 15th, 1916, vigorous suggestion with the aid of an intralaryngeal electrode during light etherisation restored the power of whispering. It was then found that he had total amnesia: he had no idea who or what he was, he did not realise that his anæsthetic legs belonged to him, and he had no knowledge of the meaning of words. During the following months he learnt to talk a kind of pidgin-English, but the meaning of every word had to be taught, and he used each word in his limited vocabulary for a variety of meanings. All forms of drink were "tea," "hand" represented a hand and a glove and "to hand" was to hit; a word taught by other patients in fun would never be given up, so that all forms of meat, chicken, and fish were called "puss." His only numbers were one, and six, which represented anything more than one, except a very large number which was sixty-six, or a still larger number six-sixty-six. All attempts to teach ideas of time, space and colours failed, and he did not recognise any of his relations, even when his father was brought to him in the middle of the night in the hope that he might know him at the moment of waking. He remembered recent events and called people by names, which he often invented: a bald patient was "no-haired chick," two men who limped badly were "no-legged chick" and "six-legged chick"; all officers in uniform were "Major," and civilians "Mr." or, if friends, "Mr. Chick." In spite of treatment no improvement in the condition of his limbs had occurred by October, 1917, though he was able to sit up in a chair and enjoyed being taken out of doors. When the contractures relaxed under light anæsthesia or during sleep all superficial and deep reflexes

were normal and the muscles responded briskly to faradism. He delighted in childish toys, and in a general way his mind was that of a year-old child. He was quite happy, but was becoming very emaciated, as it was difficult to persuade him to eat.

On November 22nd, 1917, for no obvious reason he had a headache and became excited in the evening. His memory began to return during the night, and he talked incessantly. The next day he realised the deficiencies in his speech and wished to have them corrected. When told a word, he now repeated it correctly and remembered it, and began to form proper sentences. On November 24th I cured a man suffering from hysterical aphonia with a laryngeal sound in his presence. Though this had failed on many occasions since he learnt to whisper nearly a year before, it now cured him instantaneously to his intense delight. He lost his voice once more on the 27th, but it returned on passing the sound again. This time he felt something snap in his head, and immediately afterwards he talked quite normally and his memory of his home and his past life flowed back. His father came the next day, and he knew him at once. He soon remembered his experiences in France, but his life in the hospital was almost a blank, as it seemed to him that he was in France only a few days instead of 21 months ago. He had a vague recollection of very recent events, and he knew the men in the ward, but did not remember friends who had gone out only a week before. He remembered "feeling funny with a buzzing in his head," then "something in his head was suddenly relieved," and the buzzing stopped when his memory returned.

(ii) Psychasthenia

Prolonged mental strain may give rise to the purely mental symptoms, which can be most conveniently grouped together under the somewhat unsatisfactory name of psychasthenia. The most important and common of these are nightmares and day-dreams, which in turn give rise to insomnia, headache, and tremor. Obsessions and phobias are less common; uncomplicated tics are curiously rare.

Nightmares and Day-dreams.—Nightmares are very common, and many men live through their most terrifying experiences

night after night in their dreams. One officer was found every morning sleeping on the floor, as he invariably dreamt that he was fighting on the parapet, which was represented by his bed, and that he finally saved himself by tumbling into the trench, which was represented by the floor. In some cases the dream is forgotten, the patient only recollecting that he woke with a start and found himself in a cold sweat. In severe cases the patient constantly thinks of his horrible experiences whilst awake, and he may even have hallucinations, which occasionally result in insane conduct. The terrified aspect and the constant tremor, rapid pulse and profuse sweating, which sometimes continue for many weeks after the onset of symptoms, are to a great extent due to the mind remaining fixed on past experiences.

Nightmares cured by Hypnotism.—A nineteen-year-old soldier saw an old woman's head blown off by a shell whilst he was standing near her in front of her cottage. The episode constantly appeared as a nightmare, which prevented him from getting any real rest, and hardly an hour passed in the day in which the scene was not vividly recalled to his mind. The want of refreshing sleep at night and the disturbing thoughts during the day made it impossible for him to recover from the neurasthenia, which had resulted from the exhausting experiences he had passed through. Suggestion under hypnosis repeated on three occasions resulted in the disappearance of the nightmares and of the constantly recurring recollection of the horrible scene he had witnessed, and his general condition consequently improved so rapidly that he was able to return to duty a month later.

The disturbed nights caused by war-dreams almost invariably result in headache, which is worst in the morning and only disappears towards evening. Many patients resist the desire to sleep during the day, as they fear a recurrence of their dreams, or they think that sleeping in the daytime will make their nights still less restful. A condition of profound exhaustion may ensue, and the patient loses his appetite and feels intensely depressed.

Obsessions.—Unaccustomed responsibility may give rise to obsessions in men whose resistance has been lowered by the strain of prolonged service at the front, or who are predisposed by having had a mental breakdown or by having a family history of mental disorder.

Obsession of Fear cured by Hypnotic Suggestion.—A captain, who, with one lieutenant, was the sole survivor among the officers of his battalion at the battle of Ypres, received the D.S.O. for his gallant conduct on that occasion, but the enormous and unexpected responsibility which devolved upon him when he was left in command was too much for him in his physically exhausted condition. Though he had saved the remnant of his battalion through his own almost unaided efforts, he felt that he could never face any responsibility again, and that the next occasion he was in danger he would have so little control over himself and his men that he would disgrace himself. This obsession took complete hold of him, and he became more and more depressed in consequence. When, after many weeks spent in the trenches, he heard that there would be an attack at Neuve Chapelle the following day, he broke down completely, but with a supreme effort he got through the first day of the battle. Though he did very well, he was more miserable than ever in the evening and felt he could not face another day's fighting. He was invalided home and arrived in a condition of profound exhaustion and utter misery, as he thought he had disgraced himself for ever. His appetite remained good, but his nights were disturbed by bad dreams. Hypnotic suggestion was followed by such rapid improvement that he was soon able to return to duty.

Phobias.—The same conditions which give rise to obsessions may cause phobias of various kinds. The fear of open spaces (agoraphobia) and of closed spaces (claustrophobia) are often associated with war neurasthenia. Numerous other phobias are also occasionally seen.

"*Hydrophobia*" *in an Exhausted Soldier.*—A private, 42 years old, worked between 16 and 18 hours a day unloading ships at the base from October, 1915. He broke down temporarily in January and May, 1916, but on both occasions returned to work after a short rest. In December he felt exhausted and suffered from severe headaches. He worried about his home, and lost confidence in himself. He felt his legs give way when he approached the water-side, and he gradually became obsessed with the idea that he could not control himself from falling into the water. When admitted to hospital on December 9th, he could not even wash his hands, as the sight of the water brought back

his fear; his legs trembled and he felt that he would fall. His mental condition was otherwise normal. By the time he reached England on December 15th his fear of water had disappeared. He now recalled the fact that one morning some years ago, after having been drunk the previous evening, he had been unable to walk over Waterloo Bridge on his way to work, as he felt he might throw himself into the water. There was never any real suicidal tendency, as he only avoided the water because he was frightened of it, and he never had any desire to end his life.

Tics and Facial Spasm.—I have seen several cases in which tics have developed during active service; in most instances they have been associated with hysterical symptoms, and many of the patients have previously suffered from tics. A side-to-side movement of the head, the original object of which is to relieve the pain caused by concussion of the cervical spine, is not uncommon. It can only be controlled for two or three seconds, and it persists during light hypnosis, but not during sleep.

Facial Spasm and Nightmares following Shell-shock.—A well-educated private was blown up by a shell, and developed severe bilateral facial spasm as soon as he regained consciousness. The spasms recurred without intermission during the day, but disappeared at once under hypnosis and during sleep. The condition was not a tic, as he had not the slightest control over the movements, and opposing muscles, such as the corrugator supercilii and frontalis, as well as the extrinsic and intrinsic muscles of the ears, which he had never been able to contract voluntarily, took part in the spasm. The spasm appeared to be associated with a dream which he had had every night since he was blown up: he saw with extreme vividness the grimaces of a German he had bayoneted in the face a few days before, during an attack in which he had killed two others, who never appeared in his dreams. The dream was unusually resistant to hypnosis, but as it gradually became less vivid, the spasm became less constant and severe. In another case a man developed a severe tic involving both platysma muscles and to a less extent the risorius, but I could not discover why these particular muscles were involved.

Disappearance of Spasmodic Torticollis after an Attack of Malaria.—A soldier developed very severe spasmodic torticollis whilst on light duty in England after having been sent back from

France on account of neurasthenia. It resisted all treatment from the onset on July 3rd until September 4th, when the patient suddenly became acutely ill with fever and delirium. This proved to be due to a return of malaria which he had had some years before in India. When at the end of a fortnight the rigors ceased as a result of quinine injections, it was found that the torticollis had completely disappeared, but slight hypertrophy of one sterno-mastoid was still present. There had been no relapse when he was discharged to duty six weeks later.

Treatment.—The general treatment of a man suffering from psychasthenia is identical with the treatment for neurasthenia. In slight cases nothing more is required, but special treatment is generally necessary for the more prominent psychasthenic symptoms.

In some cases a nightly dose of 30 gr. sodium bromide will cause *nightmares* to disappear, and in severe cases a combination of acetyl-salicylic acid with medinal, the dose of which is gradually reduced, as described in the treatment of neurasthenia, is often effective. But when a dream has persisted for any length of time, nothing but psychotherapy will remove it. In the form of hypnotism it should be practised at night, so that the patient can pass from the hypnotic state into natural sleep, the suggestion being repeatedly made that he will not dream. As an alternative, intelligent patients should be taught, as advised by Rivers, to think of their unpleasant war experiences occasionally during the day, and try to convert them from entirely painful into supportable memories, instead of attempting to banish them completely from their minds.

Phobias and *obsessions* sometimes disappear after rest and "therapeutic conversations," in which the cause of the symptoms is explained to the patient, and he is encouraged to hope that he will quickly lose them as his general condition improves. It is of the utmost importance that his confidence should be gained, and that he should feel that he can discuss his troubles with perfect freedom and without any fear of being misunderstood. In severe cases, and especially when the phobia or obsession is of a very distressing character, suggestion under hypnosis should be employed without delay. The result is generally very satisfactory, improvement occuring with remarkable rapidity.

Tics are more resistant to treatment, as they tend to persist

after the other symptoms have disappeared, and though they rarely continue during hypnosis, suggestion has comparatively little effect upon them. Re-education is the most valuable treatment. Regular exercises are given for the affected muscles, and in order that complete control may be regained over them the patient is also made to inhibit the movements by an effort of will for a certain period, the duration of which should be slowly increased, several times every day. I have seen a number of soldiers suffering from gastric symptoms due to aerophagy and eructation tics, who were cured by teaching them to clench a stick or cork between their teeth whenever they felt a desire to eructate.

Hypnotism is so useful in the treatment of psychasthenia and various other war neuroses that everybody who has to deal with soldiers suffering from these conditions ought to learn to hypnotise them himself. There is no difficulty about it. Every one of the numerous clinical clerks in my Neurological Department at Guy's Hospital who has tried has succeeded after the first or second attempt, and the process is not at all fatiguing either to the patient or the hypnotist.

It is quite unnecessary to use any elaborate methods, as soldiers are remarkably easy to hypnotise, partly owing to the strain of active service making them very suggestible and partly owing to their great desire to get rid of their distressing symptoms. The patient should be alone in a room or surrounded by screens, and lie comfortably with his muscles relaxed in the position he would assume if he were about to go to sleep. He should be told that the treatment will cure him rapidly, and examples of similar cases may be related to him. He is instructed to think of something pleasant and not to listen to what is said to him; he may, for example, try to picture his home to himself and imagine himself walking through the various rooms. He should fix his eyes for a few moments on a lens or other object before closing his eyes, but this is not essential, as shown by the ease with which patients with hysterical amblyopia and blepharospasm, who cannot open their eyes, can be hypnotised. One hand can be put firmly on the forehead and the other may stroke the eyes,

but this also is not essential. The patient is next told in a low monotonous voice that he is going to sleep, that he is getting drowsy, that he will soon be asleep, and so on, for two or three minutes. Curative suggestions are then repeated over and over again without attempting to ascertain whether he is really hypnotised, as the success of the treatment does not depend upon the degree of unconsciousness, and it is even unnecessary for consciousness to be lost at all. After five or ten minutes the patient is told to wake. He should not as a rule be examined at once, but should be allowed gradually to realise for himself how much better he is. In some cases a single treatment is sufficient, but more often it must be repeated once or twice, and occasionally several times.

(iii) Hysterical Symptoms

"Vouloir guérir un psychonévropathe, c'est engager avec lui une lutte morale, dont on doit sortir victorieux" (Roussy and Lhermitte).

Hysterical symptoms are symptoms which result from suggestion on the part of the patient himself (auto-suggestion) or on the part of somebody else (hetero-suggestion), and which are curable by persuasion and suggestion acting alone. The difficulty which has always been experienced in defining hysteria is due to the fact that there is no such thing as hysteria apart from hysterical symptoms. It might be supposed that the underlying mental condition which makes an individual liable to develop hysterical symptoms is hysteria, but there is no mental condition of this kind constantly present, as nobody is free from liability to develop hysterical symptoms if the suggestion is sufficiently strong, and when such symptoms have been removed by suggestion the individual may be perfectly well, and no underlying abnormal mental condition or "hysteria" may remain to be cured. Charcot taught that the hysterical individual could be recognised by the presence of permanent stigmata, of which he is unaware, and that these exist independently of the obvious symptoms, for the cure of which he seeks medical aid. Among the so-called hysterical stigmata are retraction of the field of vision, pharyngeal anæsthesia, and hemianæsthesia, which is often accompanied by diminution in the acuity of the special senses of the same side. My own

experience entirely supports Babinski's view that the stigmata are invariably produced by suggestion, generally by the observer himself. They are not permanent symptoms, the manifestations of the hysterical soil on which the new hysterical symptoms have grown, but like the latter they have been produced by suggestion and can be made to disappear by suggestion, only they are produced by hetero-suggestion instead of by auto-suggestion. Their presence cannot be taken as evidence that the symptoms which accompany them are hysterical, as individuals suffering from organic lesions and functional conditions other than hysterical, especially soldiers who have spent many months at the front, may be more suggestible than the average man, so that a careless examination may produce every stigma which is looked for. The doctrine of hysterical stigmata is most misleading, as their discovery may cause organic symptoms to be regarded as hysterical, and the organic element may be missed in conditions in which organic and hysterical symptoms are associated together. Conversely hysterical symptoms, which have developed as a result of very powerful suggestion in an individual, who is not more suggestible than an average healthy man, as not infrequently happens in soldiers, may be regarded as organic, because the methods used in looking for stigmata do not produce a sufficiently strong suggestion to cause them to appear. The nervous symptoms regarded as stigmata of hysteria vary in frequency according to the observer. They are very frequently found by those who have been taught to look for them as genuine signs of hysteria, but they are never found by those who know that they only occur as a result of the physician's suggestion.

Retraction of the field of vision has long been regarded as the most characteristic "stigma" of hysteria. Janet considered it to be "the emblem of hysterical sensibility in general," and it led him to describe hysteria as a condition due to retraction of the field of consciousness. Babinski and Froment quote the opinion of the distinguished French oculist, Morax, a former assistant of Charcot, from whom he learnt the supposed significance of the retracted field of vision. Although at first he continued to find it in almost every patient suffering from hysterical symptoms, his experience in the war has confirmed the opinion

he has now shared with Babinski for several years—that it never occurs unless it is produced by suggestion on the part of the observer, and that it is therefore not a stigma of hysteria at all. No patient ever spontaneously complains of it. In using a perimeter on a highly suggestible patient suffering from hysterical symptoms it is exceedingly difficult to avoid suggesting a narrow field of vision, the mere explanation of what is going to be done being often sufficient. Since recognising the fallacious method he had used for testing the fields of vision, Morax has substituted others, in which the finger or some other familiar object is used. At first they may seem less accurate, but they are in fact much more accurate, as suggestion can be more easily avoided.

No patient suffering from hysterical symptoms ever mentions *anæsthesia* when he is first seen, although asked to describe his complaints in the greatest detail. But after it has been produced by suggestion, he is aware of its presence and may now complain of the inconvenience it causes, but it never leads to burns or other injuries, such as result from the anæsthesia produced by organic lesions, and if unaccompanied by paralysis it does not prevent the performance of delicate movements, such as picking up a pin with the eyes closed, although cutaneous sensibility is essential for such purposes, as shown by the incapacitating effects of organic anæsthesia.

If great care is taken to avoid suggestion, hysterical anæsthesia is never found in a man whose sensory functions have not already been investigated. This fact was first pointed out by Babinski, and it has been confirmed by numerous authors in France, and with two possible exceptions by my own experience, although until I first visited Babinski's clinic in 1907 I had believed that anæsthesia was a common hysterical symptom and had found it very frequently in the well-known forms such as hemi-anæsthesia. The anæsthesia may vary from day to day according to the conscious or unconscious suggestion of the observer, and tends to disappear spontaneously if ignored. In investigating sensibility Babinski never allows the patient to compare one side with the other—a method which always suggests to him that some difference is present. He examines the various superficial and deep sensations over all parts of the body indiscriminately without any order, the patient simply pointing to the part stimulated and

stating what he feels, so that it is almost impossible for the patient to develop definite areas of anæsthesia to some or all stimuli as a result of suggestion.

It is natural that hysterical anæsthesia should develop very easily in an hysterically paralysed limb, when great care is not taken to avoid suggestion, as every one of a hundred people of average intelligence, who were asked by Adrian and Yelland whether they would lose the sensation in their hand if for any reason it became paralysed, answered in the affirmative. The anæsthetic areas which are often considered as characteristic of hysteria are simply the areas of anæsthesia which are likely to result from a careless examination, in which sufficient care has not been taken to avoid suggestion. My experience in soldiers as in civilians is in complete agreement with that of Babinski, who believes that when hysterical anæsthesia is present, it is almost invariably the result of suggestion on the part of the examining physician. In several published reports on war neuroses the history given clearly shows that this has occurred, as the anæsthesia has steadily increased at each examination, and in the only examples of hysterical anæsthesia I have seen among a very large number of soldiers suffering from hysterical symptoms the medical history sheet indicated how at each new examination by a medical officer a larger area of increasingly well-marked anæsthesia was observed. Roussy and Lhermitte not only failed to find anæsthesia in any patient suffering from hysterical symptoms who had not previously had his sensibility investigated, but they often failed to confirm its presence in patients whose medical history sheets showed it had been found a few days before, especially if a contra-suggestion was produced by reprimanding a patient who appeared to hesitate in responding to their tests of cutaneous sensibility.

In an investigation carried out with Captain E. A. Peters and Captain J. L. M. Symns we have found that when care is taken to avoid suggestion *pharyngeal anæsthesia* is never present in hysteria. The degree of sensibility of the pharynx varies to exactly the same extent from extreme hyper-æsthesia to almost complete anæsthesia in normal as in hysterical individuals. Pharyngeal anæsthesia, which has been regarded for forty years as an important stigma of hysteria, is thus devoid of any significance.

The stress and strain of active service render many men

unusually suggestible. They are, therefore, abnormally liable to develop hysterical symptoms as a result of auto- or hetero-suggestion. The greatest care is thus required to avoid aggravating hysterical symptoms, adding hysterical to organic symptoms, or producing new ones by an injudicious medical examination.

In their chief characteristics there is nothing peculiar about the hysterical symptoms which occur in soldiers, whether as a sequel of an emotion, true shell-shock, an injury due to a wound or an accident, or some acute illness. But they are so common that in two years I have seen more cases among soldiers than I had seen in men, women, and children in the previous ten years. It is not surprising, therefore, that hysterical manifestations are occasionally seen in soldiers, which differ from anything which one has seen or heard of in civilians, but the one invariable feature of all cases both in peace and in war is their production by suggestion and their curability by suggestion or persuasion alone.

It is unusual for hysterical symptoms to develop as the immediate result of a strong emotion. When this does occur it is due to the physical results of some overpoweringly strong emotion becoming perpetuated and exaggerated by auto-suggestion. Sudden extreme terror causes inhibition of activity instead of the physical preparation for flight, which is produced by the sympathetic and suprarenal stimulation following more prolonged but less excessive fear. The knees give way, the breath is held, and the "tongue cleaves to the roof of the mouth," rendering speech impossible. These are, perhaps, manifestations of the instinct to hide, as when flight is impossible, recourse must be taken to concealment. Similarly when the pain caused by an injury is so excessive that any further attempt to fight or to escape could only lead to death, bodily activity is depressed instead of stimulated and concealment takes the place of flight.

The inability to move or speak under these conditions is in most cases only momentary. But a man, whose nervous system is already suffering from the strain of active service, may be so suggestible that the incapacity is perpetuated and exaggerated, when he realises that he is physically unable to escape from his terrifying surroundings or call for help, and the possibility that this inability may be permanent and not merely a thing of the moment flashes through his mind. True hysterical paraplegia or

mutism is the result. These conditions are thus produced by auto-suggestion without physical aids.

The tremor, which is also common after sudden terror, has a similar origin. As Meige points out, the movement in well-marked cases consist of a sudden contraction of almost all the flexors of the body : the arms are pressed to the sides, the elbows and fingers bent ; the thighs approach the abdomen and the calves approach the thighs ; the trunk bends forward ; the shoulders are raised and the head is lowered ; the eyelids close, the teeth are clenched and the lips pursed. The individual is then in a position which reduces the surface of his body to a minimum—a defensive reaction, which is common to all animals from the amœba upwards when faced with danger. The contraction is followed by partial relaxation, and these repeated contractions and relaxations produce the tremor. Like the incapacity to move or to speak the tremor may be perpetuated by auto-suggestion.

Sudden terror occasionally gives rise to hysterical convulsions, particularly if some special incident at the moment or in the individual's past history has suggested convulsions to him.

It is much more common for an interval to elapse between the incidents which gave rise to the emotion and the onset of symptoms. It is thus very rare for hysterical symptoms to develop actually in the trenches. They are comparatively common in casualty clearing stations, and still more so at base hospitals. Not infrequently the hysterical symptoms only appear after the patient has arrived in England. In the dazed condition which results from prolonged mental strain a man is abnormally suggestible; his critical faculties are lost and his initiative is diminished. As he gradually comes to himself, he tends to exaggerate and perpetuate the difficulties he experiences in the performance of the various functions of his body. The absence of movement due to absence of initiative leads to the suggestion of paralysis, the silence due to absence of any stimulus to speak in his confused mental condition suggests mutism, and the inattention, which prevents him hearing what is said to him, suggests deafness. The following case is a typical example of hysterical symptoms developing as a result of auto-suggestion in the process of recovery from the stupor caused by prolonged fear.

Hysterical paraplegia and mutism following stupor caused by prolonged fear.—For three months Pte. C., aged 21, a tall, well-built guardsman, had been getting more and more stupid and useless and had shown great terror whilst in the trenches. On December 30th, 1916, when his battalion had just retired from the front line, he was sent to look for some lost kit and did not return ; it is quite certain that he was not blown up, but he appears to have become dazed, and was brought into a hospital by two men, who had found him in a coffee-bar. He was in a condition of deep stupor, and made no attempt to answer questions or to obey commands, such as to put out his tongue, but he suddenly clutched at the clothes of any one who talked to him. All his reflexes were normal. When admitted to Netley on the 10th January, 1917, he was still in a state of stupor and passed his urine and fæces into the bed, but he took his food well. There were no signs of organic disease. The next day he wrote answers to some questions after a very long latent period. His memory of recent events was much confused, and he could not remember what his occupation was before the war. After vigorous persuasion he was made to walk, but he staggered and tended to step backwards. He was also persuaded to say a few words, such as his name, regiment, and various numbers, but only after a prolonged period during which he moved his right arm in circles. With further persuasion the dirty habits were overcome, and his walking and talking gradually improved. At the end of a month he could walk almost normally, though he had a tendency to advance in curved lines and constantly moved his right hand. He could write quite normally, but he could only speak extremely slowly in a very low voice after a prolonged effort, during which he moved his right arm, and when this was held he moved his right leg, but if the arm was held so firmly that he could not move it at all, he was quite unable to speak. He was hypnotised on February 10th, but in spite of suggestions of recovery he became completely dumb. After a few days speech returned as a result of simple persuasion. In April as the result of the excitement involved in breaking off his engagement, his speech suddenly became exceedingly rapid instead of slow, but he still spoke after a long latent period and only when the right arm was allowed to move. As the condition had become completely stationary in spite of all

attempts at re-education, he was told on June 12th that a small operation would be performed on his head, which would have the effect of separating his speech from his arm movements and curing him completely. He was given ether rapidly with the result that he became extremely excited. When he was sufficiently quiet, an incision was made on the left side of his scalp and three sutures were inserted, vigorous suggestion being applied the whole time. On completely regaining consciousness, he was talking normally without moving his right arm. He returned to duty some weeks later, no relapse having occurred.

Diagnosis from Malingering.—The diagnosis of hysteria from malingering is exceedingly difficult, as the symptoms are identical. In hysteria the paralysis or other symptom is produced by auto-suggestion and corresponds with the patient's own conception of paralysis ; it is consequently indistinguishable from the paralysis which the malingerer voluntarily assumes. Though malingering may be suspected in other cases, it can only be diagnosed with certainty under two conditions. Occasionally an unskilful malingerer may be detected *flagrante delicto*. A momentary movement of a limb when the patient is taken by surprise is compatible with hysterical paralysis, but the appropriate treatment for a paraplegic man, who is discovered walking in the ward when he thinks he is alone and unseen, is to send him to the military authorities for punishment. Very rarely a malingerer confesses that he is shamming, but a confession should only be accepted if it is not forced from a man and if it fits in with the facts. This happened with an unwilling conscript who was sent into hospital for supposed epilepsy. Such cases should be sent back to duty at once, but without punishment. Pure malingering is very rare in the British and French armies. Conscious exaggeration of symptoms and conscious prolongation of incapacity, which is primarily involuntary, are comparatively common. Malingering may in this way be associated with both organic disease and hysteria. It should also be remembered, as Gilbert Ballet has pointed out, that malingering may end in hysteria ; a man who pretends to be paralysed for a sufficiently long period may end by genuinely believing he is paralysed, just as the German people have repeated the official lies as to the cause of the war so

frequently that many now doubtless believe in the truth of what they originally knew was untrue.

Treatment.—Hysterical symptoms in soldiers have proved very amenable to treatment. Some cases sooner or later recover spontaneously, but others show no tendency to improve if left to themselves, and I have seen several cases of hysterical paraplegia, dumbness, blindness and deafness, which had persisted for many months, sometimes for over a year and in one case for over two years, which would almost certainly have remained permanent if vigorous treatment had not at last been instituted. As it is impossible to tell when the patient is first seen whether spontaneous recovery will take place, active treatment should be given immediately. Thus 80 per cent. of the men treated by Captain W. Johnson at a casualty clearing station are sent back to the firing line within four weeks of the onset of symptoms. Hysterical symptoms should rarely persist for more than twenty-four or forty-eight hours after admission to a hospital in England. The longer hysterical symptoms are allowed to remain, the more difficult they are to cure. Although immediate improvement generally results from correct treatment in cases of very long standing, complete recovery is less easy to obtain: a man who recovers from hysterical paraplegia within two or three months of the onset often walks normally within an hour of the commencement of treatment, but his gait may remain abnormal and require careful re-education for many weeks if the paraplegia has been present for a longer period. Similarly early treatment of mutism is followed by rapid and complete recovery, but if left too late the patient whispers or stammers when he regains his power of speech and often needs prolonged re-education before he can talk normally. Experience has shown, however, that continuous treatment by suggestion and persuasion rarely fails to cure at a single sitting, however long the symptoms have been present, if the physician has sufficient patience. Thus a case of hysterical teeth-chattering yielded after Captain Symns had persisted for six hours, although little improvement had occurred after five hours, and we have had many cases which recovered completely at the end of an hour or an hour and a half, although if the treatment had been discontinued a quarter of an hour earlier, the partial recovery would have required many weeks of re-education before becoming complete.

It is essential for success in treatment that the medical officer should feel convinced that the patient's symptoms are not organic or are at most only in part organic. It is sometimes impossible to distinguish with certainty between hysteria and malingering, and the patient should then be given the benefit of the doubt, for the distinction is of no great importance, if, when malingering seems possible, the statement is made in the man's hearing that "nervous" cases are cured by the treatment to be adopted, but that "skrimshankers" are not. If malingering is not merely possible but probable, the same statement should be made, but a painful faradic stimulation of the affected part should be the form the treatment by suggestion should take.

Whatever treatment is employed, the encouragement produced by the presence of cured patients in the same ward and their exhibition to those about to be treated is most helpful. I have found that deaf-mutism rarely lasts for more than twenty-four hours after a patient's admission to my section, even if he has been in this condition for many months and many forms of treatment have been tried in vain, as there is always a cured deaf-mute present, who at once tells the new patient by writing or by the deaf and dumb language how he was quickly cured after being unable to hear or speak for five or six or even twelve months, so that his mind is prepared for the treatment which he is told he will have on the following day. An "atmosphere of cure" in which the medical officer, the sisters, and the recovered patients in the wards all play their part, is of the greatest value for newcomers, however long they may have been ill before admission.

The patient is also made to understand that there is nothing unusual about his case. The extreme interest and sympathy with which he is surrounded account for the frequent persistence of hysterical symptoms for many months spent in some luxurious auxiliary or convalescent hospital. He should be made to realise that many others have had similar symptoms before and have rapidly recovered, and that his early recovery is regarded as a matter of course. The nursing staff should be instructed to speak in the same way, and to tell such patients that they will be perfectly well in a very short time like all their predecessors in the ward.

Hysterical symptoms, being invariably due to suggestion, can

invariably be cured by suggestion or simple persuasion, and to diagnose a symptom as hysterical can only be justified if the next step is to cause it to disappear. Although the same kind of treatment can be used for all hysterical symptoms, I shall describe in later chapters the methods which our experience has shown are most suitable for paralysis, disorders of speech and hearing, convulsions and blindness.

Simple persuasion followed, when necessary, by re-education is all that is required to cure most hysterical symptoms. We have had numerous patients with hysterical paraplegia or mutism, which had often been present for several weeks, who were persuaded to walk or talk in the course of a few minutes after our examination on their entry into hospital had convinced us that there was no organic lesion. A few had previously received no treatment beyond masterly inactivity, as it was assumed that they would in time get up and walk or begin to speak spontaneously. The majority had received treatment of various kinds, especially massage and electricity, but massage is only of use in neglected cases of hysterical paralysis in which muscular atrophy has resulted from prolonged disuse, and then only as an adjuvant to persuasion and re-education. Electricity is quite useless unless employed solely as a means of suggestion; strong faradism applied with the wire brush over paralysed muscles can be used to convince a sceptical patient that the muscles are capable of strong contraction, and the pain produced acts as a powerful means of persuasion for the fortunately rare type of man, who appears to prefer to remain paralysed to recovering. On the other hand, massage and electricity employed regularly by sympathetic nurses only help to confirm the patient in the belief that he is suffering from serious paralysis. Instead of rubbing paralysis away, massage has too often the effect of rubbing the idea of paralysis in. In our experience electricity is particularly harmful when any tremor is present.

The notion that hysteria is nothing more than a form of malingering has prompted some medical officers to treat hysterical symptoms by disciplinary measures, the patient being isolated and allowed no cigarettes and only a limited diet. Such treatment is occasionally successful, but it is the suggestion that it will cure rather than the punishment itself which may produce a

favourable result. I have, however, seen many cases in which no improvement resulted, but in which simple persuasion or suggestion after the patient had been transferred to my section resulted in immediate recovery. I have indeed seen cases, in which the resentment caused by what the patient regarded as cruel and unfair treatment for what was to him a very real and distressing incapacity has actually aggravated his symptoms and made subsequent treatment difficult by causing him to view anything new with suspicion and fear. Thus a man who suddenly developed complete hysterical paralysis of his left arm was treated by isolation and a strict milk diet. He wept copiously and became greatly excited, as he thought he must be regarded as insane to be treated in this way. Explanations were useless and the treatment had to be discontinued. Five months later the arm was still totally paralysed, although he had received daily electrical treatment and massage. He was then transferred to my section: persuasion and re-education at once produced some improvement, which was followed by slow but steady progress, so that at the end of two months recovery was complete and he returned to duty.

As soon as the diagnosis of hysteria has been made as a result of a thorough examination, the patient is told that real as his incapacity has been, there is no doubt at all that it has now disappeared, and that nothing more than a properly directed effort is required to produce normal movements. If he has already had treatment of some kind, it can be said that it has really resulted in recovery, although up to this moment the patient has not realised it. A paralysed man is told that he may feel weak at first and the effort may cause pain, but that both weakness and pain will rapidly disappear when he has once accustomed himself to using the affected muscles again.

At one time I was so greatly pleased with the results of suggestion under hypnosis that I used this method systematically. I then met with four cases in which hypnotism had been used for considerable periods without any success. Private C. had been paraplegic and dumb as a result of shell-shock for a year, during the greater part of which he was treated by hypnotism; he was made to walk and talk by simple persuasion the day he was admitted, but much re-education was

required to correct his stiff gait and stammer, which not unnaturally developed after such prolonged disuse. Private S. had been similarly treated for four of the eight months during which he had been suffering from hysterical paraplegia, but without improvement. I then tried hypnotism myself, but as all suggestions of recovery proved unavailing, I substituted treatment by persuasion and re-education, with the result that he very slowly learnt to walk. I am sure that if persuasion had been used at first, recovery would have followed in a few days. Private A. developed a curious hysterical dancing gait in Salonica and was treated by hypnosis for three weeks in Malta on his way home, but without the slightest improvement. By means of vigorous persuasion he was taught to walk normally the day he reached Netley. The fourth case, Private M., has already been described (p. 13).

I have thus come to the conclusion that valuable as hypnotism is for many war neuroses, especially those which can be classified as forms of pychasthenia, it is generally not the most satisfactory means of treating hysterical paralysis, contractures, gaits, or speech defects. In very resistant cases, in which treatment has previously proved unavailing owing to the hysterical symptoms being associated with fear of permanent ill-health, paralysis, or insanity, hypnosis should be used to remove this idea, and no harm is done in trying to cure the hysterical symptoms at the same time, as in spite of occasional failures, it is often possible to make a paralysed man move the affected limbs whilst he is asleep : if this is done, it is a good plan to let him wake while he is still performing the movement. On three occasions I have allowed paraplegic patients to wake to find themselves walking quite normally. One man whose fingers were so tightly clenched that it was impossible to force them open, the attempt producing considerable pain, was hypnotised and told to open his hand : he did this, and the moment he woke he looked with astonishment at the palm of his hand, which he had not seen since the contracture developed after he had been blown up by a shell four months before.

The objection is sometimes raised that hypnotism treats symptoms without dealing with the underlying abnormal condition of the nervous system. This may be true in non-traumatic

cases of hysteria seen in civil life, but it does not apply to the hysterical manifestations which occur in soldiers. The symptoms have often followed some quite exceptional circumstance, such as a horrible or terrifying incident or the explosion of a big shell, in spite of the fact that the individual's nervous system was either normal or more frequently somewhat exhausted as a result of the strain and stress of war; in any case it was in a very different condition to the quite abnormal nervous system of the young woman, who in civil life is particularly liable to hysterical symptoms, and for whom other methods than hypnotism are almost always successful and are certainly preferable.

With the disappearance of his hysterical symptoms a soldier may often be regarded as cured and fit again for active service. More frequently, however, other symptoms are still present, but the disappearance of the most obvious and distressing symptom, such as blindness, fits, insomnia, and nightmares, removes his chief source of worry and encourages him so greatly that the other symptoms indirectly benefit to a remarkable degree. It is easy to understand how the return of vision to a man, who is suffering from both hysterical amblyopia and neurasthenia, will do his neurasthenic symptoms more good than months of rest, encouragement, or other treatment.

(iv) Hyperadrenalism and Hyperthyroidism

The emotion of fear is related to the instinct of flight and the emotion of anger to the instinct of combat. McDougall's psychological studies on this subject have been amplified by the physiological investigations of Cannon and of Crile with their pupils. The physical manifestations of fear and anger are identical with those of such forms of violent exertion as flight and combat, which were the natural sequels of these emotions in primitive man.

Strenuous action is normally rendered possible by the sympathetic nervous system, which is stimulated to great activity by the emotions of fear and anger under conditions which imperil the safety of the individual. Impulses reaching the suprarenal glands by their sympathetic nervous supply stimulate them to secrete adrenalin. It has been shown experimentally that both the

injection of adrenalin and the increased secretory activity of the suprarenal glands, which occurs in times of stress, give rise to all the effects produced by stimulation of the sympathetic nerves. Suprarenal activity thus powerfully reinforces the direct action of the sympathetic nervous system, which enables the individual to respond to the emotions of fear and anger by strenuous action. There are no constrictor fibres to the cerebral and pulmonary arteries, and the sympathetic fibres to the coronary vessels produce dilatation instead of constriction, so that the vaso-constriction produced by adrenalin is confined to the splanchnic system; the blood-pressure is thus raised and the blood is driven to the heart, lungs, brain and muscles from the abdominal viscera, the action of which is simultaneously suspended by the inhibitory action of the sympathetic nerves on the secretion of gastric and pancreatic juice and bile, and on the movements of the stomach and intestines. At the same time the heart is stimulated to beat more rapidly and vigorously. The skeletal muscles not only receive an increased supply of blood, but also of the sugar required for their activity, as adrenalin augments the stimulating action of the sympathetic on the production of sugar from the glycogen in the liver. The products of muscular activity, the accumulation of which leads to exhaustion, are neutralised or destroyed by adrenalin independently of any sympathetic action. The central nervous system, the heart, and the muscles of the limbs and trunk are thus prepared for great activity at the expense of the digestive organs, which do not contribute to the efficiency of the individual during strenuous exertion. The evaporation of sweat, the secretion of which is increased, prevents the rise of temperature, which would otherwise occur with excessive muscular activity, and the deep respiration and the relaxation of the bronchioles allow more oxygen to enter the lungs and excess of carbon dioxide to escape.

Cannon has shown that adrenalin increases the coagulability of the blood. Pain as well as anger and fear stimulates the secretion of adrenalin, and the pain caused by a wound thus helps to arrest the hæmorrhage which would otherwise weaken the individual and prevent his escape.

The partial asphyxia, which is the cause of the painful gasping caused by over-exertion, stimulates the secretion of adrenalin; the increased supply of sugar and of blood to the muscles, the

diminution in muscular fatigue and the relaxation of the bronchioles which result are the cause of the "second wind," which makes the individual capable of renewed activity, when he is about to fall a victim to his enemy, owing to being unable to continue in battle or flight after the initial secretion produced by anger or fear is beginning to wane.

Adrenalin acts very rapidly, but for a short period, as it is quickly oxydised. It is consequently of special importance when an emotion calls for sudden activity. When more prolonged activity is required, the internal secretion of the thyroid gland probably helps to maintain the activity initiated by adrenalin, as it acts after a longer latent period and its action is more prolonged. Unfortunately experimental evidence for this is lacking, as there are no biological tests for thyroid secretion, similar to those which prove the presence of excess of adrenalin in the blood.

The physiological effects of the fury and excitement of battle enable the born soldier to perform feats of strength and endurance which may be in striking contrast to his comparatively feeble physique. But the changes which accompany fear and anger are entirely useless if the emotions are not followed by the associated instinctive activity. So fixed, however, is the primitive association of anger with fight and fear with flight, that when the natural sequels of these emotions are restrained, they continue to give rise to suprarenal and probably thyroid activity.

Thus the ceaseless fear felt by the constitutionally timid when exposed to the horrors of war results in constant over-secretion of the suprarenal and thyroid glands, the physiological results of which are not followed by the muscular activity of flight for which they are the preparation. The unexpended energy may be so extreme that the soldier is incapacitated by it. On reaching the safety of a base hospital, the hyperactivity of the suprarenal and thyroid glands and the signs and symptoms to which they give rise often disappear. But they may be perpetuated by war-dreams, and in severe cases the mind is absorbed by day as well as by night by pictures of the horrors which the individual has witnessed; every sound reminds him of shells and every movement suggests the approach of danger. The activity of the suprarenal and thyroid glands is consequently maintained, and

the patient presents a picture suggestive of Graves' disease, although hyperactivity of the suprarenal glands is probably of more importance though less easily recognised than that of the thyroid. The pulse is rapid, especially on the slightest exertion, and the heart may be slightly enlarged, but there are generally no murmurs. The blood-pressure of the average soldier in the front-line trenches is slightly subnormal, but in these cases it is always raised whilst the patient is at the casualty clearing station, and it is often 150 or 160 mm., or in severe cases even as high as 180 mm. of mercury on arrival in England. Menard happened to be measuring the blood-pressure of a man a few seconds before a shell exploded in the immediate neighbourhood. It at once rose from 125 mm. of mercury to 130, and ten minutes later was 150 ; an hour later it had again fallen to 125 mm.

I have observed a very characteristic cutaneous reaction in these cases, but in no other condition. On moving the finger over the skin of the chest so lightly that in normal individuals no local reaction would occur, a pilomotor reflex is almost instantaneously produced ; goose skin is obvious, and occasionally the hair can actually be seen to stand on end over the area touched by the finger and for some distance on each side. After about five seconds the pilomotor reflex fades away and is at once replaced by a vasodilator reflex ; the blush, which may have a white border on each side, often lasts for several minutes. As improvement occurs the vasomotor reflex disappears, but the pilomotor reflex generally persists for a few weeks longer. Captain J. L. M. Symns and I never found this sign in its fully developed form in 100 consecutive cases of other war neuroses, 21 cases of other forms of disordered action of the heart, and 47 men rejected for military service on account of various diseases ; a very slight reflex was present in 6 of the first class and 2 of the second, probably owing to the association of slight hormonic over-action with the other neurosis, and in 6 of the third class, 5 of whom had an enlarged thyroid and the sixth had organic mitral disease. Captain W. H. Nutt failed to find it in four patients with parenchymatous goitre and one slight case of Graves' disease in a woman. The circulatory symptoms may be so prominent that the case is often diagnosed as " disordered action of the heart." The condition may then be described as

Fig. II.

1 Pte. E., Severe hyperadrenalism and hyperthyroidism with exophthalmos, resulting from prolonged terror.

2 L.-Corpl. W., with hair continuously on end after prolonged terror: (*a*) before going to France; (*b*) after being sent home.

3 Pte. L., Hysterical contracture of left foot following a kick.

4 Pte. S., Hysterical ataxic gait developing 3 months after recovery from a flesh wound of right leg.

5 Sergt. M., Hysterical "main d'accoucheur" of 8 months' duration, following wound of forearm; cured day after admission.

6 Pte. W., Hysterical contracture of hand following wound of palm. The hand was cold, blue and œdematous, and the thenar muscles slightly atrophied on admission 11 months after onset. Cured in a week by suggestion (*vide* p. 90).

7 Sergt. B., Hysterical "senile gait," following burial: (*a*) on admission, 20 months after onset; (*b*) after 6 weeks' treatment by suggestion and re-education.

8 Pte. I., General paralysis with left ophthalmoplegia following gunshot wound of brain (*vide* p. 68): (*a*) shows external strabismus due to paralysis of all muscles supplied by the third nerve, and dilated pupil; (*b*) shows ptosis.

9 Pte. A., Hysterical gait, following appendicitis at the front.

10 Pte. R., Malingering epilepsy; (*a*) opisthotonos during attack; (*b*) abruptly terminated on command (*vide* p. 97).

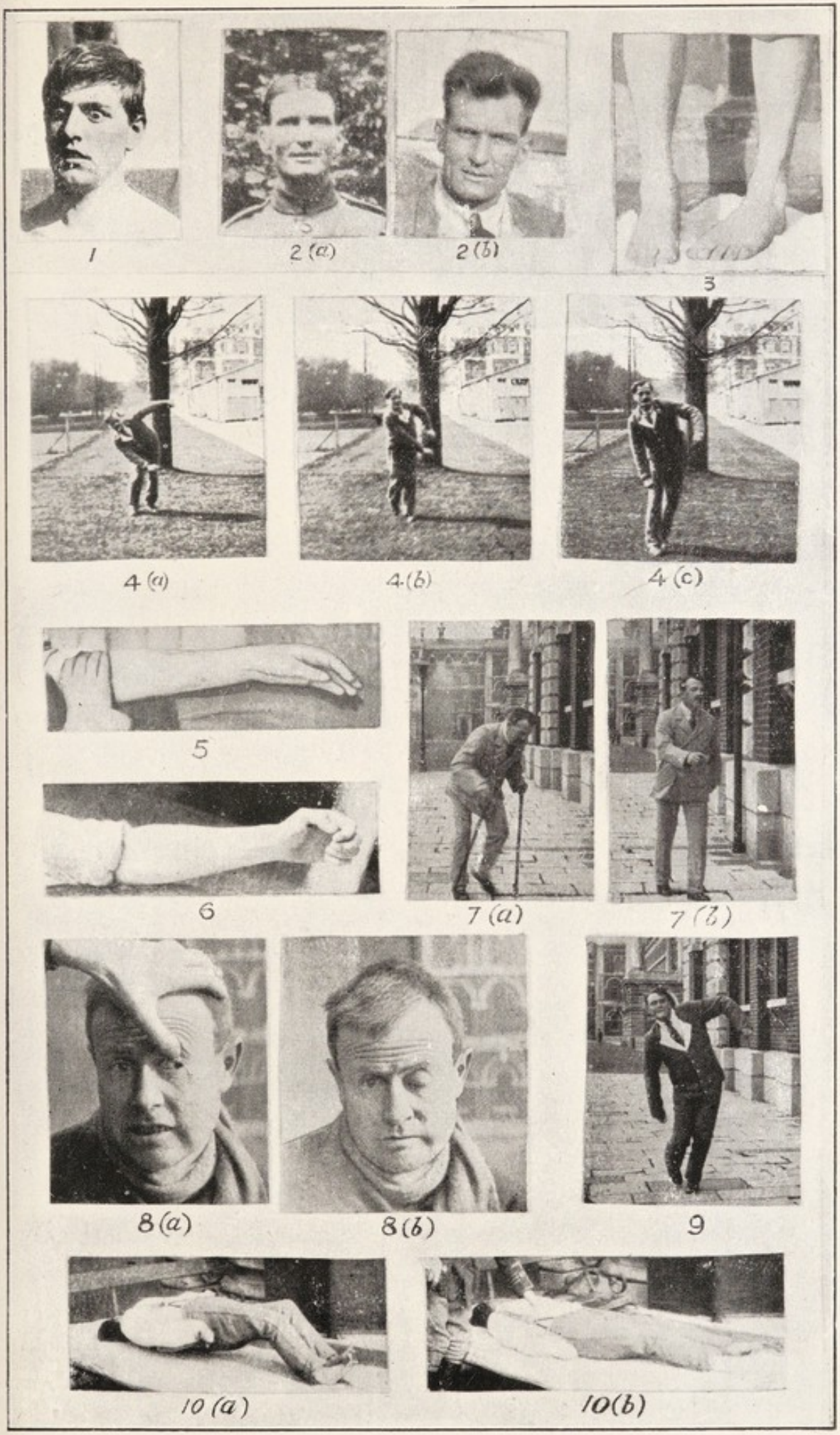


FIG. II. PHOTOGRAPHS ILLUSTRATING WAR-NEUROSES.
(1, 4, 7, 8, 9 and 10 reproduced by permission from Cinematograph Films, obtained with a grant from the Medical Research Committee).

the "hormonic type of soldier's heart." Excessive sweating often occurs, sometimes in paroxysms, especially over the palms of the hands and soles of the feet, and the patient often loses weight. The hands and occasionally the eyelids are tremulous, and the patient is highly nervous and excitable. The eyes are often slightly prominent and von Graefe's sign may be obtained (Fig. II, 1). The thyroid gland, though over-active, is generally not obviously enlarged, but in some cases moderate enlargement is present. McNee and Dunn have found that the average weight of the thyroid gland in 65 apparently healthy men killed in action was 26·7 grms.—notably above the normal for civilian men of corresponding age—although there were no external signs of enlargement. An officer told me that he felt a curious tingling sensation over a broad band of skin round the trunk every time he heard the whistle of an approaching shell; this sounds like a pilomotor reaction. I have seen several men suffering from the effects of severe emotional strain, whose hair persistently stood on end and could not be kept down by means of grease. In some cases I have had the opportunity of comparing their appearance with what it was formerly, and the change from the sleek appearance when in civil life was most remarkable (Fig. II, 2). One man, who kept his hair closely cropped, said his hair reminded him of the bristles of a hedgehog. The persistent action of the pilomotor nerves appears to last longer than any other symptoms, as it has still been present several months after a man's return from France, at a time when he had become otherwise quite fit. In all cases it is associated with an exaggeration of the general cutaneous pilomotor reflex, though this is not often well marked and is unaccompanied by any vasomotor reflex. In some cases the hair on the body as well as on the head has been persistently erect. It is difficult to give a physiological explanation for the case of an officer whose hair turned white after he had been buried in a prone position for twenty-two hours, and a seventeen-year old boy who became gray after a week's heavy bombardment, although similar events have often been described in civil life.

Though hyperthyroidism is the most obvious condition present, it is not the most important, as in all probability it is accompanied by hyperadrenalism, which accounts for the high blood-pressure,

and perhaps for the exaggerated pilomotor-vasodilator cutaneous reflex.

Considering that adrenalin gives rise to an increased production of sugar, glycosuria has been a less common result of the stress of active service than might have been expected. It seems to have been observed much more often among German than British soldiers; among 250 diabetic soldiers treated by Lenné at Neuenahr, only 5 were predisposed by their family history and only 27 had had glycosuria before. In 95 the onset had been acute with sudden great thirst, and in many the condition seemed clearly to be the direct result of exhaustion and mental strain.

Treatment.—The patient must be isolated at first from the other patients in the ward by screens, and should only see such visitors as he believes will allay rather than increase his nervous irritability. He should be protected from any chance of being reminded of what he has passed through by thoughtless conversations or illustrated papers. It is important to bear this in mind even during convalescence. A sixteen-year-old boy who had apparently recovered from this condition had a severe relapse after seeing the Somme pictures, and another man who was sent out to dig too early in convalescence broke down and wept, as digging reminded him too forcibly of the trenches and the horrors he had recently witnessed.

Mental activity and restlessness are lessened by small doses of opium, this being the only war neurosis in which this drug can be safely employed, and also by suggestion, the good effects of which I first saw in cases treated by Captain J. B. Tombleson in Malta. I have since had similar experience in several cases myself, especially when nightmares or some special sources of worry were present. The thyroid and adrenal secretion are kept in check by belladonna. The blood-pressure rapidly falls to normal, and all the symptoms gradually disappear. X-ray applications to the thyroid gland have been used with success in some cases, but it is impossible to regulate the destructive action of the rays with sufficient accuracy for perfect safety, and the other ductless glands are not dealt with. Moreover the excitement and exertion associated with obtaining the treatment often more than outweigh any possible good effects, which it might otherwise

produce, as rest in bed is essential in the early stages except in the mildest cases.

(v) Exaggerated Defensive Reflexes, Hyperaceusis, and Photophobia

The "flinch-reflex," which is caused by the sight of danger approaching, consists in the assumption of a crouching attitude; the arm is raised in front of the face and the eyes blink. It has the object of hiding the individual and defending him from attack. The "jump-reflex" consists of a sudden movement of the limbs and trunk, and is associated with blinking of the eyes and dilatation of the pupils; it is caused by sudden sounds, and is really the preparation for the immediate activity, which would be displayed if the individual obeyed his instinctive desire to save himself by flight. The special conditions at the front lead to ducking of the head being added to the ordinary jump reflex of civil life. Our clinical observations and Sherrington's experiments on animals show that these responses are true defensive reflexes and have their centres in the mid-brain.

The common exaggeration of the jump reflex in soldiers suffering from certain war neuroses is not, therefore, correctly described as hyperaceusis, for the sense of hearing may be no sharper than normal, and actual hearing need not occur at all, as the reflex occurs when they are asleep and hypnotised as well as in some cases of hysterical deafness. It is simply a part of the general exaggeration of the defensive reflexes, which is a characteristic feature of those war neuroses, which are due rather to emotions, such as prolonged fear, than to the actual concussion of pure shell-shock. It is specially common in men who are constitutionally timid or who have become timid owing to their resisting power having been worn out by prolonged mental strain, their special senses and the reflexes associated with them becoming tuned up to such an extent that sights and sounds, which men with stronger nerves learn to disregard after they have been in the trenches for some time, give rise to more and more exaggerated motor responses.

The patient is abnormally sensitive to sound and hates the slightest noise; he is unable to sleep in a town, and sudden noises

often frighten him. The condition of most patients suffering from war neuroses is much aggravated by a thunderstorm or the sound of distant firing, as during an air-raid. Many are reduced to tears, and few are able to sleep the following night. Bright light is also disagreeable, but vision is less sensitive than hearing. In most cases these symptoms are simply due to increased irritability of the nervous system, but in one severe case true hyperaceusis was present, and Captain E. A. Peters and I estimated that the patient heard sixteen times more acutely than the average normal individual. It was possible to carry on a conversation with him by whispering in one corner of the ward when he was lying in the opposite corner, although men with normal hearing who were standing halfway between in the centre of the room could not hear a word of what was whispered. The hyperaceusis and exaggerated defence reflexes were quite uninfluenced by the administration of 100 grs. of bromide a day, and were only slightly reduced by plugging the ears with plasticine.

Most men rapidly improve when removed from the sights and sounds which caused their abnormal state to develop, and the defensive reflexes become less and less exaggerated. If, however, the emotion of terror is kept alive by vivid dreams, and still more if the individual continues to picture the horrors he has witnessed to himself even when he is awake and every sound reminds him of the bursting of shells, the symptoms persist.

The flinch-reflex now occurs whenever anybody, even the patient's friends and relations, comes near, and the jump-reflex, which may at first be produced by nothing less than the explosion of a shell in the near neighbourhood, now follows other noises, until in time quite harmless and comparatively faint sounds give rise to it.

In severe cases the patient appears to be in extreme terror; he jumps violently and trembles from head to foot at the slightest sound; he raises his arm as if to protect his face from a blow and hides his head under the bedclothes when anybody approaches him. After all trace of terror has disappeared as a result of suggestion under hypnosis, and the patient no longer thinks or dreams of the horrors of war, the appearance of terror may continue unabated, the intense emotions to which the patient has been subjected having led to such an increase in the excitability

of the central nervous system that the exaggerated jump and flinch reflexes persist after the cause of their exaggeration has disappeared.

Treatment.—Complete physical and mental rest are essential. No visitors should be allowed, and frequent changes of nurses are most undesirable. Severe cases should be kept in bed and isolated until considerable improvement has occurred. Absolute quiet should be ensured, and recovery is most rapid if the patient is kept in the open air, but protected from bright sunshine. The patient should not read at first, and later all exciting literature should be avoided. He should not talk about his war experiences, but should try to forget the scenes he has passed through. Nightmares and day-dreams should be treated by hypnosis. Bromides have very little effect, except in mild cases, but medinal and aspirin often help to produce sleep. When true hyperaceusis is present the ears should be plugged with wool, and in severe cases, in which this has little or no effect, large woollen pads should be tied over the ears at night or during a thunderstorm.

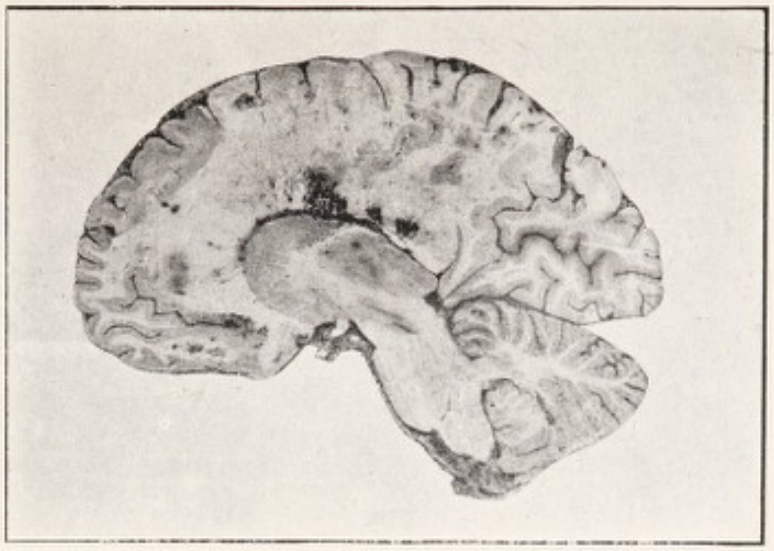
CHAPTER IV

SHELL-SHOCK

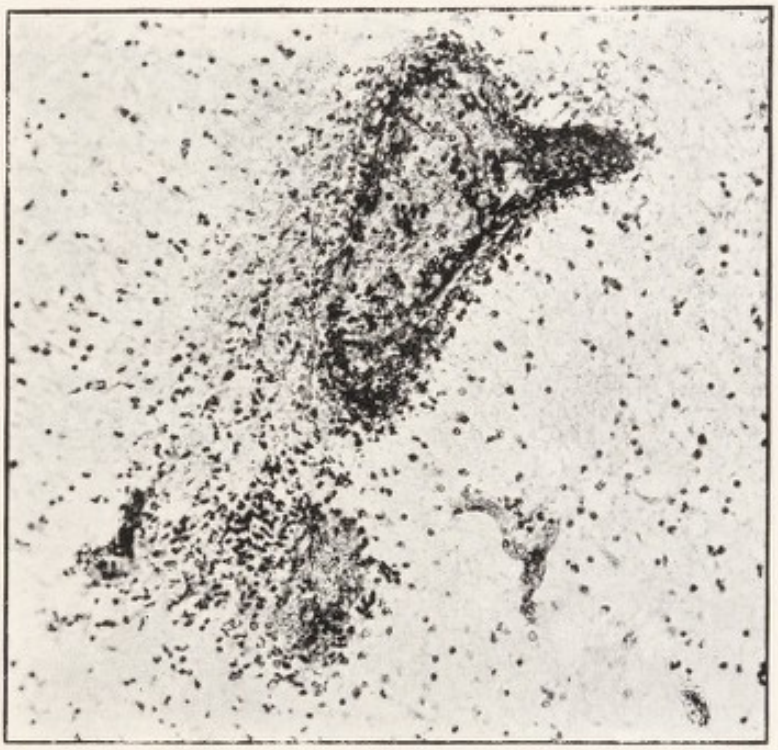
The term "shell-shock" should be reserved for the condition which follows exposure to the forces generated by the explosion of powerful shells in the absence of any visible injury to the head or spine. In all cases there is an organic basis, which consists of the more or less evanescent changes in the central nervous system resulting from the concussion caused by aerial compression, to which is often added concussion of the head or spine caused by the sandbags of a falling parapet or by the patient being blown into the air and falling heavily on to his head or back. On this organic basis hysterical or psychasthenic symptoms are often superposed.

The aerial compression generated at the moment of detonation of a high-explosive shell may amount to ten tons to the square yard, and is followed by an equally great decompression. This may be transmitted through the cerebro-spinal fluid to all the neurones of the central nervous system, the concussion of which results in temporary loss of function, due probably to slight and temporary, but none the less definite, changes in the central nervous system. The sudden violent dispersion of the cerebro-spinal fluid may rupture the delicate lymph-channels and injure the adjacent tissues. Post-mortem examinations on men who have died without regaining consciousness after being blown up by high-explosive shells in the absence of any external injury, have shown multiple punctate hæmorrhages in the white matter of the brain, especially the corpus callosum, internal capsule, and cerebral peduncles; and the nerve cells, especially of the cardiac and respiratory nuclei of the medulla, show chromatolysis with eccentric nuclei (Fig. III).

In every battle enormous quantities of carbon-monoxide are produced from cordite. The diffusive power of the atmosphere is so great that the concentration of the gas under ordinary con-



SHELL SHOCK WITHOUT EVIDENCE OF EXTERNAL INJURY—Section through the whole brain 1 inch external to the mesial surface. Punctiform hæmorrhages in the white matter, especially the corona radiata and corpus callosum in which they have coalesced.



MICROPHOTOGRAPH OF SECTION OF CORPUS CALLOSUM—Shows inflammatory charge around a small vein, a branch of which has ruptured. Magnification 200.

SPINAL CONCUSSION WITHOUT EVIDENCE OF EXTERNAL INJURY.—Section of the fifth cervical segment of spinal cord with degeneration of grey matter, posterior column, and antero-lateral column of one side. Weigert-Pal staining.

FIG. III. Microphotographs of brain and spinal cord in shell-shock. (After Lieut.-Col. F. W. Mott.)

ditions is never likely to reach 1 per cent., which is the strength necessary to cause unconsciousness after a very short exposure. But Mott has pointed out that if a man is buried under sandbags in a heavily shelled trench or dug-out, or if for any other reason he is confined in a small space, in which the gas produced by an explosion collects without being able to diffuse rapidly away, he will be rendered unconscious, if he is not already unconscious as a result of concussion. The continued inhalation of the gas will cause him to suffer from the effects of carbon-monoxide poisoning, although he will not have realised during his period of consciousness that he was inhaling a poison, as the gas is odourless. Carbon-monoxide poisoning leads to deficient oxygenation of the blood owing to the gas combining with some of the hæmoglobin, which has a more powerful affinity for it than for oxygen. The pathological changes produced are identical with those found in fatal cases of shell-shock, so that carbon-monoxide poisoning may, perhaps, be responsible for some of the symptoms in a small proportion of cases.

Several French observers have found that if a lumbar puncture is performed in a case of shell-shock within a few hours of the onset of symptoms, the cerebro-spinal fluid is generally under increased pressure and contains albumin, blood, and slight excess of lymphocytes. If the examination is repeated in forty-eight hours, these abnormalities are no longer present. This accounts for the fact that the cerebro-spinal fluid is almost invariably normal when the lumbar puncture is performed at a base hospital.

In such cases it is clear that organic changes have occurred in the central nervous system, which are, however, so slight—consisting probably of minute capillary hæmorrhages and chromatolysis of nerve cells—that they rapidly and completely disappear.

Symptoms.—Two groups of cases may be recognised. In the first the symptoms are due entirely to concussion of the brain and spinal cord; in the second hysterical manifestations are grafted on to this organic basis.

(i) Pure Concussion

(*a*) *Cerebral Concussion.*—The symptoms of uncomplicated shell-shock are identical with those of concussion in civil life.

The immediate effect of a high-explosive shell is to render a man unconscious. In the severest cases the patient's breathing is stertorous, and he may die instantaneously like Nelson's clerk at Trafalgar, who was "killed by the wind of a ball, though his person was untouched," or after an interval of a few hours or days without regaining consciousness. In the more serious of the cases in which recovery ultimately takes place the patient passes into a condition of stupor. The patient is at first entirely unconscious of his surroundings. He appears not to see or hear, he cannot be induced to speak, and pinching his skin produces no response. The reaction of the pupils to light is impaired or lost. He does not ask for food, but chews and swallows whatever is given him. He lies inert, and involuntary micturition and defæcation are frequently present during the first few hours. Complete insensibility is often followed by a dazed condition, in which automatic complex acts may be performed. A man may be found several miles away from his unit, but he will never recall how he covered the distance.

Several cases of this sort have been sent home diagnosed as "acute confusional insanity," and it has only been discovered some days later when the patient's memory has returned that the condition was due to shell-shock. It is, however, always necessary to obtain confirmation of the patient's statement from his unit, as very similar symptoms, which are often labelled as shell-shock, occur as a result of emotional strain without any concussion due to the actual explosion of a shell, and the automatism is also indistinguishable from that of epilepsy. The stupor following emotional strain, however, comes on gradually and is never accompanied by any sign of organic disease or change in the cerebro-spinal fluid, and it is not followed by severe headache.

The duration of stupor varies from a few minutes to several days, but it rarely lasts more than a week. It may suddenly pass away, the patient having no recollection of what occurred between the onset and the moment of recovery. More frequently improvement is gradual. Some patients remain lethargic for a long time and take no interest in what is happening around them, but they may obey simple commands, such as to put out the tongue, but only after a considerable latent period. Others become child-like, and I have seen numerous cases, in which the patient's condition

has so closely resembled dementia præcox that he has been sent home with this diagnosis, which has proved to be incorrect as complete recovery has occurred.

As a rule the patient soon recovers his memory up to the time of the explosion, and may even recollect that he heard the sound of the shell coming, but from this moment his mind is a blank. Less frequently his memory is perfect up to a certain date, such as the day of his arrival at the front, but all subsequent events are forgotten. The patient appears to live over some of the forgotten events, especially those of a terrifying nature, in his dreams, but on waking he remembers nothing of them. In severe cases more or less complete retrograde amnesia may be present. But although the patient does not remember his name, and has no recollection of his past life, faculties which have become automatic as a result of years of practice are not forgotten, the patient being able to read, write, talk, dress, and feed himself.

The return of memory may be hastened by anything which tends to recall a man's previous occupation: McDougall relates the case of a man who showed great aptitude for the task when given a pair of scissors and told to cut the hair of another patient; he said that the occupation seemed familiar, and it eventually turned out that he had been a barber. A man who is fond of music may remember tunes and words of songs, especially if they are begun for him, long before he remembers anything else; this is due to the fact that the singing of a familiar tune can normally be continued without any effort of consciousness after it is once started, knowledge which appeals particularly to the emotions, such as music, being most deeply engraved in the mind.

In the following case the lost memory was brought back by a fortunate accident.

Complete Amnesia cured by reading the "Tatler."—A man in an R.F.A. uniform, but with no papers or identification disc, was found wandering by the military police. He had no recollection of his name or of any past events. In spite of numerous attempts to restore his memory under hypnosis it remained a complete blank. He appeared to be well educated and remembered everything that had occurred since he was brought in by the police. One day, six weeks later, he saw the portrait of "Madame D., the famous Spanish *prima donna*," in the *Tatler*. His memory instantaneously returned, as he recognised Donna

as his name. The next day he wrote the following history of his illness, the main facts of which were subsequently verified.

"I enlisted in the early part of the war and went to France in December, 1914. After being in action for some weeks I had my horses killed with a shell and got a shock, and got a few days' excused duty. In August, 1915, I had fever, dysentery, and shaking. Was sent to hospital for six weeks and got better, except for weakness. Was sent home and got passed as fit in May, 1916. Sent to France in August, 1916. Got sent to the Somme Front, and first sign of loss of memory happened when I got posted to —— Battery. Although I was a full sergeant, and qualified, I found that I had lost complete remembrance of gunnery. I did not even know how to lay a gun, and as I was in charge of a detachment I was placed in a peculiar position. I felt very nervous, and as the lines of infantry were in danger through any mistake I might make I did not know what to do, and I was afraid to tell my Commanding Officer. I was afraid he would think I had gone mad. . . . After thirteen weeks on the Somme I reported sick with shivering and pains in my head. I had four days off duty, and on the 5th day I went back to work with the battery at night. When the work was finished I felt queer again and went to lie down, but do not remember any more until the following night when I woke up in my billet and found I was a prisoner for being absent and drunk. I did not know what to say, and being afraid my reason was leaving me, I pleaded 'guilty' as the lesser of the two evils. I got reduced from sergeant to gunner, and sent to another battery. I reported sick again; got sent home, and got well again in a month. Went to Newcastle on six days' leave. Remember getting to Newcastle on Monday night, but felt very ill. I went to a public house and had some drink, but do not remember leaving, or what I did until I found myself on the following Saturday morning on the Town Moor. I had no kit; my pass and railway warrant were missing, but I had some money in my pocket. My head felt queer, but not painful, but I had no recollection of where I had been since Monday. I reported myself to the Transport Officer at the station, and I was detained there for an escort. I felt very ill, stupid and miserable. I was brought back to Romsey and felt ill. I went to my tent on Saturday and lay down, but remember no more until on Sunday I found myself in Southampton. I took train to London, but stopped at Basingstoke as I felt so ill. The police took me to hospital, but I had no recollection of where I had been or who I was for six weeks, when I saw my name in the *Tatler*.

"I have had these fits in civilian life, but I have always managed to conceal them, as I was always afraid of telling any one, as I might have been sent to an asylum, as my mother died in an asylum, and I have been in dread of it all my life."

Headache is invariably present. Whilst the patient is still dazed as a result of the shell-shock, his head feels heavy and uncomfortable, but severe pain only develops as his mind becomes clearer. It is increased by the smallest mental effort, and is sometimes worst at night, when it may prevent sleep. Lumbar puncture often shows that the pressure is raised, and the removal of cerebrospinal fluid then relieves the headache, but rarely for longer than a few hours. In several cases the fluid has given a positive Wassermann reaction; recovery has then been accelerated by antisyphilitic treatment. It is greatly aggravated by nightmares and the recollection of horrors through which the patient has passed. It varies in character and position, but it is most often in the occipital region and back of the neck. For many months after the severe headache has disappeared, a heavy, full sensation may still be caused by mental concentration or excitement, and sustained attention is impossible because of the sense of weariness it causes. The patient finds it difficult to make up his mind even about trifling affairs; this and the inability to use his brain for any length of time are often a great source of worry to him.

Mental irritability is very common, especially among officers and the better educated men. It is an early symptom, and often continues after the patient is otherwise well. He loses his temper for trivial causes, and may get himself into trouble for insubordination.

A feeling of great fatigue is often present after shell-shock. The patient is unable to exert himself either physically or mentally. In severe cases he likes to lie like a log in perfect silence and a darkened room with his mind completely blank; it is only when improvement begins that he desires cheerful surroundings, but even then thinking requires an unpleasant effort.

One officer was greatly worried on account of seminal emissions, which had occurred three or four times every night since he was blown up by a shell a month before I saw him. They were always accompanied by war dreams, and disappeared permanently after suggestion under hypnosis on a single occasion.

(*b*) *Spinal Concussion.*—Spinal concussion is generally due to an explosion in which the patient is buried under earth or sandbags. The skin and muscles over the spine may be bruised, but often no sign of injury can be found. I have seen several cases in which concussion has resulted from an actual wound, in which the missile passed near the spine without actually injuring it. Thus in one severe case a bullet entered the apex of the right lung, traversed the whole of the right side of the chest, producing a pneumo-hæmothorax, and emerged on the right side of the lumbar spine. Severe paraplegia resulted although the spine was not touched. In a case described on p. 54, cervical hæmatomyelia appears to have resulted from the concussion caused by aerial compression due to the explosion of a shell without any direct blow on the spine.

Roselle and Oberthur examined a number of men suffering from spinal concussion whilst still in the trenches within a few minutes of being injured. They found that the tendon reflexes were exaggerated and the cutaneous reflexes were absent, except the plantar reflex, which was extensor ; extreme hypotonus of all muscles was present.

In slight cases the hypotonus passes off in a few hours, often whilst the patient is still unconscious, the legs becoming slightly spastic with normal or exaggerated knee-jerks, and the plantar reflex is now absent or flexor. In more severe cases the muscular tone is diminished for a longer period and an extreme degree of flaccidity may persist ; the knee- and ankle-jerks are then weak or unobtainable ; in the course of time the tone returns, the jerks become normal, and the paralysis disappears.

When, however, more profound changes have been produced in the cord the flaccidity is replaced by increasing spasticity with increased jerks, ankle clonus, and extensor plantar reflexes. In most cases complete recovery with disappearance of all abnormal physical signs occurs, but in some cases slight spasticity with exaggerated jerks and occasionally extensor reflexes persists, the concussion having resulted in some permanent lesion of the spinal cord.

Partial or complete anæsthesia or analgesia may be present over an area of varying extent. In mild cases only the feet or legs are affected but in the severe cases in which the knee-jerks

are absent diminished sensation may extend as high as the area supplied by the spinal segment, which received the greatest degree of concussion, and there may then be a girdle of increased sensitiveness to pain at the upper limit of the area of diminished sensation. In most cases the spine is tender, and in severe cases the tenderness is definitely localised to the spine corresponding with the segment of the spinal cord which supplies the hyperæsthetic zone. In cases of this sort there may be some spinal rigidity, and pain may occur on bending or twisting. An X-ray examination shows that there is no actual injury to the spine. In one case well-marked striæ atrophicæ were present on the left of the lumbar spine and to a less extent on the right, although no bruising was present; the patient had been lying flat on his back for seven months after being buried, but learnt to walk the day after his admission to Netley.

In many cases fæces and urine are at first passed involuntarily. The initial incontinence does not last more than a few hours. It may then be replaced by retention for twenty-four hours, and this is sometimes followed by difficulty in micturition for two or three days, but the condition of the bladder is generally normal by the time the patient reaches England.

(ii) Hysterical Symptoms grafted on to Organic Basis of Cerebral or Spinal Concussion

In uncomplicated cases of shell-shock recovery often occurs with remarkable rapidity. The majority of slight cases probably never reach a base hospital, and some, indeed, never go sick at all. Severe cases are generally due to hysterical manifestations becoming grafted upon the organic basis of shell-shock, which is caused by aerial and direct concussion and possibly carbon-monoxide poisoning.

In the confused mental condition which follows shell-shock a man is abnormally suggestible, particularly if he is already exhausted owing to the stress and strain of active service. The organic paraplegia, which is the result of transient structural changes in the spinal cord produced by concussion, disappears with the return of the cord to its normal condition; it is then often replaced by hysterical paraplegia. Finding that he is

unable to move his legs when he first regains consciousness, a man becomes convinced that he is paralysed and makes no further efforts; the paralysis thus produced by auto-suggestion can be removed by persuasion or a counter-suggestion. In the same way when one side of the brain has been chiefly affected by the concussion, an initially organic hemiplegia merges into hysterical hemiplegia. I have watched several cases, in which all the physical signs of organic paraplegia or hemiplegia were at first present, but have gradually disappeared in the course of a few days or weeks, although the paralysis has remained, until by suggestion or persuasion it has been cured in a few minutes. Sometimes, however, some organic signs remain, and psychotherapy then can only produce an incomplete cure, a slightly spastic gait or some slowness and lack of accuracy in the first movements of an arm being left as the permanent result of the shell-shock. Less frequently the original changes in the central nervous system are so severe that the symptoms remain organic in origin throughout, and in exceptional cases the injury may be fatal, although no external wound can be discovered. It is thus impossible to give an accurate prognosis so long as well-marked organic signs are present. As, however, complete or almost complete recovery occurs in the vast majority of cases, the prognosis can be regarded as good, and an attempt should be made as soon as the initial stupor has passed away to persuade the patient to walk. It is extremely difficult so long as any organic signs persist to judge to what extent the symptoms are organic in origin, but experience has shown that some physical signs of organic disease, such as an extensor plantar reflex, greatly exaggerated knee-jerks and true ankle-clonus, and unilateral absence of the abdominal reflex, may still be present when the character of the gait and its rapid improvement with persuasion and re-education show that the symptoms are almost entirely hysterical.

Hysterical Paraplegia following Spinal Concussion.—Pte. P., aged 28, was buried on 25th September, 1916, as a result of the explosion of a shell. The falling parapet struck him on the back, and he was only dug out six hours later. His legs were completely paralysed, and he could only micturate with a great effort. He remained lying in bed until he was transferred to Netley on the 18th May, 1917. The muscles of the legs were well developed and were not completely paralysed, but a great effort was required

to contract them, the attempt leading to clonic convulsive movements. The knee-jerks and ankle-jerks were normal, and the plantar reflex was flexor. On the day of admission his attention was distracted by making him count and look at the ceiling at the same time as he clasped his hands and pulled one from the other. His legs were alternately flexed on his abdomen until the movement became quite easy. Less and less assistance was given until he was finally able to move them quite well by himself. He was then made to balance himself and repeat walking movements whilst sitting up with his legs hanging over the edge of the bed. About five minutes after the commencement of treatment he was able to stand. He next began to walk, at first with assistance, and finally at the end of another ten minutes without any assistance at all. For a time he was very unsteady, but at the end of half an hour he walked round the ward. The rapid partial recovery, which was produced by vigorous persuasion, was followed by slow but steady improvement with re-education. As generally happens in cases of spinal concussion of long standing, the gait continued to be somewhat stiff and he was not fit for light duty until September. At first he complained of a considerable amount of pain in the back, but this gradually disappeared as walking improved.

Hysterical Paraplegia with Organic Hemiplegia due to Shell-Shock.—Pte. M., aged 25, enlisted in September, 1914, and served for six months in France and a year in Salonica. He was very fit the whole time, and was never worried by the shell fire. On 22nd November, 1916, he was blown up by a shell, and remained unconscious for four days with signs of complete organic left hemiplegia and incontinence of urine and fæces. He began to answer questions on December 2nd, and complained of severe headache. His knee-jerks were then greatly exaggerated, especially the left, and the plantar reflex on both sides was extensor. The headache soon disappeared, and the hemiplegia gradually improved, but he was still quite unable to walk when he reached Netley on January 24th, 1917. He had no recollection of anything between the fight, in which he was blown up, and the last few days he was in Malta. The right knee-jerk was 6, the left 7 (average normal 4); the left plantar reflex was still extensor, but the right was now flexor, and the left abdominal reflex was absent. The inability to walk was clearly hysterical, and it disappeared the day after admission as a result of persuasion; with further re-education he soon learnt to walk without even a limp. By February 2nd the left plantar reflex had also become flexor, and the left abdominal reflex was as brisk as the right; but

Babinski's second sign (combined flexion of thigh and pelvis) was still very definitely positive and the knee-jerks were as before. The Wassermann reaction was negative. He was discharged to duty in April, the superficial and deep reflexes being normal and equal on the two sides, but Babinski's second sign was still present, though less marked.

Hysterical Paralysis associated with Organic Paralysis due to Hæmatomyelia, the Result of Shell-Shock.—Pte. A. C., aged 24, was in a charge on 11th April, 1917, when he heard a crash and was blown three feet into the air, falling heavily on his face. He did not lose consciousness, and he is quite certain that his head was not doubled under him. He was unable to move for several hours, except that he managed to raise his face out of the mud in order to breathe. On being taken to hospital he remained quite helpless. His elbows were kept acutely flexed, as in lesions of the fifth cervical spinal segment. His right arm and leg were completely paralysed, and only very feeble movements were possible on the left side. He had much aching and tingling pain in his limbs and a spasmodic gripping pain in the calves. He had some retention of urine during the first day, and a catheter was passed on one occasion, but after this his bladder and rectum showed no abnormality. Though listless and suffering from headache his mind was not confused, and his speech was normal. Knee-jerks were very weak, and no definite plantar reflex was obtainable. By the 20th a slight degree of power had returned in the right arm ; both arms were still painful. On the 12th May it was noted that although there was no anæsthesia, sensation to light touch was diminished up to the region of the clavicle. His urine contained some albumen and a few granular casts, although it had shown no abnormality before this date. The headache had disappeared, and the pain in the limbs was less marked. Slight improvement in power occurred as a result of massage, but the muscles remained flabby and began to waste. By the 22nd May the muscular tone had improved, the knee-jerks were now increased, ankle clonus was elicited on the right side, and the plantar reflex was extensor on the right side, but normal on the left ; both abdominal reflexes were absent.

On admission to Netley on the 30th May he could move both arms and legs, but they were very weak, the right side being worse than the left. Slight pain was still present in the hands and arms, but the pain in the legs had disappeared. There was marked wasting of the muscles of the upper limbs, especially of the hands, the atrophy and weakness of the right hand being severe. The right knee-jerk was 5 and the left 4½ (normal 4) ;

true ankle clonus was present on the right side, and also, though less well maintained, on the left. No abdominal reflexes were obtained, and the plantar reflex was definitely extensor on both sides. The skin was much thickened over the palms of the hands and soles of the feet.

It seems clear that a hæmorrhage occurred into the cervical spinal cord at the time of the explosion, probably as a result of aerial concussion rather than of the concussion caused by falling after being blown into the air, as the patient is quite clear that the fall did not hurt him particularly and that he could not put out his arms to save himself when he was in the air.

On June 11th, as the patient still said he could not even sit up in bed and no definite progress had been made with his power of moving his arms and legs, it was thought that some of the incapacity was hysterical in spite of the definitely organic basis. He was therefore treated by very vigorous persuasion, and although he would make no effort at first, at the end of five minutes he was sitting up in a chair, and at the end of a quarter of an hour he was able to stand and take a few steps with comparatively little support. During the next ten days he learnt to stand and walk with an almost normal gait and without assistance. His condition must therefore by now have been largely hysterical, and due to auto-suggestion causing the perpetuation and exaggeration of symptoms, which were originally entirely organic and were still to some extent a result of organic changes in the spinal cord. Since then steady improvement has occurred both in the hands and legs ; his gait is almost normal, and he can use his hands for all ordinary purposes, though there is still some atrophy and weakness of the small muscles. On October 23rd the wrist-jerks were normal ; the right knee-jerk was 7, the left 6, and slight ankle-clonus was obtained on the right side. The right plantar reflex was extensor, the left flexor. The abdominal reflexes had not returned.

Spinal Concussion involving Posterior Columns associated with Hysterical Paraplegia.—Pte. W., aged 32, was buried by a collapsing trench on the 30th July, 1917 ; he was fit in every way before this happened. When admitted to hospital in France he was unable to speak or move his legs, and it was found that he had no knee-jerks. His speech returned in a few days after stimulation with faradism, but he continued to stammer. On admission to Netley

on 28th August he was still completely paraplegic and had a severe stammer; both knee- and ankle-jerks were completely absent, and there was considerable rigidity of the legs. The plantar reflexes were normal. As a result of vigorous suggestion with the aid of faradism he was induced to walk on the day of admission, and with re-education his speech and gait slowly improved. At the beginning of January, 1918, the knee- and ankle-jerks were still absent, and a slight Romberg sign was present, but he walked almost normally. The Wassermann reaction of the blood and cerebro-spinal fluid was negative, and no abnormal cells were found in the latter. It seems probable that the loss of jerks and the inco-ordination were due to the spinal concussion having involved especially the posterior columns, as in the fatal case from which the cord shown in Fig. III, was obtained. The response to treatment by suggestion and persuasion shows that in spite of this the paraplegia was largely hysterical in origin, the paralysis due to the concussion being perpetuated by suggestion. The speech defect was of course entirely hysterical.

Apart from the hysterical manifestations which result from the perpetuation of symptoms produced by transient organic lesions of the central nervous system, others may be produced by auto-suggestion without any primary organic condition. These hysterical manifestations are very varied, and it is at first difficult to explain why one man should become blind, another deaf and dumb, and another hemiplegic under apparently identical conditions. From careful inquiry into the history of numerous cases I have come to the conclusion that the variability of the symptoms can best be explained in the following way. As a result of the various factors which give rise to shell-shock all the functions of the body are in abeyance for a period which varies in different cases: the patient cannot see or hear or feel, he cannot talk, he cannot move, and his mind is a complete blank. In many cases all these lost functions gradually return in the course of a few hours, though some come back more rapidly than others: the patient is then in much the same condition as a man who has been concussed by falling on his head, having a number of symptoms, such as headache, difficulty in concentration, and lack of energy, but there is no paralysis nor loss of any of the special senses. In other cases, as consciousness returns, the patient's mind becomes fixed on some part of his body which is painful, the pain being the

first impression powerful enough to attract his awakening attention; or the temporary inability to see or hear or speak, which generally remains unnoticed when consciousness first returns because of the absence of any desire to see or hear or speak, is suddenly realised owing to a special call being made on one of these functions. The patient's dawning intelligence becomes fixed upon this single missing function, and he suggests to himself that the disability will be permanent. The fact that the other functions are missing remains unnoticed, and after a time they spontaneously return. The persistent localised loss of function is thus due to auto-suggestion leading to the perpetuation of what would otherwise be a very temporary incapacity: it can thus correctly be described as hysterical.

Less frequently the impressions received by the patient between the moment the explosion occurs and the moment when consciousness is lost give the key to the symptoms. The first thought of a man on regaining consciousness, after being deafened by the noise or "struck dumb" with terror when the explosion took place, may result in the suggestion of deafness or dumbness.

The importance of the terror caused by the approach and final explosion of the shell in the few seconds immediately preceding loss of consciousness in the production of the hysterical and mental symptoms, which form part of the picture in many cases of shell-shock, is shown by the fact that they do not develop if the explosion occurs whilst a man is asleep. A man lay down to sleep in the trenches on November 8th, feeling quite well, and came to himself two days later in a hospital train with a pain in his back and feeling dizzy and shaky. A note accompanied him to say that he had been blown up and buried by a high-explosive shell. On reaching England on November 22nd he complained only of slight pain in the back. There was no tremor or other symptom, and on December 16th he was well enough to be discharged to duty.

Treatment.—Rest in bed is required for the symptoms of shell-shock, which are the result of actual concussion. In civil practice it is a common event to see patients suffering from chronic headache after cerebral concussion and chronic backache after spinal concussion, both of which might have been prevented by sufficient rest immediately after the injury. The same is true

after shell-shock and burial, and many men, who pass from one hospital to another for many months receiving all sorts of expensive treatment with little or no benefit, could have been rapidly cured by simple rest immediately after the injury. On the other hand, the rest must not be too prolonged, and it requires considerable experience to judge the right moment when such patients should be allowed to get up. If kept in bed too long, they are very liable to lose their power of standing and walking, as the care taken to keep them at rest may give them the idea that they are severely injured and hysterical astasia-abasia or paraplegia develops. In order to prevent this the patient should be made to get up to go to the lavatory and to have a bath from the first day, except when severe stupor is present, in which case he should get up as soon as it has sufficiently diminished. When the patient no longer complains of pain in the head or back he should be encouraged to take exercise, the amount of which should be steadily increased. Massage often aggravates the pain, and it tends to increase the tendency to introspection.

As a rule no special treatment is required for the stupor and amnesia. The stupor disappears spontaneously, and in many cases complete recovery from the amnesia follows. When partial amnesia persists, it is generally unnecessary to make any attempt to restore the lost memory. In most cases the blank in the memory is for a comparatively short period, and as the events which occurred during this period were probably very unpleasant and perhaps extremely horrible or terrifying, they are best forgotten, and the loss of memory may be regarded as protective. The harm which may result from restoring the memory under such circumstances is shown by the following case, which is only one of several similar cases I have seen.

Tremor and Stammer following Restoration of Memory.—Pte. K., aged 22, was admitted into a casualty clearing station in France on 23rd April, 1917, having been found wandering and unable to give any account of himself: he did not know his name and had no idea what had happened to him. The same day he was hypnotised, and his memory, for all but recent events, was restored. In spite of this he continued to be given hypnotic treatment for over two months, and every detail of the forgotten horrors he had recently witnessed as well as of painful experiences in his past life were daily recalled to his mind. This soon resulted in

the development of a severe stammer and general tremor, although neither symptom was present when he was first admitted to hospital. When he reached England it was found exceedingly difficult to control these symptoms, but with rest, isolation, re-education and encouragement recovery has occurred.

Active treatment is only necessary when there is more or less complete retrograde amnesia, which shows no signs of disappearing after the patient's general condition has improved. Hypnosis is the only treatment which has any effect. Three cases of complete amnesia gradually recovered after I had induced the patients to write their names and other details during hypnotic sleep. Great perseverance is generally required to restore the memory. Sometimes whilst the patient is hypnotised he describes forgotten facts or events, which at first mean nothing to him when he is awake, but in such cases the memory gradually returns in time. In the following case, recorded by Feiling, all attempts to associate the hypnotic with the waking state proved unsuccessful.

So-called Double Personality following Shell-Shock.—A soldier was buried in a trench for fifteen hours in October, 1914. When he came to himself five days later he found that he was in Manchester, but he had lost all recollection of his previous life and had to learn to speak, read, and write again; he did this so quickly that at the end of January he was normal in every way, except that he had forgotten all events up to his re-awakening in Manchester. He did not recognise his parents or any other people or objects with which he had previously been familiar. When hypnotised he could answer questions about his previous life, giving vivid details of his experiences in Flanders. He wrote answers to questions whilst under hypnosis, but when he woke up he did not recognise the handwriting, which was quite different from that which he had since acquired. When hypnotised he believes that it is November, 1914, and that he has just arrived in Manchester from the front; he recognises his father with a degree of affection, which is quite absent from his usual state, as when awake he has to take him on trust.

The associated hysterical symptoms should be treated in the manner already described (p. 29).

CHAPTER V

CONDITIONS PREDISPOSING TO THE DEVELOPMENT OF SPECIAL SYMPTOMS

The stress and strain of active service and the occurrence of actual shell-shock may give rise to a great variety of symptoms, the exact nature of which in many cases depends upon the presence of predisposing conditions. Thus a man with a family or personal history of epilepsy or insanity is particularly likely to develop epilepsy or insanity, and a man infected with syphilis is liable to develop some syphilitic disease of his central nervous system. Previous or simultaneous disease, injuries, or wounds of a limb predispose to hysterical paralysis, spasm and tremor of the limb. Past emotional disturbances or illnesses in rare cases influence the form which the war neurosis takes. In addition to these predisposing factors, which will be discussed in this chapter, ocular and aural defects, already present or produced simultaneously, predispose to the development of ocular or aural symptoms respectively; previous speech defects and laryngitis predispose to mutism, aphonia, and stammering; and excessive smoking predisposes to tremor and soldier's heart.

(i) Family or Personal History of Epilepsy, predisposing to Epilepsy

True epilepsy may develop as a result of the strain of active service, but I have only seen two undoubted cases, in which the individual was not predisposed by having previously suffered from some form of major or minor epilepsy or by having a family history of epilepsy, except when an actual head injury was the exciting cause. Mott expressed almost the same view, but he had met with more exceptions. In my experience almost every exception has proved to be apparent, as further observation has

shown that the fits were really hysterical or were the first manifestation of commencing general paralysis. One of the exceptions was an only child, and his parents both died before he was ten years old, so that the absence of a family history was of comparatively small importance, as it is common to find that one or more brothers or sisters are the only epileptics in a family. The other exception was a case of epilepsy associated with hypothyroidism.

Epilepsy with Hypothyroidism developing whilst a Prisoner in Germany.—Corporal P., aged 24, had no family history and had always enjoyed good health until he was taken prisoner during the retreat from Mons. After being very badly treated and half-starved for a year he began to suffer from epilepsy. He was repatriated on this account in April, 1916, and as the fits did not diminish in number he was invalided from the service in June, 1916. He had numerous attacks of both major and minor epilepsy, in many of which he was seen by his doctor. He became cyanosed, his pupils dilated, and his conjunctival reflex was lost during the major attacks, and he wet his bed when these attacks were at night. No improvement occurred in spite of being given a drachm of bromide three times a day. When admitted into Netley, his appearance suggested that he was suffering from hypothyroidism. The bromide was discontinued and he was given gr. ½ dried thyroid and later gr. 1 dried thyroid three times a day. The number of fits at once dropped from an average of twenty to an average of three a day, the reduction being most striking with the minor attacks. The addition of bromide, which had no effect when given by itself, now stopped the fits completely, and he was discharged on June 1st, having had no fits of any kind for a fortnight.

Many men, who have inherited a predisposition to epilepsy but have never suffered from it, or who have had no fits since early childhood, suffer from epilepsy when exposed to the strain of active service, even if they are never within range of shell-fire, the change from a protected life to one of greater excitement being sufficient to light up the latent disease. Sometimes an acute infection may be the exciting cause of the return of epilepsy in a man who has had no fits for years.

Epilepsy following Malaria.—Pte. W., aged 20, has had fits at long intervals since he was fifteen, but he had had none for a year when he joined the army in July, 1915. He continued to be free from them until he contracted malaria in Salonica in September,

1916. They then became frequent and have since recurred with each recurrence of malaria. Since he reached England in March, 1917, he has had very few fits except during the night following an attack of malaria. When the latter became infrequent as a result of the subcutaneous injection of quinine, the fits also became more rare and were finally almost completely controlled by bromide.

In other cases actual shell-shock has occurred, but unless there is a head wound, this also only gives rise to epilepsy in the predisposed.

Epilepsy resulting from Shell-Shock.—Pte. R., aged 24, had been in the Army since he was sixteen. He had never suffered from fits, but had an epileptic sister. He went to the front in September, 1914, and was wounded four times. The shell fire did not worry him and he enjoyed the life at first, but he became depressed after his father and five brothers had been killed on active service. He was blown up and buried three times in one day in July, 1916. He was unconscious for two hours on the second occasion, but carried on after resting another couple of hours until he was blown up again. He then became shaky and nervous, and slept badly. A month later he had a typical attack of major epilepsy. Fits now recurred with increasing frequency, and he has had as many as nineteen in one day. Rest and bromide led to their gradual cessation, and he had had no fit for six weeks when discharged from hospital.

The resisting power of this man was remarkable, as it was only after two years of fighting, and after he had been wounded four times, that severe family worry and repeated shell-shock led to the development of nervous symptoms ; it is probable that even then nothing more would have occurred had he not, like his sister, inherited a predisposition to epilepsy.

Even in severe lesions involving the membranes of the brain itself epilepsy is not likely to develop, except in those predisposed by a family history of fits or by the occurrence of fits in childhood, but in such individuals it may sometimes develop even with comparatively slight injuries. As, however, nobody can be regarded as completely immune, I agree with Craig and Buzzard that every man who has received a head injury, however trivial it may be, and whether an operation has been performed or not, should be given bromide regularly for at least a year. If he is specially predisposed, the bromide should be continued for two

years. The initial dose should be 30 grains a day, which can be gradually reduced to 10 grains. If, in spite of this, fits develop, the question of operation requires consideration. None should be performed if the wound has healed and left a healthy scar and no foreign body can be detected with the X-rays. But if fragments of metal or bone are lying superficially and easily accessible, they should be removed whether the wound has healed or not. Deeply situated fragments should be left, as more harm than good would result from the injury to the brain caused by the operation. An exception to this rule, to which Buzzard has drawn attention, is the presence of aseptic encephalitis, which gives rise to headache, vomiting, optic neuritis, and focal symptoms, often only after the patient has returned to a life of activity. This condition is a definite indication for operation. As an operation for epilepsy does not itself cure, but only improves the chances of cure, treatment with bromide is as important after as before its performance.

A soldier who has suffered from true epilepsy, whether traumatic in origin or not, is unfit for service abroad. The strain of active service is likely to increase the frequency of the fits, and there is considerable danger that post-epileptic automatism may occur, in which case serious results may follow both to the man himself and to his companions. On the other hand, men suffering from infrequent fits can often be employed with perfect safety in various capacities in the Army at home, as regular mental and physical employment, especially out of doors, is of the greatest value to them. They should, of course, continue to take bromide, as in civil life, until they have had no fit for three years.

Automatism and amnesia have been exceptionally frequent manifestations of epilepsy in soldiers. Many men, who had previously only suffered from ordinary major epilepsy or the common types of petit mal, and often only at rare intervals or not at all since childhood, have been found wandering far from their units and have been unable to give any account of themselves for several hours or even days. In such cases they run grave risk of being punished for deserting, unless the true nature of their condition is recognised. At the time it is generally impossible to ascertain whether their automatism and amnesia are due to epilepsy, as there are several other causes of these conditions.

such as simple concussion or the concussion of true shell-shock, extreme nervous exhaustion, heat-stroke, and even migraine. But when the patient's memory returns, a personal or family history of fits is strong evidence in favour of the epileptic origin of the amnesia. In some cases an accurate history has only been obtained after communicating with the soldier's relatives.

Automatism in a Man who had previously suffered from Epilepsy.—Pte. B., aged 29, was strange in his manner when last with his regiment at 7 a.m. on November 10th. The next day he was found by the police at a seaport many miles away without his equipment. He could give no account of his recent doings, and did not know what day it was. He gradually regained his memory during the following week. He then stated that from the age of twelve he had been subject to fits, which were preceded by an aura and were often accompanied by involuntary micturition and occasionally defæcation. He had had none for several months when he enlisted in February, 1916, and had remained well until the present attack. With this history there could be little doubt that the attack of automatism and amnesia was post-epileptic. He was recommended for employment at home.

(ii) Mental Disorders

I have seen a large number of men who have been sent home as congenital mental defectives with a note from their company commanders that they are too stupid to be of any use in the Army. They are slow and inaccurate in carrying out the simplest orders, and cannot be trusted with any duty which entails the slightest display of initiative. Many of them are so dirty in their habits that they become a danger to the health of their companions.

On investigating their past history it is found that the majority are true congenital defectives, who have never been of any use in civil life, but among them are a considerable number of stupid and badly educated men, who were able to do useful work and earn a fair wage in some manual occupation before they joined the Army, but they have been unable to adapt themselves to their new surroundings. They constantly get into difficulties with their N.C.O.s, and are teased by their companions, with the result that their mental capacity becomes more and more limited until they finally give the appearance of being hopelessly deficient, but sometimes not until they have served abroad for many months.

The change to the quiet life of a hospital in England is followed by rapid improvement, and many of them are soon able to do hard work in the hospital grounds, but it is doubtful whether any become fit for more than home service, and many would be more useful working on farms than in the Army.

A man who has once had a mental breakdown is very unlikely to be able to stand the strain of active service without relapsing. Fortunately recovery generally takes place on removal from the conditions which lit up the latent disease, but such men should never be allowed to return to military duty.

(iii) Syphilis, Predisposing to Tabes, General Paralysis, and Cerebro-spinal Syphilis

There must be a stage in the development of general paralysis and tabes, in which the spirochætes have invaded the central nervous system, but the patient has not yet noticed any symptoms. This explains the occasional discovery of latent tabes in the examination of patients with aneurysm and of apparently healthy individuals for life assurance or other purposes : the pupils may not react to light or the ankle-jerks may be unobtainable, and further examination shows that the cerebro-spinal fluid contains excess of lymphocytes and gives a positive Wassermann reaction, whilst the blood still gives a negative reaction.

When men affected in this way are exposed to the excessive strain of active service, instead of simply becoming neurasthenic they rapidly develop tabes or general paralysis.

Latent Tabes developing acutely as a Result of Excessive Fatigue.—A colonel, who went out with the original expeditionary force, regarded himself as perfectly fit, though he had occasionally suffered from pains in the legs, which had been ascribed to rheumatism and neuritis. He had an exhausting time during the great retreat, and was invalided home directly afterwards. I found that he was suffering from severe tabes, which had doubtless been present in a latent state before, but had become acute as a result of over-fatigue. With rest and active anti-syphilitic treatment he has improved greatly, but he has not been able to return to duty.

Tabo-Paresis developing during Active Service.—Pte. J., aged 38, had syphilis ten years ago. After having been in the trenches

at Salonica for some months, he became strange in his manner, doing foolish and dangerous things. On claiming to be the commander of an Army Corps on October 31st, 1916, he was sent into hospital. He had incontinence of urine both by day and night, but said that the urine was sweat. His pupils were unequal and reacted sluggishly; his tongue and facial muscles were tremulous, the right knee-jerk was absent and the left was only obtained on reinforcement; the right ankle-jerk was obtained with difficulty and the left was normal. The vibration sense was very deficient over the sacrum and malleoli, a sign which Captain J. L. M. Symns has shown is strong evidence pointing to the presence of tabes.

When admitted on December 14th his mental condition had much improved, but the physical signs were unaltered. The Wassermann reaction was strongly positive, and a diagnosis of tabo-paresis was made. He was given salvarsan, mercury, and iodide. By January 20th the ankle-jerks had become normal, the right knee-jerk was easily obtained and the left was slightly exaggerated. The tremor of the lips and tongue was less marked, and the patient's mental condition was quite normal. In spite of continued treatment the Wassermann reaction was still positive on February 23rd, but the patient's general health was now excellent, and there had been no return of mental symptoms. He was discharged for military employment in March.

General Paralysis developing on Active Service in an apparently Healthy Man.—A storekeeper, 33 years old, enlisted in March, 1915, and went to France in a labour battalion the following June. He had never done any hard work before, and the constant difficulty he found in avoiding reprimands and punishment for the non-performance of work which was beyond his strength was a constant source of worry to him. He carried on until December, 1916, when he was sent home as being no longer of any use. He was mentally very dull and his memory was much impaired. The only physical sign of disease was a very characteristic slurring speech which was accompanied by inco-ordinated movements of the lips; a diagnosis of general paralysis was made, and this was confirmed by the positive Wassermann reaction of his cerebro-spinal fluid, the reaction of the blood being negative. Great improvement followed treatment with mercury, iodides, and eight injections of salvarsan. By the beginning of February he was brighter mentally and his memory had improved; he was much stronger physically and enjoyed digging in the hospital grounds. His speech was slightly better, but was still very slurring.

Exposure of a man with incipient, but still latent, tabes or

general paralysis to the physical effects produced by the explosion of a powerful shell in the near neighbourhood is even more likely than simple physical and mental fatigue to cause these diseases to become suddenly obvious, at the same time as it gives rise to the composite clinical picture of primary organic and secondary hysterical symptoms which is known as shell-shock.

Tabes developing with Shell-Shock.—Pte. D., aged 31, fought in the retreat from Mons, and was blown up by a shell and buried in May, 1915. He returned to the front after being home two months, and remained well till he went into action on December 12th, 1916, when he was knocked unconscious by a shell. He came to himself two days later in hospital, but he was still confused and lethargic on reaching England on the 21st. His mental condition then improved and in a few days was normal. His legs were still weak and he could only walk a few paces very unsteadily. His right pupil was irregular in outline, eccentric, and much larger than the left; it reacted neither to light nor accommodation. The left pupil did not react to light, but reacted normally to accommodation. Very early primary optic atrophy was present. The right knee-jerk was slightly exaggerated (6), the left was normal (4); both ankle-jerks were normal. The vibration sense was much reduced over the sacrum and malleoli. The possibility of tabes was confirmed by the positive Wassermann reaction of the blood and cerebro-spinal fluid. With rest, mercury, iodide, and seven injections of salvarsan he improved rapidly. By the middle of February he could walk quite well, and felt almost as fit as before he was blown up. On the 17th March the right pupil reacted to accommodation, but was still inactive to light, irregular in outline, and larger than the left, which now reacted slightly to light as well as to accommodation.

Most cases of tabes and general paralysis in soldiers are due to the lighting up of latent and incipient disease as a result of the stress and strain of active service and the concussion caused by shell-shock, but it is not unreasonable to suppose that the same factors may so lower the resistance of the central nervous system that these diseases may develop in a syphilitic man, whose nervous system has not hitherto been invaded by spirochætes.

There can be little doubt that in syphilitic subjects shell-shock and the concussion of the spine produced by burial under collapsing parapets may be the direct cause of cerebro-spinal syphilis, in which the nervous system is attacked from the blood-stream by way of the meninges and blood-vessels, as distinct from the

parenchymatous syphilitic diseases, such as tabes and general paralysis.

Cerebral Syphilis developing with Shell-Shock.—Corporal B., aged 26, was blown up by a shell on September 7th, 1916. He was admitted on the 13th in a dazed condition. The right pupil was much larger than the left, all the signs and symptoms of left-sided organic hemiplegia were present. There was a superficial abrasion of the scalp in the right parietal region. He had had syphilis ten years before and the Wassermann reaction of the blood was strongly positive. The general symptoms and hemiplegia gradually disappeared with rest, salvarsan, mercury, and iodides; on December 12th he had still some weakness of the left side with exaggerated jerks, absent abdominal reflex and extensor plantar reflex, but he had otherwise almost recovered. As the Wassermann reaction was still strongly positive, he was given more salvarsan, mercury, and iodides. On January 6th, 1917, there was no appreciable weakness of the left side, the plantar reflex had become flexor, the abdominal reflex had returned, and Babinski's second sign (combined flexion of the thigh and pelvis) was the only evidence of organic disease. With further anti-syphilitic treatment this also disappeared, and the only physical sign remaining on February 28th, when he was discharged fit for light duty, was inequality of the pupils, but the Wassermann reaction was still positive, and he had a complete blank in his memory from the time he was in the trenches just before he was blown up until he found himself in hospital about four weeks later.

The following case shows how an actual wound of the head may be the exciting cause of general paralysis.

General Paralysis developing acutely after a Shrapnel Wound of the Head.—Pte. I., aged 31, was perfectly fit until he was wounded on December 7th, 1916, by a small fragment of shrapnel, which entered his skull 2 inches behind the left ear and lodged in the brain 1 inch above and 2½ inches behind the middle of the right orbital margin. When admitted to Netley on December 30th he had complete internal and external ophthalmoplegia of the left side with the exception of the external rectus, with complete paralysis of the superior rectus and partial paralysis of the inferior rectus and levator palpebræ superioris of the right side (Fig. II, 8); the left side of the face was also paretic, but there was no paralysis or anæsthesia elsewhere. The knee-jerks were equal, the epigastric and abdominal reflexes absent, and the plantar reflexes flexor, though the right one was said to have been extensor at the clearing station. The optic discs were normal. The patient was at first in a stuporous condition and had incontinence of urine and fæces

for two days. Shortly after admission he began to answer questions in a slurring and indistinct voice after a long latent period. As it was clearly inconceivable that the shrapnel fragment should have caused the isolated lesions of the third and seventh nerve nuclei without injuring any of the ascending and descending tracts of the crus, quite apart from the fact that the track it had taken must have been entirely above the crus, we thought that the concussion it had produced must have lit up some latent syphilitic disease. This view was strengthened by the positive Wassermann reaction obtained with the cerebro-spinal fluid, that of the blood being negative. Considerable improvement in the mental condition and some diminution in the degree of paralysis followed treatment with iodide and mercury. The speech now (March, 1917) is very suggestive of general paralysis; his mental condition favours the same diagnosis, as he is extremely pleased with himself and says he has never been in better health in spite of his disabilities.

When fits occur in soldiers who have not suffered from them in childhood, have no epileptic relations, and have not received any head injury, they almost invariably prove to be hysterical, unless they are syphilitic in origin. I believe that syphilis may give rise to a form of true epilepsy without other symptoms, but in most syphilitic cases the convulsions prove to be the first manifestation of commencing general paralysis. I therefore make it a rule in all cases of epilepsy occurring in soldiers, who have not previously suffered from it, to test the Wassermann reaction of the blood and the cerebro-spinal fluid. A thorough examination for early physical signs of organic nervous disease should, of course, also be made. I have discovered several cases of commencing general paralysis in this way, and vigorous treatment with mercury, iodide, and injections of salvarsan has proved unexpectedly successful in preventing the recurrence of fits and in improving the mental and physical condition of the patient, when these were also affected.

Syphilitic Epilepsy.—Pte. B., aged 27, had syphilis ten years ago. On March 21st, 1917, he had a typical epileptic fit. He had been two years in France but had not been under fire. He had never previously suffered from fits and none of his relations were epileptic. For some time he had had occasional visual hallucinations but was otherwise normal mentally. There were no physical signs of disease except a fine tremor of the hands which disappeared after a short rest. Both the blood and cerebro-spinal fluid gave a positive Wassermann reaction.

The headache associated with shell-shock and less frequently with neurasthenia is sometimes partly syphilitic in origin, as the cerebro-spinal fluid gives a positive Wassermann reaction, and rapid improvement follows anti-syphilitic treatment.

From what has been said it is clear that the possibility that syphilis is a factor should be considered in all cases of nervous disorders in soldiers, even when there is an obvious exciting cause such as painful emotions or shell-shock. The presence of abnormality of the pupils, absence or diminution of the ankle- or knee-jerks, tremor of the lips and tongue are particularly suggestive symptoms, and indicate that the cerebro-spinal fluid should be examined as well as the blood for the Wassermann reaction and the former for excess of lymphocytes.

Treatment.—Active anti-syphilitic treatment should be given in every case in which the Wassermann reaction is positive, even if it is uncertain whether syphilis is wholly or in part the cause of the symptoms. I begin with 2 drachms of iodide and 4 drachms of Liq. Hydrarg. Perchlor. a day ; a week later mercurial injections replace the mercury given by mouth and a course of salvarsan injections is begun. If severe cerebral symptoms are present, the latter should be postponed until the mercury and iodide have produced a certain amount of improvement. Intrathecal injections of salvarsan are not only theoretically irrational, but in practice they produce less good results than intravenous injections, and they occasionally give rise to spinal meningitis and thus actually aggravate the condition.

The disposal of soldiers suffering from syphilitic disease of the nervous system is a matter of considerable difficulty. It is probably unwise to allow any man who has had definite signs of his nervous system being affected by syphilis to return to active service abroad, even if apparent complete recovery has occurred and the Wassermann reaction has become negative, because with the strain of active service relapses are likely to occur. There is, however, no reason why he should not be given military employment at home, and I know of several men suffering from commencing tabes, which has improved or been arrested with treatment, as well as men who have recovered from various forms of cerebro-spinal syphilis, who have been able to carry out their military duties efficiently without suffering from any return of symptoms.

Men suffering from general paralysis, on the other hand, however much they appear to have improved, are almost certain to relapse, unless they live a particularly protected life, which would be impossible if they remained in the Army.

(iv) Previous or Simultaneous Disease, Injury or Wound

The pain caused by an injury to a limb may lead to hysterical paralysis in a man recovering consciousness after shell-shock, even if there is no actual wound and the injury is comparatively trivial. I have also seen several cases of hysterical paralysis following injury to peripheral nerves. It is very important to recognise that this occurs, as an accurate diagnosis greatly accelerates recovery. If a nerve is injured in such a way that it becomes functionless, the patient becomes so accustomed to the paralysis that when the nerve recovers sufficiently to convey voluntary impulses, no such impulses originate in the brain. Early in January, 1915, an officer received a bullet wound in his arm which resulted in complete paralysis and anæsthesia in the distribution of the median nerve. On January 20th Major F. J. Steward exposed the nerve and found that it was embedded in connective tissue, but it was not actually divided. The nerve was freed and the wound healed. I saw him on March 9th and found that the anæsthesia and paralysis were still complete, but all the muscles contracted briskly with faradism: this showed that there was no longer any nerve degeneration. With a little persuasion I managed to get him to move his fingers, and by repeating the faradism and persuasion a considerable amount of improvement occurred in the course of a few days. At the end of this time the paresis and partial anæsthesia, which still persisted, were probably organic, the hysterical element having been cured by suggestion and persuasion. In all cases of injury to peripheral nerves, whether they have been completely divided or not, the muscles should be tested from time to time by faradism, and as soon as they respond, the patient should be persuaded to make an effort to contract the muscles voluntarily, as otherwise the paralysis is likely to be maintained by auto-suggestion for several weeks or even months.

Babinski records the case of a patient with paralysis and reaction of degeneration of the muscles supplied by the circumflex,

musculo-cutaneous and musculo-spiral nerves caused by a bullet wound in the axilla. Flexion of the hands and fingers was also impossible, although the electrical excitability of the muscles of the front of the arm and forearm three months after the injury was normal. This showed that the paralysis of the flexors could not be due to an organic nerve lesion; the immediate recovery with purely psychical treatment proved that it was hysterical in origin and not due to nerve concussion. I have observed a similar combination of organic and hysterical paralysis in several cases of bullet wounds involving the brachial plexus.

A previous attack of neuritis and the slight weakness which may persist after almost complete recovery from anterior poliomyelitis are occasionally the predisposing factors which lead to a particular limb being affected by paralysis or tremor after shell-shock. In one case tremor developed in the left arm, which was slightly smaller than the right and in which some of the small muscles of the hand were weak and atrophied owing to the presence of a cervical rib. In another case a cervical rib gave rise to weakness and pain in the right hand, which had hitherto been unaffected, immediately after a man had been buried by the explosion of a shell so that only his head and right arm remained free, the violent struggles which followed having apparently caused the brachial plexus to be damaged by the cervical rib. Rapid atrophy of the small muscles of the hand followed, but removal of the rib by Major Graham Simpson four months after the injury resulted in complete recovery.

The statement has been made that it is very rare to find shell-shock and hysterical symptoms among the wounded, who are indeed wonderfully cheerful, although they must have been exposed to the conditions which induce it to a greater extent than the unwounded, among whom the large majority of cases occur. I do not, however, agree that functional neuroses are rare among the wounded, as I have seen large numbers of cases in which hysterical symptoms of various kinds were associated with wounds, the onset of the symptoms being due to circumstances connected with their infliction. Such symptoms as mutism are, however, undoubtedly rare in the wounded. The probable explanation is that in the confused state, which generally precedes the development of hysterical symptoms, the incapacity and pain caused by

a wound absorb the patient's entire attention, so that the only symptoms which are likely to develop as a result of auto-suggestion are hysterical paralysis, contractures and pain in the neighbourhood of the wound.

Shrapnel Wound of Chest associated with Hysterical Paraplegia and Tremor.—Sergt. B., aged 24, was wounded on August 12th, 1916, in the chest. A hæmothorax developed and was aspirated in France. The X-rays showed on January 2nd, 1917, that a small piece of metal was situated in the left lung near the heart, but there was no abnormal opacity in the chest, and no physical signs of disease. The patient had been kept in bed for two months, and when he was allowed to sit up he found that he was quite unable to walk, and the least effort resulted in general tremor, attacks of severe dyspnœa and pain in the chest. He was then transferred to the neurological section from the surgical wards. As a result of suggestion under hypnosis, he soon realised that his chest was really healthy, the dyspnœic attacks ceased, and the pain disappeared. Prolonged re-education was required, however, before he learnt to walk normally.

(v) Past Emotional Disturbances. Psycho-Analysis

In rare cases past experiences, which were associated with powerful emotions, but which may in the course of time have been almost forgotten, are reawakened by recent terrifying events and give colour to some of the symptoms. I have seen a few cases, in which nightmares have appeared to result from experiences at the front becoming blended with episodes, which occurred many years before and made a profound impression on the patient's mind.

A number of Freud's disciples have expressed their opinion that war neuroses are due to unconscious mental conflicts, mostly of a sexual character, and that they should be treated by means of psycho-analysis. The fallacies of Freud's doctrines have been so admirably and conclusively exposed by Mercier in his paper on " Psycho-analysis " in the *British Medical Journal* for December 30th, 1916, that the subject would hardly require mentioning, were it not for the fact that psycho-analysis is still being used in treatment by the few followers of Freud, who happen to be in charge of British soldiers suffering from functional nervous disorders. The mental attitude of the pyscho-analyst is well shown

in a paper on "Functional Nerve Diseases and the Shock of Battle," by David Forsyth, who states that "the neurasthenia of civil practice is known to be the specific result of excessive onanism"! He proceeds to describe two cases of exhaustion following the stress and strain of active service, one of which his analysis showed was due to "unconscious homosexuality" combined with "anal erotism, which has played a leading part in the patient's psychical life since an attack of diarrhœa in childhood," and the other was due to "a strong Oedipus-complex"—the psycho-analyst's name for incestuous lust.

I have seen a considerable number of medical case sheets of soldiers who had been in charge of psycho-analysts. In almost every case an attempt appears to have been made to discover some sexual origin of the symptoms, obvious causes such as shell-shock and the stress and strain of active service being more or less ignored. Some form of "Oedipus-complex" was suggested in many instances, simply because the patient had occasionally slept in bed with his mother when a small child, or because her death had caused him much distress. A man sent home from France on account of epilepsy had suffered from fits when a small boy. When eight years old his parents had sent him to live with relations in California so that he could live an open-air life. The fits had become very infrequent, and he had not had one for a year and a half when he enlisted, but the stress of active service caused the fits to become more frequent again. This very straightforward case had been "analysed," with the result that the fits were said to be due to unnatural lust for his mother, whom he had not seen since he left home in 1904.

The nasty ideas suggested by a sexual cross-examination and by pyscho-analysis are bad enough, but I have seen still more deplorable results of the method. An unmarried sergeant, twenty-six years old, who was worn out and worried as a result of unaccustomed responsibility, was slowly improving with rest and encouragement, when his "unconscious mind" was subjected to analysis. A petty larceny he had committed as a boy and had almost forgotten was raked up, and in his somewhat emotional condition it caused him acute distress. He had never felt any great sexual desire, but was told on discharge from hospital that he must indulge himself at least twice a week.

He was readmitted a month later very much worse, as his mind was torn between the reluctance he felt to follow this advice, both on account of conscientious scruples and absence of desire, and fear that disobedience would result in insanity. At the same time he had felt an almost irresistible impulse to return to the hospital to murder the analyst of his "unconscious mind." Encouragement and occupation resulted in slow improvement, but he continued to hide himself at the approach of the Freudian medical officer under whose care he had originally been.

Lastly, as an example of the psycho-analyst's explanation of war-dreams, the following interpretation by Captain M. D. Eder is quoted from his book on "War-Shock." The patient had served in France and the East and was admitted for physical and mental exhaustion. He dreamt he was "on a boat running out of A—— Harbour, going down the River B——. We went through a narrow passage which came gradually to a point, and got stuck in the mud. Ship did not stop. Looking out of the porthole window I saw a Zeppelin in the distance. It was attacked by a fleet of balloons." Psycho-analysis showed that "the narrow passage stood for the vagina and the mud was the anal region; the boat coming down the passage (the vagina) was the patient himself." The balloon was his mother's womb, and the Zeppelin his own phallus, "his phallus (Zeppelin) being the prey of women (balloon) which will destroy his male power." The effect of such an explanation of a very ordinary war-dream on the mind of a war-worn soldier cannot be anything but disastrous, and even Eder admits that no improvement resulted. Few will disagree with the conclusion that the sooner that psycho-analysis of this kind ceases to be practised in England the better it will be for all concerned. I entirely agree with Rivers that the symptoms which are regarded by Freud and his followers as pre-eminently the result of conflicts between repressed sexual tendencies and experiences (so-called complexes) and the general personality are common in soldiers, but their sexual life is absolutely normal, and the symptoms are directly due to the stress and shock of warfare. The unburdening of the soul, the only healthy part of Freud's teaching, is nothing new, as it has been used from time immemorial by the laity as well as by doctors for helping the distressed in mind.

CHAPTER VI

MOTOR DISORDERS

In this and the following chapters some of the more common war neuroses, which have already been referred to in the discussion on their pathogenesis, are more systematically described.

(i) Hysterical Paralysis

A great variety of hysterical paralyses may occur in soldiers either alone or associated with tremor of the affected limbs. In other cases contractures may be present, but they generally involve only a few of the paralysed muscles. The paralysis may be more or less complete, but in many cases it takes the form of *astasia-abasia*, in which movements can be carried out normally or almost normally whilst lying down or even sitting, but standing and walking are impossible.

I have already described how temporary organic paralysis caused by shell-shock or spinal concussion may be the starting-point of hysterical hemiplegia, paraplegia, or monoplegia (p. 51).

Occasionally paralysis follows a slight head injury as a result of an idea, and not of actual concussion. A man receives a superficial wound of his scalp from a glancing bullet, or as a result of being thrown on to the ground or against a wall or parapet by the explosion of a shell. The brain is not injured and no sign of organic disease is found, but hemiplegia develops—most commonly on the side of the injury, corresponding with the patient's own idea of the paralysis which he has heard may result from an injury to the head.

Hysterical paraplegia may also result, as I have already described, from the perpetuation of the feeling of weakness in the legs caused by terror.

Hysterical Paraplegia following Extreme Fear.—Pte. C. was

cut off from his companions during a night raid at Salonica. He thought that he would be tortured if he fell into the hands of the enemy, but found that he could not move as he was "paralysed with fear." He was rescued next morning, but remained paraplegic for three and a half months. He was then admitted into the neurological section at Netley, and was cured by vigorous persuasion on the following day.

In the following case of hysterical paraplegia the origin of the suggestion of paralysis was quite clear, and again there was no organic basis.

Hysterical Monoplegia and Contracture cured by Hypnotism.—A Belgian soldier was observing the enemy, when the roof on which he was sitting collapsed, and he fell a short distance into the mud below without hurting himself in any way. It was over an hour before he could extricate himself, as his left leg was embedded in the mud. When at last he was able to crawl away, he found that he could not move the leg, which was fixed rigidly in an extended position. As no improvement occurred during the next few days he was sent to England. When I saw him three months later, his condition was unaltered, and he was still unable to walk without assistance. The left leg was absolutely rigid, but he did not walk as if he had organic spastic paralysis, but dragged the leg behind him. There was no voluntary movement, and the knee and ankle could only be bent by using considerable force. The whole leg was completely anæsthetic to all forms of stimulation. The plantar reflex of both sides was flexor; the knee- and ankle-jerks were unobtainable on the left side owing to the impossibility of relaxing the muscles. There was no other evidence of disease in his nervous system. The absence of any organic sign, combined with his extraordinary gait, which was quite different from that of any organic disease, made me quite certain that the paralysis, spasm, and anæsthesia were hysterical. Additional proof of this was obtained from Babinski's second sign: when he lay down with his arms folded and legs widely separated and tried to sit up, the normal leg rose in the air and the paralysed leg remained fixed to the ground. The paralysis and rigidity resulted from perpetuation by auto-suggestion of the immobility in an extended position caused by the leg being fixed in the mud. In all probability the anæsthesia was produced by suggestion by one or more of the numerous people who had examined him during the three months he was incapacitated, as hysterical anæsthesia is very rarely the result of auto-suggestion, but is almost invariably produced by the observer. Having decided that the condition resulted from suggestion, it

only remained to remove it by suggestion. I passed a strong faradic current through the affected leg and told the patient that this treatment was quite certain to restore his sensation and his power of movement. He improved at once, but was still only able to walk with difficulty. I therefore hypnotised him, and suggested that he should move his leg in a normal manner. Mr. C. H. L. Harper repeated the treatment on several occasions, and at the end of three weeks he was sufficiently well to be able to return to duty, though he still kept his left leg somewhat stiff when he walked.

Severe pain in a limb leads to inability to move it. This is a reflex protective reaction, as movement would be likely to aggravate the injury which caused the pain. The inability to move is partly due to paralysis and partly to spasm of the muscles, which generally regain their normal activity in a short time. But in soldiers who have become abnormally suggestible from the strain of warfare the incapacity is likely to become perpetuated by auto-suggestion.

The suggestibility may be so extreme that hysterical symptoms result from auto-suggestion under conditions which rarely if ever lead to them in civil life. Thus I have twice seen hysterical paraplegia develop after an operation for acute appendicitis, which had developed in the trenches (Fig. II, 9); it has followed malaria, cerebro-spinal meningitis, trench nephritis, and gassing, and in several cases muscular rheumatism, which also gave rise to hysterical paralysis of the left arm in another patient. Hysterical paralysis of a leg followed trench fever in one case and paratyphoid arthritis of the hip in another, and an officer suffered from hysterical hemiplegia for two years after an attack of sunstroke. In these cases the temporary incapacity caused by the pain or the weakness associated with the primary illness or with some past injury was sufficient to give rise to the idea of paralysis.

Hysterical Astasia-Abasia following Appendicitis.—A man developed an acute attack of appendicitis whilst in the trenches, and his appendix was removed in a field ambulance the same day; when he tried to get up a fortnight later he was found to be almost completely paraplegic. The attack was accompanied by pain in the right and to a less extent in the left leg, and both legs were flexed on the abdomen. It was this pain which presumably gave rise to the idea of paraplegia, the right leg being more severely paralysed than the left. I saw him six months

afterwards; he could then move his legs in bed, but could not stand or walk. There were no signs of organic disease, and with persuasion and re-education he slowly learnt to use his legs again.

Hysterical Monoplegia following Trench Fever.—A man received a blow on his right knee on August 1st, 1916, from a piece of shell, which knocked him down and produced a bruise, but the skin was not broken, and he was able to carry on. On August 16th he suddenly felt weak and dizzy whilst digging; a severe headache followed and he found he could not walk. This proved to be the onset of a typical attack of trench fever. When the fever had subsided, he found that his right leg was completely paralysed. There were no signs of organic disease. The inability to move the leg was primarily due to a protective reflex, as any movement increased the pain; but when the pain had disappeared this inability was perpetuated by auto-suggestion, as it had revived the memory of the injury and he thought himself that the pain was due to the injury. After the mental origin of the paralysis had been explained to him he was persuaded to walk normally in the course of half an hour.

Hysterical Paraplegia following Cerebro-Spinal Meningitis.—I was asked to see a corporal, aged 24, who had been unable to sit up in bed or move his legs since an attack of cerebro-spinal meningitis five months before. Examination showed that there was no evidence of organic disease. His serious illness had made him abnormally suggestible; having found during the earlier stages that any attempt to move his legs gave him pain, he ceased to make any effort, and in the absence of any contra-suggestion by those who were looking after him he came to believe he could neither sit nor walk. Five minutes talk followed by vigorous persuasion was all that was necessary to produce an immediate and lasting cure.

Hysterical Hemiplegia of Two Years' Duration and Fits following Sunstroke cured by Suggestion.—Lieut. B., aged 24, a well-built, powerful man, was a farmer before the war. In May, 1915, after being a month in Egypt, he had sunstroke. He was unconscious for about three days, but was able to leave hospital at the end of three weeks. A few days after returning to duty his horse fell and rolled on to his left leg; at the same time he bruised his left shoulder, but he was not badly enough hurt to go to hospital. In the following month he had sunstroke again. The slight injury to his arm and leg now brought the idea of paralysis to his mind, which was in an abnormally suggestible state during the semi-stuporous condition which followed the sunstroke. On

leaving hospital three weeks later he returned to duty, feeling fit except for weakness and aching of the left arm and leg. The arm got steadily weaker until it was completely paralysed by January, 1916. He was first treated by strict isolation, but no improvement occurred in four months. He was then given two months' leave and improved for a time, but he was back in hospital in August, 1916, with complete left-sided hemiplegia. He was told that the electrical treatment he was having would suddenly cure him, and he woke up one morning in January, 1917, with the arm and leg very much better. He returned to light duty, but by March the paralysis was as complete as ever. On June 22nd, after a thunderstorm and standing in the sun, he had an hysterical attack, in which he struggled violently and bit everything he could get near to, including the medical officer and his own arm, but he did not bite his tongue. His eyes were wide open and the pupils and pulse remained normal. The attack continued with intermissions from 10 p.m. to 2 a.m., when it ceased after an injection of ½ gr. of morphia. He had five more similar attacks, but he was less violent unless an attempt was made to hold him. During the fits the muscles of the left arm contracted, but the arm did not move.

On admission to Netley on 13th July, 1917, complete flaccid paralysis of the left arm was present, except for some weak movements of the hand. The left leg was very weak, and the patient dragged it behind him as he walked. All superficial and deep reflexes were normal, and the muscles contracted briskly to faradism, but the prolonged disuse had led to definite atrophy of the muscles of the left arm, especially of the shoulder, and the hand became blue, cold, and slightly œdematous after hanging by his side most of the day. By means of persuasion and re-education some improvement occurred in the leg, which he no longer dragged, but he still limped. Suggestion with the aid of electricity had very little effect, and though the fits ceased as a result of hypnotic suggestion, this treatment had no effect on the paralysis. He was therefore given ether on the 17th July after being told that the treatment by suggestion would have more effect when he was partially anæsthetised. He became very excited ; his right arm and leg were held down when he began to struggle, and with the aid of vigorous persuasion and suggestion his left arm and leg soon moved violently. He quickly woke up, and five minutes later he was walking without a limp, and was able to perform every movement of his left arm in a perfectly normal manner. He was still perfectly well in October, 1917, when he returned to full duty.

Hysterical Attitudes and Gaits

A great variety of hysterical attitudes and gaits result from the perpetuation and exaggeration of the attitude and gait, instinctively assumed by a patient suffering from some organic injury or disease of the leg or trunk, as being those in which he finds he has least pain and in which he can stand or walk best in spite of any weakness which may be present. The injury or disease may be quite trivial, and the posture and gait often persist long after all pain and weakness have disappeared. The patient says he limps because of pain; the truth is that he has pain because he limps. The attitude which gave relief when the original painful condition was present now causes pain, because some muscles which were hitherto little used are called into continuous activity, while ligaments and other muscles are stitched.

A good example of an hysterical attitude is the condition described by French writers as *campto-cormie* or *campto-rachis* (κάμπτω, I fold), in which the back is bent like that of an old man with spondilytis deformans. It is generally only present when the patient stands or walks, as he can lie comfortably on his back or face. Sometimes, however, it persists even when he lies down, so that he can only rest on his side. The attitude on standing is exactly that which would be assumed by a man who voluntarily bent forward, the same muscles being contracted and the same folds appearing on the abdominal wall. The head is thrown far back so as to increase the field of vision and facilitate walking. The patient progresses slowly, leaning forward on two sticks, without which he is convinced he cannot move. I have seen this condition follow a blow on the back by the earth of a collapsing trench, and in three cases it followed a spontaneous attack of pain, which was presumably caused by muscular rheumatism. Another case occurred after tetanus, which had caused pain in the back, but had not led to actual opisthotonos. In all cases the posture appears to have been at first assumed because of the comparative comfort it gave; when the painful condition originally present disappeared the patient was convinced he could not give up the posture, the fixation of which was thus hysterical in origin. In December, 1915, I saw an eighteen-year-old soldier at Lemnos, who had developed the condition gradually during the previous three

months at Gallipoli; this case and another, which followed a blow by a falling sand-bag four months before, were cured within forty-eight hours by hypnotism. A third case, which had lasted for an equal period, was cured as rapidly by simple persuasion. Improvement was, however, much more slow in a sergeant, who had already been treated without success for over a year by massage and electricity, and by hydrotherapy at Bath, with the result that he was firmly convinced of the permanent and serious nature of his complaint. Explanation and vigorous persuasion, followed by re-education, led to gradual improvement.

I have seen several cases in which sciatic neuritis or a wound in the neighbourhood of the sciatic nerve has led to the adoption of an abnormal attitude and gait, which were not given up when the pain due to the primary condition had disappeared. Such cases are very resistant to treatment, but a combination of suggestion, persuasion, and re-education eventually lead to recovery.

Abnormal gaits are not uncommon after trench foot; the patient generally walks on the outer side of his feet with his toes in a position of extreme dorsi-flexion. In all these conditions the abnormal postures generally disappear on lying down, although in the pre-hysterical stage, when the posture is protective, they are maintained in all positions.

In the following case the gait was very abnormal, although no paralysis or contracture, or abnormal attitude on standing was present.

Hysterical Dancing Gait following Muscular Rheumatism.—A man, who had been through the 1916 campaign in Salonica, was taken ill on January 1st, 1917, with severe pain in the legs. He was carried to the hospital, where the muscles of his legs were found to be very tender. On trying to get up on January 4th he found he could not walk owing to the pain, but he could get along by a sort of dancing gait, in which he slid each foot backwards about half a pace every time it touched the ground; in this way he managed to progress at a considerable speed. The pain soon disappeared, but he now found that he could not move slowly, and he continued to dance along in the same extraordinary manner. The condition was recognised as hysterical when he reached Malta, but treatment by hypnotic suggestion for three weeks produced no improvement. On March 20th he came under my care. I explained to him that the gait which had at first been the means of saving him from pain had now become a bad

habit, and that he must try to cure himself by copying every movement I made when I walked. He did this for ten minutes, at the end of which he could walk fairly well. By the next morning, greatly to his delight, he was walking quite normally, and now found it difficult to reproduce his old dancing gait.

Diagnosis of Hysterical from Organic Paralysis.—As hysterical paralysis is a result of auto-suggestion, the paralysis corresponds with the average layman's conception of paralysis. A number of signs occur in organic paralysis, which would not be expected by the uninitiated. They are, therefore, not observed in hysterical paralysis and serve to differentiate organic from hysterical conditions. In organic hemiplegia, if the supinated arm is completely relaxed and then tossed, it falls in a position of pronation, but in hysteria it does not turn. When a patient with organic hemiplegia grips the observer's hand with his normal hand, an associated movement occurs in the paralysed hand, sometimes even when no voluntary movement can be performed, but no such movement occurs in hysteria. The tendon reflexes of the arm and leg are almost always exaggerated on the affected side in organic hemiplegia, though they may be absent or deficient in the early stages, and ankle-clonus is often obtained ; in hysteria they are invariably equal on the two sides, and true ankle-clonus, the clonus produced in a completely relaxed limb, is rarely present. The abdominal reflex on the paralysed side is weak or absent in organic disease, but equal to the opposite side in hysterical paralysis. The plantar reflex is generally extensor in the former, but invariably flexor in the latter (Babinski's sign). When a patient tries to raise himself from the horizontal to the sitting position with his arms folded and his legs widely separated, the paralysed leg rises higher than the other in organic disease, but remains on the ground in hysterical hemiplegia (combined flexion of the thigh and pelvis—" Babinski's second sign ") ; I have seen several cases of hemiplegia following shell-shock in which this was the only evidence of its organic origin. In a well-marked case of organic hemiplegia most or all of these signs are present ; in hysteria no single one is present.

In most cases of hysteria the diagnosis is made still more certain by the gait or some other prominent feature being of a nature quite unlike anything seen in organic disease. Astasia-

abasia, in which the patient can move his legs normally whilst lying or sitting, but cannot stand or walk, is common and is always hysterical. Occasionally some inconsistency about the symptoms points to hysteria. An officer became hemiplegic on the right side after being blown up by a high-explosive shell in the Dardanelles. He was taken to Cairo and then sent home. When he said good-bye to his father, who came to see him at Cairo, he shook him strongly by the hand. He told me of this as a remarkable incident when I saw him in London, so it was obvious that he was not malingering. He was cured at once by suggestion during hypnotic sleep.

The following case shows how great may be the difficulty of diagnosis when organic and hysterical symptoms are associated together.

Paraplegia following Shell-Shock: Organic or Hysterical.—Pte. M., aged 29, a champion heavy-weight boxer, was knocked over by the explosion of a high-explosive shell in December, 1914, and remained unconscious for two days. When he regained consciousness, he found that he could not move his right arm or his left leg. Power in both limbs soon returned to some extent, but as soon as he tried to stand, violent involuntary movements occurred in his left leg.

I first saw the patient on April 1st, 1915. His mental condition seemed to be impaired; he only answered questions after a considerable latent period, and his speech was slow. The whole of his right arm was weak, the grip being particularly feeble. When he clenched his left hand an associated movement occurred in the right hand, but on clenching the right hand no similar movement occurred in the left hand. The muscles of both arms were equally well developed. The tendon reflexes in both arms were brisk, but were no better marked on one side than the other. The patient was unable to localise light tactile stimuli accurately, but otherwise there were no sensory disturbances. All movements of the left leg were somewhat weak. The muscles were equally well developed in both legs. Both knee-jerks were brisk, the left one being slightly brisker than the right. Well-marked ankle-clonus could be obtained on the left side. The plantar reflex was constantly flexor on both sides, but Babinski's second sign was very well marked on the affected side. As soon as the patient attempted to walk, violent involuntary movements were set up in the left leg: the leg rapidly oscillated round the point where the toes were in contact with the ground. When

a step forward was taken with the right leg, the left leg dragged behind and very irregular movements occurred. The gait seemed to be so obviously hysterical, and the signs pointing to organic disease were so slight, that it was thought that all the symptoms would probably be cured by suggestion. The patient was kept in hospital for a month, but all efforts to cure him by means of suggestion entirely failed. He proved very easily hypnotisable, but even when deeply hypnotised the movements of the leg could not be controlled when he was told to walk. Dr. Batten suggested that etherisation might be of use, but the first whiff of ether had the effect of hypnotising him. In May, 1917, he was in exactly the same condition as when he was first seen, except that his mental condition had greatly improved.

The associated movement of the paralysed hand when the normal hand moves, the slight exaggeration of the left knee-jerk, and the tendency to ankle-clonus, and above all the presence of Babinski's second sign, indicate that some organic changes have occurred in the brain as a result of the concussion. The complete failure of suggestion to produce any improvement raises the question whether all the symptoms, in spite of their unusual character, may not be organic in origin.

Treatment.—A paraplegic man is given rubber-soled shoes, but is not allowed to use crutches or a stick. He is first helped to move his legs whilst he lies in bed, and he is then made to move them without help. When the patient is already able to move his legs whilst lying down, although he cannot stand or walk, he is told that his muscles are now so strong that there can be no doubt he will walk if he summons up sufficient courage to try. In both cases without further delay he is made to sit up in bed, stand up and walk ; both hands are held for a moment, then one hand and then his coat is lightly held, and finally he is made to walk alone : in most cases the whole process takes less than ten minutes.

In long-standing cases the patient generally assumes a stiff, unsteady gait on first learning to walk ; this only disappears as a result of re-education, the patient being made to walk, perform exercises whilst lying and sitting, and swing his legs whilst sitting on a table or holding a chair, for at least a quarter of an hour three times a day. Exercise on a rowing machine or tricycle is also useful.

Some cases of long-standing hysterical paraplegia improve to a certain extent with persuasion and re-education, but then become

stationary. This is generally due to the occurrence of clonic spasms, when an effort is made to move the legs on lying down or even when they are passively moved. Acting on a suggestion of Claude's, we performed spinal anæsthesia on a man with this condition, who had shown no improvement for many weeks. As its effects passed off we carried out passive movements of his legs, which had become flaccid for the first time, and then encouraged him to perform similar active movements, and by the evening he was walking normally. We have repeated the treatment in several similar cases; with one exception improvement followed, but not often so rapidly.

(ii) Tremor

Hysterical tremor is very common and is due to the perpetuation by auto-suggestion of the tremor caused by fear. Although it is more resistant to treatment by persuasion and suggestion with and without hypnotism than any other hysterical symptom except in the very early stages, it is much less persistent than the finer tremor produced by nervous exhaustion and hyperthyroidism. It is generally coarse in character, and may simulate the tremor of paralysis agitans or disseminated sclerosis. It may affect the whole body or be limited to the head, jaw, one side of the body, both arms or both legs, or a single limb. It generally persists when the patient's attention is drawn away from himself, though it is increased when he is excited or spoken to. The tremor is always accompanied by more or less rigidity of the affected parts, and ceases when complete relaxation occurs. Thus it always stops during sleep, and under hypnosis if sufficiently deep. It is generally increased when any effort is made.

A form of spasmodic movement, which can hardly be called a tremor, is common in hysterical paraplegia. It is absent when the patient lies quietly in bed, but begins as soon as he tries to move his legs; extremely violent irregular movements may then occur, and they may spread from the legs so as to shake the whole body. An attempt to produce ankle-clonus has a similar result, but the violent movements are not true ankle-clonus, as they only occur when the muscles of the legs are contracted, and if complete relaxation can be obtained with the knees bent and the leg resting

on the bed, no clonus is present. Tremor often begins on sitting up the moment the foot touches the ground; this also is due to a kind of false ankle-clonus, and can generally be stopped by stretching the legs forward with the feet resting flat on the ground so as to relax all the muscles. The patient should be instructed to keep his legs in this position, as the less he shakes the quicker he will get rid of the tendency to shake. On standing up and attempting to walk, the same clonic movements occur; they give rise to a characteristic jerky gait, and the trembling may be so excessive that walking is quite impossible, although the muscular power of the legs may be good.

In the earliest stages hysterical tremor can be rapidly cured by vigorous persuasion and re-education. When it has been present for many weeks or months it becomes extremely resistent to all forms of treatment; in most cases isolation with suggestion and re-education give the best results. The fine tremor of neurasthenia and hyperthyroidism very slowly disappears with prolonged rest; it is most important that the patient should be taught to relax his muscles completely.

(iii) Hysterical Contractures

Hysterical contractures may be present alone or associated with hysterical paralysis, which is generally more widespread than the contracture. Thus a man with hysterical paralysis of his right arm had his fingers tightly clenched, but the rest of the limb was flaccid. In another case flaccid paraplegia was associated with contracture of the right hand. Hysterical contracture may follow wounds, often of a very trivial nature, but the attitude assumed has no anatomical or physiological relation to the wound; it depends upon the patient's own ideas and is often unlike any position assumed in organic disease, but it is always capable of exact imitation by voluntary action. On attempting to overcome organic contractures no change occurs in the muscles involved, but in hysterical contracture increased resistance, which is often intermittent, is felt, and if the patient's attention is distracted, it may be possible to overcome it completely. An Esmach's bandage applied to the limb diminishes organic spasticity, but increases hysterical contractures. The former relaxes incompletely

in sleep and slowly under general anæsthesia, the latter completely in sleep and rapidly and completely under anæsthesia. The diagnosis of hysterical contractures from reflex contractures and from local tetanus is described elsewhere.

The treatment is on the same general lines as hysterical paralysis. Forcible passive movements in the direction opposed to the normal action of the contracted muscles, combined with verbal suggestion and continued without interruption until the resistance is broken down, however much pain may appear to be produced, almost invariably cures hysterical contractures in a few minutes even if they have already lasted for weeks or months. The passive movements are then combined with active movements until the condition of the limb is normal. In rare instances, as pointed out by Reeve, the passive movements may have to be continued for several hours, in which case relays of convalescent patients carry on the treatment. Finally the muscles become fatigued and toneless and no longer capable of producing the original deformity.

(iv) Reflex Paralysis and Contracture

Babinski and Froment believe that many of the paralyses and contractures, which have hitherto been regarded as hysterical or due to some obscure condition such as an ascending neuritis, are really reflex in origin. This view at first seems to offer a satisfactory explanation of many cases, the nature of which had hitherto remained obscure, but a critical investigation of the subject leads to the conclusion that though such reflex conditions do indeed occur, they are very rare indeed, and that there is a danger of accepting the diagnosis for certain hysterical conditions, which would consequently not receive the benefit of the psychotherapy, which would otherwise have cured them, and for local tetanus, when the mistake might be even more serious.

Etiology.—According to Babinski and Froment reflex paralysis and spasm may occur as a result of injuries to the soft parts of the limbs with or without the bones and joints being involved. The commonest cause is a wound through the hand, foot, forearm, or leg, the symptoms generally developing above and below as well as in the immediate neighbourhood of the injury in areas

corresponding with no special anatomical distribution. The severity of the reflex symptoms does not vary with the amount of infection or extent of the injury, which is often trivial. They are in fact very rarely associated with severe wounds or in amputations, even of a finger. A feeble peripheral circulation appears to predispose to reflex symptoms, especially those depending upon vasomotor changes.

Symptoms and Diagnosis.—The symptoms are generally discovered when the dressings are removed, but may only develop gradually several weeks later. A single segment of the limb is generally involved, but the whole is occasionally affected. Paralysis and contracture may be present together in the same or different segments of a limb. They are often accompanied by muscular atrophy, exaggeration of tendon reflexes, and hypotonus, as in the similar reflex symptoms associated with arthritis; increased excitability and slow contraction of muscles on mechanical stimulation and increased or diminished electrical excitability without reaction of degeneration occur, together with exaggeration of mechanical and sometimes of electrical excitability of nerves. Excessive sweating and diminution or loss of sensation are present over the affected segment. Decalcification of bone, trophic changes in the skin, with excessive growth of hair and thinning of the nails may be present. When the surrounding temperature is low, the cutaneous reflexes are lost and the affected part becomes painful, cyanosed and cold, and the maximum oscillation of the pulse, as measured when the blood-pressure is estimated, is less than on the opposite side.

These reflex symptoms resemble hysterical symptoms in being quite out of proportion with the often trivial injury which gave rise to them. But they differ in being entirely unaffected by suggestion; the tendon reflexes are often exaggerated, and the muscles and sometimes the nerves are abnormally irritable to mechanical stimulation, the response being unusually prolonged. Babinski and Froment have shown that in some cases these signs are only obvious when the patient is given a general anæsthesic; at the same time any contracture which is present relaxes less readily than in hysteria. The position assumed by the limb does not necessarily correspond with any position which could be assumed voluntarily, as is always the case in hysteria, and fibrous

contractures, which are very rare in hysteria, rapidly develop. Marked atrophy is often present in the paralysed limbs, in contrast, according to Babinski and Froment, with its complete absence or the very slight amount caused by disuse in hysterical paralysis. The vasomotor and trophic changes and excessive sweat are said by them to be never present in hysteria. I believe, however, that the distinction between hysterical and reflex symptoms is much less clearly defined than these authors assume. I agree that muscular atrophy and trophic, secretory, and vasomotor changes never occur as primary hysterical symptoms, as they cannot be produced, modified, or caused to disappear by suggestion. But I have seen paralysis, which was proved to be hysterical by the immediate cure following suggestion, lead to a considerable degree of atrophy after persisting for two years, and the paralysis and contracture may be so profound that secondary trophic and secretory changes may occur as a result of the venous stasis, which is a natural sequel of prolonged and complete immobility, especially if accompanied by contracture in a position in which the veins are obstructed by the rigid muscles. Moreover, the response to psychotherapy does not always afford conclusive evidence ; whilst early hysterical symptoms can invariably be cured with great rapidity by suggestion, symptoms which have lasted for long periods, owing to their nature having been misunderstood before psychotherapy is employed, may be extremely resistant. The success of psychotherapy depends largely upon the physician's confidence in his diagnosis. If he believes a symptom may be organic or reflex in origin, his treatment will be far less effective than if he is convinced it is purely hysterical. The following case was regarded by Captain J. L. M. Symns, Dr. Hilred Carlill, and myself as corresponding so closely to the description given by Babinski and Froment that we regarded the condition as probably reflex. In spite of this we tried the effect of vigorous suggestion : to our surprise it cured the paralysis, although it had lasted for over a year, so that its hysterical nature was finally proved.

Hysterical Contracture and Paralysis, at first supposed to be Reflex, cured by Suggestion.—Pte. W., aged 21, received a slight wound in the hand in May, 1916. When the dressings were removed, his hand was found to be fixed with the fingers semi-flexed and the thumb adducted. Movements of the elbow and wrist

were normal, but he could make no movements with the fingers or thumb. The wound soon healed, but the condition of the hand persisted in spite of massage and electricity. It had been supposed that the contracture was due to adhesions involving the tendons and palmar fascia. I first saw him fifteen months after the wound was received; no improvement had occurred, the fingers and thumb being still flexed, but it was found that the contracture could be overcome by a considerable effort. When a voluntary effort was made to move the fingers or thumb the affected muscles were seen to contract, but no movements resulted, as the muscles opposing the desired movement contracted instead of relaxing. There was no atrophy and the electrical reactions were normal (Fig. II, 6). The hand was swollen; it was red when the room was warm and blue when it was cold, a considerable difference in temperature between the two hands being always present; excessive sweating also occurred on the palm of the affected hand.

The condition would at once have been diagnosed as hysterical, had it not been that a recent study of Babinski and Froment's work on reflex paralysis and contracture led to the suggestion that this was an example of such a reflex condition, as the position of the hand was identical with that shown in one of the figures in their book. Œdema, vasomotor changes, and excessive sweating, according to them, never occur in hysteria, but are common in reflex disorders.

Although a provisional diagnosis of paralysis and contracture due to reflexes from the scar was made, the effect of vigorous suggestion with the aid of electricity under light etherisation was tried, as I was still uncertain whether the condition might not be hysterical. The contracture relaxed more slowly than would have been expected in hysteria, this seeming at first to confirm the alternative diagnosis, but as a result of the treatment the muscles relaxed to a great extent and all movements became possible. During the next few days treatment by suggestion and re-education was continued, with the result that ten days later he was able to use his hand for all ordinary purposes, but a very slight degree of contracture of the fingers persisted, which was possibly due to secondary fibrous changes. Simultaneously with the disappearance of the contracture and paralysis, the œdema, sweating, and vasomotor disturbances completely disappeared. It is clear, therefore, that they were secondary, and resulted from the disturbance in circulation due to the absence of the normal movements of the hand, together perhaps with obstruction of the veins by the contracted muscles. The sweating of the palm

might be due to the warmth and saturation with moisture of the air within the clenched hand.

Other cases of contracture, in which all the signs described by Babinski and Froment were present, have occurred in the absence of any wound, so that a reflex origin was excluded, their hysterical nature being subsequently proved by the cure which followed psychotherapy.

Whilst it is doubtful whether reflex neuroses are as common as one would conclude from the current French literature, I have no doubt that they do exist. The following is the most convincing case I have seen.

Reflex Atrophy and Paralysis of the whole Leg following a Wound of the Foot.—A man received a slight shrapnel wound in his heel. Before this had healed completely all the muscles of the leg and thigh became steadily weaker and more atrophied, until at the end of two months, when the wound had completely healed, the atrophy was extreme, and the patient was unable to walk on account of the paralysis, which was almost complete in the dorsi-flexors of the foot and the extensors of the knee, and rather less severe in the remaining muscles of the limb. The atrophic paralysis was accompanied by exaggerated knee- and ankle-jerks and increased muscular irritability. The foot was cold, blue, and slightly œdematous. Improvement began to take place after ten months, but fourteen months after the wound was received the limb was still weak and atrophied, though the patient could now walk with the aid of a stick. It seems impossible to offer any other diagnosis for this case than reflex paralysis and atrophy.

There is no doubt that reflex paralysis and contracture are not uncommon immediately after a wound is inflicted, the reflex being protective in nature. When the symptoms persist, this may be the result of auto-suggestion ; they are then primarily reflex and subsequently hysterical.

Organic disease may be simulated by the exaggerated tendon reflexes, the atrophy and occasional hypotonus, the alteration in electrical and mechanical excitability, and the presence of vaso-motor and trophic symptoms. But the changes are generally easily distinguished from those occurring after an injury to a peripheral nerve, as the paralysis and anæsthesia do not follow the distribution of any nerve, muscular atrophy is never so extreme, the tendon reflexes are exaggerated instead of lost, and instead of the reaction to degeneration the muscles may show exaggerated

excitability to electrical stimulation. An upper neurone monoplegia generally affects a whole limb instead of a small segment, which is commonly affected in reflex conditions; initial flaccid paralysis is followed by spastic paralysis, instead of flaccidity and spasticity being irregularly distributed in time and being often co-existent in different muscles: the posture is characteristic, and when the leg is affected, the gait is quite different and an extensor plantar reflex is present.

Localised tetanus results in much greater hardness of the affected muscles, which are subject to very painful spasmodic contractions in addition to the constant contraction, and attempts to overcome it cause great pain. I am indeed inclined to think that some symptoms diagnosed as reflex by too enthusiastic disciples of Babinski and Froment have been examples of localised tetanus, as the contracture persists to a great extent even under deep anæsthesia and is generally accompanied by exaggerated tendon reflexes and increased muscular excitability.

The etiology of Volkmann's contracture is quite distinct; the affected muscles are of a much more wooden hardness, and their electrical excitability is diminished or lost instead of being normal or exaggerated. Vascular obliteration may give rise to similar symptoms, but the pulse is lost, intermittent claudication and gangrene may occur, and the application of heat has no effect on the pulse, whereas in reflex conditions the diminution in amplitude of the pulse on the affected side disappears.

Pathogenesis.—The paralysis and contracture, which Babinski and Froment regard as reflex, are not vascular in origin, as might be assumed from the cold, discoloured, and often swollen limb and the diminution in the maximum oscillation of the pulse, for the blood-pressure is unaltered and the vasomotor changes often extend a considerable distance above the injury.

It has also been suggested that the muscular atrophy is simply a result of disuse. But it occurs much too rapidly, and there may be no disuse of the muscles at all. Thus great atrophy occurs in the muscles of the forearm in cases of rheumatoid arthritis, in which the patient may never cease to use the limb. On the other hand, little or no atrophy need occur in complete hysterical paralysis, which has lasted for months or years. The muscular atrophy around a tuberculous joint varies with the condition of

the joint and not with the amount of disuse, as the muscles may actually grow when the disease has been cured, although they are still completely at rest; and extreme atrophy occurs if a wrong diagnosis has been made and the patient is made to take exercise.

The possibility of an ascending neuritis is ruled out by the anatomical distribution of the symptoms, the exaggeration of the deep reflexes, and the absence of the reaction of degeneration; moreover no anatomical evidence has been offered in support of this explanation.

Vulpian in 1886 was the first to suggest that reflex atrophy and spasm could occur; his views were accepted and his observations confirmed by Charcot. All the symptoms can be explained by this theory. The increased tendon reflexes are due to a direct reflex; the vasomotor symptoms are due to reflex spasm of the vessels and not to vasomotor paralysis, as the operation of stripping a muscle of its sympathetic fibres, which has been performed for the pain in causalgia, produces vasodilatation, a rise of temperature in the limb, and a rise of blood-pressure of 20 to 40 mm. of mercury. whereas in the reflex conditions there is vasoconstriction, a fall of temperature, and no change in blood-pressure. A reflex explains the spread of vasomotor and other symptoms far above the injury, and even to the opposite limb; thus in addition to the local effects caused by an injury of the hand, symptoms involving the muscles of the shoulder and a slighter degree of vascular changes and excessive sweating of the opposite hand may occur.

The exaggerated muscular irritability to mechanical excitability and the slow muscular contraction which occurs are not due to direct reflexes, but are secondary to the vasomotor changes, as they are most marked in cold weather, when the limb is coldest, and disappear if it is warmed and the blood supply is increased. When a leg is affected, the plantar reflex may be lost, but this also reappears on warming the foot. The muscular atrophy may be due to the effect of afferent impulses from the injured or diseased parts on the nerve centres in the spinal cord, but possibly it is also in part secondary to the vasomotor changes.

Treatment.—Suggestion is quite useless in the treatment of reflex paralysis and contracture. They improve very slowly if untreated, but continuous extension and methodical and gentle mobilisation, together with whirlpool baths, heat, and especially

diathermy, hasten recovery. Forcible manipulation of contractures under anæsthesia and tenotomies only do harm, but the excision of cicatrices with liberation from the subjacent tissue is sometimes useful.

(v) Hysterical Fits

Etiology.—Hysterical fits are much more common in soldiers than might have been expected from their rarity among men in civil life.

The idea of convulsions may be already present in the man's mind, if he has previously suffered from true epilepsy or if he has been the witness of convulsions in some near relation; horror or fright may then suggest an attack. Such a history may arouse the suspicion that the fits are genuinely epileptic and not the result of suggestion, as strong emotions may be the exciting cause of a fit in epileptics. But if a description of the fit is obtainable, or if subsequent spontaneous or induced attacks are witnessed, their hysterical nature becomes clear.

Hysterical Fits in a Man who had had Convulsions as a Child.—A New Zealand soldier was rendered unconscious for a few minutes as a result of concussion due to a high-explosive shell. He had had a few fits after falling on his head when eight years old, and the recollection of this probably led by a process of auto-suggestion to the occurrence of hysterical convulsions at least once and generally several times a day. He was subsequently hypnotised and recovery was suggested, with the result that the fits immediately ceased and have not recurred.

Hysterical Fits following Horror, and suggested by seeing a Man with Convulsions.—An officer and his servant were blown up by a shell. The servant did not lose consciousness and ran to fetch a stretcher. On his return, the officer, to whom he was greatly attached, made a few convulsive movements and then died. He immediately had a fit, and in the following two months he had eleven more. They ceased completely after their origin and nature had been explained to him.

In rare cases there is no apparent reason why convulsions rather than some other hysterical symptom should develop.

Hysterical Fits reproduced and cured by Hypnotic Suggestion.—Pte. R., aged 27, a typical "martial misfit," who had been a professional singer and subsequently a valet in private life, joined the Army in July, 1915. There was no family history of epilepsy, and

he had never had a fit himself. Soon afterwards he was sent to France, where he worked in a canteen. A week later some men broke in at night, and a mallet was thrown at him; although he was not hit he immediately had a fit and remained dazed, dumb, and unable to walk for two days. After this he slept badly, had occasional fits, and remained very nervous. He was sent home for "debility" in September, 1916, and was discharged to duty in December. He had six fits in the first week after returning to France, three in hospital there, two on the boat, and between two and four on each of the first four days after admission. He had never passed urine or bitten his tongue in a fit, but the description sounded like true epilepsy, and this was the diagnosis made by a M.O. in France, who had seen him have one. As at that time I had not seen a single case of epilepsy resulting from the war without actual injury or a personal or family history of the disease, I doubted the diagnosis. I therefore hypnotised him, and suggested he would have a fit; he at once had one, and though very similar to true epilepsy, the plantar reflexes remained flexor instead of extensor. I told him this would be his last fit, and he had no more except on February 16th and 17th, when I talked to him about returning to duty. This is in striking contrast to the absence of any effect with bromide, which he had in France and during the first four days after admission. Soon after admission he developed a gait and speech defect, which were copied from a man with hysterical paraplegia and another recovering from hysterical mutism in the same ward. These symptoms were genuinely due to auto-suggestion and not malingering, and disappeared with vigorous persuasion.

Diagnosis.—The diagnosis of hysterical from true epileptic fits has generally to be made from the patient's own account of his attacks. A history of involuntary micturition and much less frequently involuntary defæcation, or of injury to the tongue caused by biting, are the only definite points suggesting true epilepsy which can be obtained from the patient himself, and these are inconclusive except when a visible injury to the tongue is present. A man rarely if ever has an hysterical fit when he is by himself. If his companions say that he has struggled with them or clutched at objects near him, the fits are certainly hysterical, but in many cases it is impossible to communicate with those who have seen him in a fit. Even when the medical officer is fortunate enough to be present during one, it is much less easy to come to a definite conclusion than is generally supposed. I have seen

several cases, in which a diagnosis of true epilepsy made under these circumstances before admission, has been proved by subsequent events to be erroneous, and on a number of occasions I have myself been doubtful, although I was present during the greater part of the fit. The only signs which can be regarded as conclusive evidence against hysteria are definite cyanosis, complete loss of conjunctival and corneal reflexes, loss of the pupillar reaction to light, and, most conclusive of all, an extensor plantar reflex; the plantar reflex should be tested in every case as soon as the convulsions have ceased and before consciousness has returned. Unfortunately a flexor reflex does not definitely exclude true epilepsy, though an extensor reflex is much more commonly present. In all cases of doubt, when there is no personal or family history of epilepsy, and when one has to rely entirely on the patient's unsupported statements, I have tried to induce an attack by suggestion under hypnosis. In hysteria a fit is generally produced at once; its characteristics can be studied at leisure, and it can be made to last as long as desired. I do not think that true epilepsy could be induced in this way, and I have certainly seen no case in which a fit produced by suggestion was not definitely hysterical and in which the plantar reflex was not flexor.

In rare cases soldiers sham fits. As the convulsions, like those of hysteria, have simply the character which the individual himself regards as most natural, fits due to malingering and hysteria are clinically quite indistinguishable. It is therefore only the special circumstances of the case which may lead to a suspicion of malingering, and definite proof is not obtained except in rare instances, such as in the following case, in which the man admitted he was shamming.

Malingering of Epilepsy by an Unwilling Conscript.—Pte. C., an unwilling conscript, had numerous fits for the first time in his life on board ship coming from Jersey three days after being called up, and had fifty more in the two days he was in hospital at Southampton. He was then sent to Netley. As the history pointed to the fits being either hysterical or more probably due to malingering, I hypnotised him and suggested a fit. Nothing happened. I then said to the Sister in the patient's hearing that it was quite clear he was shamming, as in all genuine cases a fit would result from this treatment. He immediately had a fit with

marked opisthotonos, which was abruptly terminated when I ordered him to stop and wake up (Fig. II, 10). I then told him I was sure he was malingering, and that he would be wise to admit it; he did so at once, and promised that he would not have any more.

Treatment.—I have already described the manner in which hysterical fits can be diagnosed from true epilepsy by inducing one by suggestion under hypnosis, whenever the patient's history is unreliable and no fits have been observed by the physician himself. I always combine this method of diagnosis with treatment by telling the patient before, during, and after the fit that this one will be the last he will ever have. The treatment has proved most successful, as several men, who had been having numerous fits every day in spite of large doses of bromide, have had no more after their hysterical nature had been proved and their repetition contra-suggested in this way, though the bromide was discontinued. On one occasion I wished to demonstrate the production of an hysterical fit in a man, who had already been told under hypnosis that he would have no more. Although the first fit had been produced with the greatest ease, the suggestion of cure had been so successful that all attempts to produce fits during hypnosis now failed completely. I was unable to produce a fit by suggestion in another patient, but the hysterical nature of his convulsions was proved by the fact that they completely ceased after the suggestion of cure which I had made during hypnosis.

CHAPTER VII

DISORDERS OF SPEECH

(a) Hysterical Mutism.—Etiology.—To have one's breath "taken away by surprise" and "to be speechless with rage" express the familiar effects of emotions on respiration and on speech. It is therefore not surprising that loss of power of speech is a comparatively common symptom in soldiers, who have become abnormally suggestible as a result of the stress and strain of active service. Dumbness may be the direct sequel of a particularly horrible event, as in the following case.

Hysterical Mutism following Horror.—A soldier, aged 17, was sleeping in a hut which was blown to pieces by the explosion of a shell. The noise woke him up, and he found that three of his companions had been killed and five wounded. He did not lose consciousness, but ran to a dug-out "speechless with horror." He remained dumb for a week, when speech was restored by suggestion under hypnosis. He continued to stammer for another week, but quickly recovered with re-education and was well enough to return to duty a month after the explosion.

In other cases mutism follows true shell-shock; a man in a half-conscious condition, whilst recovering from the immediate effects of being blown up by a high-explosive shell, is unable to speak owing to the depression of all his faculties following the concussion. He becomes obsessed with the idea that the speechlessness will be permanent, the mutism being thus maintained by a process of suggestion.

Hysterical Mutism following Shell-Shock.—Pte. C., aged 24, had been in France for eleven months and had got on well until he became very frightened during a heavy bombardment which lasted for two days; he was then blown up, and the first impression which reached his mind as he regained consciousness was extreme thirst. He found he could make no sound when he tried to call for water: the inability to speak came as a shock to his mind, and the fact

that all the other functions of his body were also more or less in abeyance remained unnoticed. With his mind fixed on his dumbness, it was impossible for speech to return when his other faculties came back. Strong counter-suggestion overcame the fixed idea of mutism and recovery took place.

Sometimes the idea of mutism dates from a few moments preceding the explosion.

Hysterical Mutism following Shell-Shock.—A dumb private admitted under my care on March 3rd, 1917, wrote the following account of himself :—

" One day, early last October, I heard a shell coming and I tried to dodge ; I opened my mouth to shout, but could not, and I then seemed to be blown up. I remembered no more till I was being carried on a stretcher when I tried to shout and could not, and I could not hear. I heard again on the train. I was in several hospitals and was kept in bed and reading was forbidden for fourteen days, then high frequency daily, sparks from my throat, and I was given chloroform, but with no effect."

I told him that he would be cured quite certainly with the treatment he would have the next day, and a man, who had been rapidly cured after being dumb for a year, was instructed to tell him about his recovery. The next morning he was convinced he was going to get well. I passed a catheter down his larynx, and after a few seconds of vigorous persuasion he whispered. In five minutes he could phonate, but still stammered, and the next day he spoke normally. There was no relapse, and he returned to duty three weeks later.

Mutism is occasionally a sequel of injuries due to other causes, particularly if they are associated with much shock. In some cases there is a history of a blow on the chest or of partial asphyxiation due to burial at the time of an explosion.

Mutism is so rare in civil life and so common in soldiers that a history of mutism in the past, as in the following case, is rarely obtained, though not infrequently the patient has previously stammered.

Hysterial Mutism in a Man who had previously been Mute.—Pte. B., aged 23, had always stammered slightly. Some years ago he lost his speech completely for a day as a result of an electric shock. On December 2nd, 1916, he was guarding some stores, when he discovered a soldier stealing biscuits, a tin of which he threw at him. Though not hit, he fell down and remained dazed for ten days. When he came to himself he was completely mute, though

he was otherwise well and could reply sensibly in writing. On December 20th, the power of speech suddenly returned; at first he stammered, but before long he was talking almost normally. The mutism which had followed a severe shock on a previous occasion probably recurred to his mind as he was recovering consciousness and suggested mutism to him.

Symptoms.—In addition to being unable to speak, the patient cannot whisper, and instead of the isolated paresis of the abductors characteristic of hysterical aphonia, laryngeal examination shows that the vocal cords do not move at all. We have never found pharyngeal anæsthesia either in hysterical mutism or aphonia, the supposed association being apparently due to suggestion of anæsthesia on the part of the observer. In the severer cases the patient cannot cough, whistle, or make any sound when he laughs. He may be unable to expire with sufficient power to blow out a candle, and in rare cases he cannot put out his tongue, but he is always able to move it in the act of chewing. Most mute patients keep the mouth closed and apparently make no effort to speak. In other cases they move the lips as if speaking, but do not make the slightest sound.

Dumbness is often associated with deafness, but recovery from the latter or less frequently from the former may rapidly occur, so that the other condition remains alone. At first the patient is frequently in a very emotional state, and in severe cases may be in a constant state of terror, which is prevented from disappearing by frequent nightmares and constantly recurring mental pictures of the horrors he has passed through.

He is generally able to convey his thoughts without anydifficulty by writing, but in the following case drawing took the place of writing.

Mutism with Ability to Draw, but Inability to Write.—Pte. A., aged 22, was admitted under my care on December 27th, 1916. He could neither hear nor speak. When asked questions in writing about himself, he made drawings of trenches, and at the same time became very excited. Suddenly pointing to a prostrate figure in the drawing, he shouted "Bunchy Turner," which proved to be the name of a chum who was killed in the attack. With vigorous encouragement he was induced to name various objects, and in a few minutes he could both hear and talk quite normally. For some days he had a headache, but this finally disappeared, and he

was sent back to duty at the end of January physically and mentally fit.

After his recovery he was able to give the following history, which was amplified from letters received from the general hospital, in which he was under observation in France. He had been in the trenches since January, 1916, but had never got used to the shelling, and an effort was required to hide his fear. He continually dreamt of the dead and had a horror of corpses. In October, 1916, he saw some comrades killed by the collapse of a dug-out ; he was so stupefied with terror that he could not move to help. He remembers taking part in an attack and reaching the third German line of trenches on November 13th ; from this moment his mind is a blank until the moment when his speech returned, and he has no recollection of making any drawings. At the General Hospital in France, in which he was under observation for a month, he was unable to hear or speak and behaved like a little child ; he was as delighted as a boy of six when given some sweets and wept if he was refused anything he wanted. Though he could read a few familiar words he could not write, and occupied much of his time in making clever copies of pictures.

This is the only case of deaf mutism I have seen in which it was impossible to converse with the patient by writing. His love of drawing whilst the condition of confusion lasted and the part played by a drawing in his recovery are particularly remarkable, as he had never been interested in drawing before he lost his speech and hearing, and he has not drawn at all since his recovery.

Diagnosis of Mutism from Aphasia.—True mutism is always hysterical ; true aphasia is always organic. In rare cases speech may be so limited in aphasia that the conditions may simulate mutism, but I have much more frequently seen cases of hysterical mutism wrongly diagnosed as aphasia. The mute can make no sound ; the aphasic can make articulate sounds without difficulty, certain words are often reiterated, and in many cases he can make almost every sound used in ordinary speech. A patient who is completely dumb when awake may call out in his dreams ; such an occurrence would be impossible in aphasia. With very few exceptions mutes can express themselves perfectly in writing, but writing is always more or less affected in patients suffering from

motor aphasia. Mutes can understand what they read and what is said to them; in aphasia this may not be the case. Lastly, as Charcot pointed out long ago, the mute contrasts with the aphasic in being remarkably clever in explaining himself by signs. A soldier who cannot express himself by speech, but can explain by gestures how he became dumb, is certainly suffering from hysterical mutism and not organic aphasia.

Treatment.—Complete mutism often lasts only a few days, after which it may disappear entirely or give place to some form of stammering or less frequently to aphonia. Recovery may be spontaneous and occur suddenly or gradually without treatment or as a result of encouragement, but more often speech returns suddenly as a result of direct suggestion, which overcomes the fixed idea of mutism, or of sudden emotion, which breaks down the subconscious inhibition of speech by surprising it, when the patient is off his guard with his mind for the moment not fixed on his dumbness. Thus speech may return as a result of crying out when in pain or in a moment of excitement, as when watching a boxing or football match or during a Zeppelin raid, or a mute may suddenly find himself joining in a familiar chorus. One man recovered after being given a cold douche just as he was getting out of a hot bath. Speech may also return after a nightmare in which the patient calls out, though more often this is not followed by recovery. A man was admitted with complete mutism; the next morning he handed me the following letter, written by himself. "Sir, I *am* surprised. I am getting my speech back. I dreamt last night I was falling down a terrible big hill, down amongst rocks, and woke up able to talk. I am so glad that I am soon able to go back to my unit. I am so glad. I would rather than sixty pounds to get such a fright, thank God, Sir, I am, S. P. G——. 200334." A few days later he returned to duty. I saw a man at Cliveden, who was sleeping when his parents came to visit him, a fortnight after he had become dumb as a result of shell-shock. They awakened him and he at once said, "Hullo, mother!" One of my patients recovered on the day of admission after an hysterical fit. Several cases have been recorded of recovery under the influence of alcohol. Buzzard ingeniously suggested that the causes to which patients attribute their sudden recovery are often incidents, which they choose as convenient

for the purpose, when they have discovered that speech is possible and realise how inconvenient it is to be dumb, but I do not think that this is often the case.

When a mute has been told that he will recover so convincingly that he himself feels no doubt about it, it is immaterial what form of treatment is applied, so long as it has not already been tried unsuccessfully. Whatever method is used it should be accompanied by vigorous persuasion. As the patient often whispers at first, the treatment should be continued until the aphonia also disappears, and an attempt should also be made to cure the stammer which follows in many long-standing cases.

In most cases the introduction of an intra-laryngeal electrode is sufficient, but it should previously be connected with a faradic battery in case speech does not return within a few seconds, as the more powerful suggestion caused by the pain and contraction of the muscles of the throat will then succeed. In cases in which electricity has already been used elsewhere, but has failed owing to being unaccompanied by sufficiently vigorous suggestion, a repetition of the treatment with vigorous suggestion has sometimes met with immediate success, as in one case in which the dumbness had persisted for five months. But in such cases I prefer to give the patient ether so rapidly that he quickly becomes very excited, when he almost always talks spontaneously, but an electrode may be introduced into the larynx if there is any delay. He is then made to talk continuously until he has completely recovered from the anæsthetic. In two cases the recovery from deaf-mutism which followed this treatment was accompanied by partial amnesia.

Hysterical Deaf-Mutism following Shell-Shock : cured by Suggestion with Etherisation and followed by Partial Amnesia.— An Australian soldier, aged 22, wrote the following letter to his relations on August 21st, 1916. " You may be a little surprised to hear that I am in the Hos. suffering from shell-shock, which has taken away my speech and hearing. It is some sixteen days now since it happened. . . . We were in the trenches and going for dear life, when two of us spotted a German machine gunner in a hole, so we made up our minds to have him. We made a charge at him, and I just remember getting to him when a high-explosive shell burst at my head ; it seemed as if it burst inside my head ; everything went black. I tried

to call out and couldn't, and I could not hear my mates—only just a terrible bursting in my head all the time. I never remembered anything more until I came to on the boat. . . . The Drs. have told me that I will get alright in time. . . . I saw a good deal of France. . . . There is not a young man there who is not in the Army. The girls and women work in the fie—— "

The letter ended abruptly at this point, as I then came to examine him. The previous day I had hypnotised him without difficulty, but was unable to make any effective suggestions, as the deafness persisted during the hypnotic sleep, so that the suggestions did not reach the higher centres of his brain, and were consequently not acted upon either whilst he remained asleep or after he awoke. He was so deaf that he heard nothing at all during an exceptionally violent thunderstorm. He was not only unable to speak, but could make no sound of any kind and could not cough.

As no improvement had taken place, he was given ether after being told in writing that it would have the effect of restoring his speech and hearing. He began to struggle after the first few whiffs, and long before he was anæsthetised he began to repeat the word " Mother," first in a whisper, then louder and louder until he shouted it with a stentorian voice that would have filled the Albert Hall. The etherisation was then discontinued, his limbs never having become relaxed. As he came round, I told him to say various words which he repeated after me, and I then carried on a continuous conversation with him. When the effects of the anæsthetic finally passed away, he was talking with a normal voice and he had completely recovered his hearing.

His memory, which had previously been unaffected, was now a complete blank from a short time before he was blown up to the moment he had regained consciousness. He had no recollection of having lost his speech or hearing; he was astonished to see the letter he had begun, as he remembered none of the events described in it, and he did not remember having seen me before. In all other respects his memory was perfect.

In this case the patient became speechless from fright at the sound of the explosion, and deaf from the accompanying noise; almost immediately afterwards he lost consciousness as a result of the aerial concussion. The moment he recovered consciousness the fact that he had lost his power of speech and hearing after the

explosion recurred to his mind, and as a result of auto-suggestion these disabilities were perpetuated. The subconscious inhibition of speech and hearing was broken down as a result of the loss of control of the higher over the lower cerebral centres when he was under the influence of ether. By keeping these faculties continuously at work whilst the effects of the ether passed off, their recovery was maintained when consciousness returned.

Amnesia following Recovery from Deaf-Mutism.—Sergt. H., a New Zealander, was admitted under my care on September 25th, 1916. He was completely deaf and dumb, and wrote the following history of his case. "About 17th September, our brigade was in support behind F——. A shell landed on our cookhouse, killing one and wounding two others, and as the Germans were bombarding very casually (about one shell a minute) I thought there was time to get those fellows in. I had just got to them—the cookhouse was 50 yards in rear of the trenches in the open—when the enemy opened battery fire and violently bombarded the sector with large shells. My man, whose both legs were broken, made progress very slow, and another shell landed within six feet of us, a piece of it hitting me in the back. I remember getting to the dressing station somehow or other, and next found myself in E—— four or five days after. I could not walk, talk, or hear. I have all other faculties and can understand and think clearly. I am just beginning to walk a few steps at a time. The M.O. at the hospital in France told me it was only a matter of time and I would be all right."

On October 1st, as there was still no return of hearing or speech, he was given a little ether, and after a good deal of persuasion his speech and hearing were restored. When he regained consciousness he could hear and speak perfectly, but he believed that it was May 25th, 1915, and thought the "boys were kidding him" when they told him he was in England, as he had no recollection of having left New Zealand and imagined he must be in the Porarua lunatic asylum. The following is an extract from an account he wrote of his doings on what he believed to be the previous day. "On May 24th, I went to church for my organ lesson at 8 p.m., and afterwards had the treat of my life. Mr. F. played the pick of musical compositions until 11 p.m. That night will ever stand out as one of the most enjoyable of my life. . . . On Tuesday night I went to Mr. B.'s house to bid farewell to Wilf. and Rol., who are going to the war. I don't think things are so bad as to warrant my throwing up my billet, as yet, and I told them so. Besides, I would miss that examination. Of course, if the worst comes to the worst I will go to the front."

His general health rapidly improved, and in a few days, with encouragement and re-education, he learnt to walk quite well. But the blank in his memory persisted, and at the beginning of December, when he returned to duty, he was still unable to remember anything between May 25th, 1915, and Oct. 1st, 1916, which appear to him to be consecutive days, and he had to learn his drill again, as he had no recollection of his military training.

(b) Hysterical Aphonia.—Etiology.—Hysterical aphonia is very rarely a primary result of severe emotional strain or shell-shock, but it frequently occurs when hysterical mutism disappears spontaneously or as a result of treatment. In the former case it may persist for long periods, but in the latter it ought not to be allowed to last more than a few minutes. The majority of cases of hysterical aphonia which I have seen in soldiers, however, followed laryngitis, the origin being identical with the aphonia seen in civil practice. The laryngitis may be caused by straining the voice, but it is more commonly a result of infection, in which case it may be present alone, or it may be only one symptom of influenza or a widespread catarrh affecting the nose, pharynx, larynx, and bronchi. In several cases it followed gassing. Owing to the discomfort caused by speaking when the larynx is inflamed, the patient whispers. The inflammation disappears after a time, but the patient has meanwhile become so impressed with his whispering that he regards it as the only important part of his illness and forgets the cough and other symptoms which he had at first. The result is that he continues to whisper and now finds that he is quite unable to phonate. This type of aphonia is so common in soldiers that the stress and strain of active service must predispose by rendering a man abnormally suggestible. A man who has previously had hysterical aphonia either before or since he joined the Army may suffer from recurrences whenever he is overfatigued, even in the absence of laryngitis. This was the case in a colonel, who consulted me on account of aphonia which had recurred for the fourth time shortly after he had taken over a very responsible post, which involved constant overwork.

Treatment.—Hysterical aphonia generally recovers after the application of a sound or in more severe cases of faradism to the interior of the larynx. If any organic abnormality, such as laryngitis, is present, this should subsequently be treated, or a

relapse is likely to occur. It is, however, a great mistake to give such treatment alone, as I have seen numerous cases in which the aphonia persisted owing to the local treatment emphasising the abnormal condition of his larynx in the patient's mind. In each of these cases immediate recovery followed vigorous suggestion with an intralaryngeal electrode, the intralaryngeal medication being at the same time discontinued. The patient should be told beforehand that the treatment is certain to cure him; if the treatment is given without any explanation it is likely to do harm by aggravating his emotional condition. One day in July, 1917, I saw seven cases of hysterical aphonia, which had been treated by electricity for periods varying between five and eighteen months without success. On passing a catheter into the larynx and at the same time promising a cure, five of the seven recovered completely in twenty minutes, and the remaining two were cured a few days later by repetition of the treatment with more vigorous persuasion after being transferred to Netley. I recently saw a man who had been aphonic for almost a year in spite of treatment by persuasion, electricity, and general anæsthesia. Vigorous suggestion during hypnosis with the aid of intralaryngeal electricity when simple hypnotic suggestion had failed, produced sudden and complete recovery. A man, who had a deep talking voice but a falsetto singing voice, became aphonic after an operation for hernia. I first saw him when the aphonia had been present for sixteen months. Vigorous suggestion with an intralaryngeal electrode brought the voice back the same day, but it was at first falsetto, and only became normal some days later after further treatment by suggestion with ether and re-education.

(c) Hysterical Stammering. — Etiology. — Hysterical stammering may follow severe emotions at the front or actual shell-shock. It is sometimes the primary condition, especially if the patient had stammered as a child. More frequently it is a sequel of mutism, as the majority of cases which recover spontaneously or as a result of treatment stammer, unless the mutism has only been present for a few days. The longer the mutism has lasted, the more severe and resistant to treatment is the stammering likely to be.

Symptoms.—Many different forms of stammering occur in soldiers. There may only be difficulty in commencing speech;

as soon as the first word has been pronounced the rest of the sentence follows easily. More frequently only a few words follow fluently and the patient has to wait again before he can produce a further instalment. One man took two or three minutes to begin a sentence, but if, as a result of a great effort, he could say "one," he could then count quickly and intersperse answers to questions as to his name or regiment without a pause in his counting. In other cases speech is extremely slow, deliberate, and drawling, without any actual stammer. An officer with this condition was completely cured when I sent him to a quiet spot where he recited and made speeches in the absence of any audience. Very often a syllable or a vowel sound in every word is repeated several times. In other cases the stammerer occupies the time during which he is attempting to begin a sentence by repeating "er-er-er," as in the speech of a nervous man at his first public meeting. A sergeant who had been blown up by a high-explosive shell continued to have ghastly nightmares, and remained exceedingly nervous for many weeks. He trembled all over his body whenever anything was said to him, and he could only say three words at a time, each group of three words being invariably preceded by the repetition of "er-er-er" from twelve to fifteen times. This continued without alteration for many weeks, but slow improvement occurred with hypnotism. Stammering is often associated with spasmodic movements of the face whenever an attempt is made to speak, and in one case these spasmodic movements after a time occurred independently of speech and persisted for a few days after the stammering was cured. Rapid rhythmical side-to-side movements of the head and shrugging movements of the shoulders also frequently accompany the effort to speak.

Treatment.—If stammering is treated the moment it develops in the course of recovery from mutism, it can often be quickly cured. Suggestion with the aid of electricity, etherisation, or suggestion under hypnosis, as in the following case, occasionally produces immediate recovery.

Stammering cured by Suggestion under Hypnosis.—Driver D., aged 31, was run over by a loaded transport waggon at Gallipoli in May, 1915. There was no external injury, but his pelvis was fractured. For three days he was unable to speak at all, although

he was perfectly conscious. He then slowly improved, but when he was admitted into the New Zealand War Hospital at Walton at the beginning of August, he still spoke with extreme difficulty, and the effort was accompanied by contortions of the whole of his face. Similar contortions of his face also occurred apart from any attempt to speak; they were accompanied by the mental condition characteristic of tics; the patient was able to control them by an effort of will, but he felt miserable when he did so, and was always ultimately forced to give way to the irresistible impulse.

He was readily hypnotised, and it was suggested to him that he would be able to speak without difficulty, and that the contortions would cease. The result was very satisfactory, for as soon as he came round he was able to talk quite normally; the next evening he sang at a concert, and a few days later took part in a play. The facial contortions occurred during the hypnotic sleep and continued afterwards, though less severely. They disappeared after he was hypnotised a second time.

Much more frequently improvement occurs slowly as a result of re-education on the general lines laid down by Mr. Courtlandt MacMahon. Lessons in breathing and talking are given regularly every day. Officers and the more intelligent men often learn in this way to talk almost normally in the course of a single lesson, at the beginning of which they could hardly utter a word. Skill in the re-education of stammerers cannot be acquired without long practise. I have only obtained really good results since my wife has devoted every morning to teaching the numerous stammerers in my neurological section at Netley. The patient is told to take slow and deep breaths; owing to a kind of respiratory tic this may at first be very difficult. He is then told to repeat first a single word and then short sentences during expiration. He must avoid the tendency to hold his breath when he begins to speak. If he has not completed what he had to say during one expiration, he must wait for another and not attempt to continue during inspiration. After some improvement has occurred, he practises reading and ordinary conversation, observing the same rules concerning the regulation of his breathing. Stutterers are never allowed to pass on to another sentence until they have said what they have to say without repeating a syllable or a word, and without introducing the sentence or breaking into it with "er-er-er."

A somewhat different method, which we learnt from Captain W. Johnson, should be adopted for the severe cases, in which a stammerer is almost mute owing to the extreme difficulty he experiences in starting each sentence. He is told to take rapid short breaths as if he had been running quickly ; he then makes a noise with each expiration, after which he repeats a syllable, such as " he-he-he," with each short breath. He next says the alphabet in the same way, one letter with each expiration. By the end of the first lesson he should be able to carry on a conversation. In a very short time, having gained confidence in this way, he learns to talk naturally.

CHAPTER VIII

DISORDERS OF HEARING

Hysterical Deafness—Etiology.—The noise and concussion produced by the explosion of a shell of high power in the near neighbourhood frequently causes deafness. The patient is dazed or unconscious as a result of the explosion, and when his mind becomes clear again he discovers that he cannot hear. Both ears may be affected, but the one on the side more exposed to the explosion of the shell is often deafer than the other. The initial deafness is doubtless due to concussion of the internal ear, and possibly also of the auditory centres. It may pass off in the course of a few hours, but more frequently it lasts for a few days. If it persists for a still longer period, it is almost always hysterical. It is often associated with other symptoms, such as headache and insomnia, and it is particularly likely to be accompanied by dumbness. In one of my cases hysterical deafness and blindness were present together. The initial concussion deafness makes such an impression on the mind of a soldier that on coming to himself, whether he has actually lost consciousness or not, his first thought is for his hearing, and he may be so convinced that he is permanently deafened that he becomes actually deaf as a result of auto-suggestion. The temporary deafness which occurs in gunners from the effect of the constant repetition of very loud noises may in the same way become perpetuated and exaggerated by auto-suggestion. Lastly organic deafness, especially if the onset is acute, as in that due to involvement of the auditory nerve trunk in cerebro-spinal meningitis, may remain complete after the disappearance of the active inflammation has been followed by restoration of some of the nerve-fibres, so that a certain amount of hearing should have returned. This again is due to auto-suggestion, the final deafness being organic with a superadded

hysterical element, which is capable of removal, like all hysterical symptoms, by suggestion. A large proportion of cases have occurred in men who had old disease of the ear.

Hysterical Deafness following Gun-Deafness in a Man with Chronic Otitis Media.—Pte. F., aged 18, says he has been slightly deaf all his life. He became gradually deafer after gun-practice whilst training in England, until he could not hear at all, although he had never suffered from any definite concussion, and had not served abroad. He was totally deaf to air and bone conduction, and it was impossible to wake him by the loudest noises when he was asleep. The deafness also persisted during deep hypnosis, so that he could not be made to respond to suggestion. The normal nystagmus and giddiness followed rotation, and he had a brisk auditory motor reflex both when awake and during sleep, but it was obvious that this was not associated with hearing. It was clear, therefore, that the deafness was hysterical.

Ordinary persuasion, suggestion, and hypnotism having failed to produce any improvement after five months, he was advised to undergo an operation, which he was told would certainly cure him. He was given sufficient ether to make him sleep, and an incision was then made behind his right ear, a plate of iron being continuously hammered at the same time. He jumped from the table in a half-drunken condition, hearing quite well, and said he must have had a dream as he thought he had been deaf. The next day he said that he could hear better than he had ever done before. He was discharged to duty ten days later, and soon afterwards was well enough to apply for a commission.

Examination of his ears a week after the operation showed that he could only hear a 30-inch watch at 20 inches; the upper and lower limits of hearing were normal. Both drums were indrawn and showed evidence of chronic catarrh, which was doubtless the cause of his long-standing deafness. He was thus predisposed by chronic ear disease to gun-deafness, and the temporary deafness produced during gun practice was perpetuated and exaggerated by auto-suggestion. The true gun-deafness had disappeared spontaneously, so that when the total deafness resulting from suggestion was cured by suggestion in the form of an operation, his hearing returned to its original state.

One or both drums are sometimes perforated owing to the sudden enormous change in atmospheric pressure; the tear eventually heals with complete restoration of hearing, unless the patient at the moment of regaining consciousness, when he realises that his hearing is impaired, becomes convinced that he will never

hear again, and converts by auto-suggestion comparatively slight deafness of one or both ears into complete and bilateral deafness.

Diagnosis.—Captain E. A. Peters and I have found it necessary to discard almost all the criteria formerly used in the diagnosis of organic deafness from deafness due to hysteria or malingering.

(i) *History.*—Complete bilateral deafness following the explosion of a powerful shell is generally hysterical, though a lesser degree of a symmetrical organic deafness of a more or less permanent nature may be produced at the same time, owing to perforation of the drum or hæmorrhage into the middle ear, both of which are often followed by otitis media. It is still doubtful whether absolute organic deafness ever results from hæmorrhage into the internal ear following shell-shock, as no anatomical evidence of such an occurrence has yet been forthcoming. Deafness following an acute illness, especially cerebro-spinal meningitis, is at any rate in part always organic, but the deafness is in our experience frequently increased as a result of auto-suggestion, no functional improvement occurring when the anatomical condition improves.

(ii) *Bone and Air Conduction.*—When deafness is not absolute, a tuning-fork can still be heard by air conduction after it has ceased to be heard by bone conduction (positive Rinne's test). This shows that the deafness does not depend on changes in the middle ear, even when these are present. But it does not distinguish between the nerve deafness due to organic disease and that due to hysteria. Moreover the test can only be applied in the slighter cases, as in many instances deafness is absolute and no hearing is possible, whether the sound is conducted by bone or through the air. The vibration of a tuning-fork held on the mastoid process is, however, often felt, and the aerial vibration caused by very loud noises is occasionally appreciated, even when no sound is heard.

(iii) *Auditory Motor or Jump Reflex* (p. 41).—A sudden noise normally causes an individual to jump and often to blink, and the pupils dilate; the "jump," at any rate, is a protective reflex and represents the preparation for flight or fight. An officer whose left motor cortex had been almost completely destroyed went to "The Man that Stayed at Home" about four months after he

was wounded. His right arm jumped violently when the gun-shot rang out on the stage, although no trace of voluntary movement returned until three months later. The efferent part of the reflex is thus sub-cortical. In certain war neuroses of emotional origin, in which the reflex is exaggerated, jumping continues during sleep and deep hypnosis, although the patient does not hear the noise which induces it, even in a dream. The afferent part of the reflex is thus also sub-cortical. That the reflex is quite independent of actual hearing is shown by the fact that during deep hypnosis with the eyes open, individuals, in whom the reflex is normal or only slightly exaggerated, do not always jump or blink, and the pupils do not dilate even when a poker is banged against a shovel within a foot of the ear, although they answer whispered questions and obey whispered commands, and the cutaneous and tendon reflexes remain unaffected.

Experiments on animals by Sherrington confirm these clinical observations and indicate that the auditory-motor reflex is a function of the mid-brain. A slight reflex was present in most of our cases of hysterical deafness, but it became less marked and sometimes disappeared completely or was confined to a slight dilation of the pupil when the test was repeated. A very nervous but totally deaf mute remained completely unmoved, never jumping or showing a flicker of his eyelid during one of the severest thunderstorms I have known, and yet the next day he was completely cured by suggestion.

It is thus clear that the presence of the auditory-motor reflex does not imply that hearing occurs, and a man who says he can hear nothing, but shows a normal reflex, is not necessarily a malingerer, but may be suffering from genuine hysterical deafness, or even organic deafness due to a bilateral lesion of the cortical auditory centres.

(iv) *Persistence during Sleep.*—As hysterical symptoms are due to suggestion it might be expected that they would not persist during sleep, and Babinski regards this as a definite law. My experience agrees with his with regard to all other hysterical symptoms which I have investigated, such as paralysis, contractures, and anæsthesia, and I have seen several deaf mutes who talked in their sleep, but we have found that hysterical deafness, the behaviour of which during sleep does not appear

to have been tested by Babinski, is an exception. Thus, greatly to our surprise, we found it quite impossible to wake two of our patients, who were suffering from total hysterical deafness, by shouting or by making other very loud noises within a foot of their heads. We convinced ourselves that deception was impossible, and the hysterical nature of the deafness in both cases was at a later date conclusively proved by their instantaneous recovery with powerful suggestion. In one patient a slight twitch of the eyelids was sometimes observed with a particularly loud noise, but not in the other. It seems possible that a malingerer could be detected by this test, as he would certainly wake if a loud noise was made when he was asleep, whereas in hysterical and organic deafness waking does not follow.

(v) *Effect of Hypnosis.*—We had expected that hearing would return in hysterical cases during hypnosis, but we found it quite impossible to make the patients obey any command or show any signs of hearing, and no auditory-motor reflex was produced, although they were deeply hypnotised on several occasions. The unswitched synapses thus appear to remain unswitched during hypnosis as they do during sleep.

(vi) *Character of the Voice.*—In almost all cases of severe deafness due to organic disorders the character of the voice changes. It is difficult to understand why there should be any difference in the effect of total deafness on the voice whether it is organic or hysterical, as the change is simply a result of the patient's inability to hear his own voice. It is, however, a fact that we could not observe any change in the timbre and intonation of the voice in any of our hysterical cases.

(vii) *Lip-Reading.*—When lip-reading is learnt by a patient without being taught the deafness must be of a high degree. It would not occur in malingering, but several patients with hysterical deafness learnt it with remarkable rapidity, so that it cannot be regarded as a sign of organic disease.

(viii) *Vestibular Symptoms and Reactions.*—Disturbances in the vestibule as a result of concussion may cause spontaneous nystagmus, which may be accompanied by giddiness and staggering, but these symptoms rarely last for more than a few hours.

We have found that the only test, upon which complete reliance can be placed in the diagnosis of absolute hysterical deafness from

absolute organic deafness, is the presence of normal vestibular reactions in the former and their loss in the latter. The vestibular reactions are entirely beyond the control of the will, and it is therefore inconceivable that they should disappear as a result of suggestion. As hysterical symptoms are always caused by suggestion, the vestibular reactions must remain unaffected in hysterical deafness. This test has proved of great value in cases of total deafness. But it must be remembered that the partial organic deafness, in which the vestibular reactions are lost in the severer cases and exaggerated in the slighter cases, may be accompanied and exaggerated by the simultaneous presence of hysterical deafness, which it is quite impossible to recognise, except by the improvement which follows treatment by suggestion.

The vestibular reactions may be investigated by the rotation, caloric, or electrical tests, in all of which nystagmus and giddiness occur in normal individuals, but not if the vestibular nerves are damaged. We have employed the rotation test, as it requires no special apparatus, the patient turning rapidly round ten times in one direction and subsequently in the opposite direction; the character and degree of the nystagmus on looking in the direction opposite to the rotation is estimated, and the subjective and objective evidence of vertigo is investigated.

The rapidity of the movements of the eyes and their duration should be the same when the individual is rotated clockwise or counter-clockwise. If an inequality is observed, one vestibule must be involved and the other spared, or one must be involved more than the other.

Hysterical Deafness of one Ear associated with Organic Deafness of the Other.—Pte. W., whose case is referred to in another connection on p. 126, was wounded over the right occipital bone on June 7th, 1917. When he regained consciousness five days later he was found to be totally deaf. As the vestibular reactions were present, the deafness was regarded as hysterical. Other treatment having failed, an " operation " was performed behind the right ear, as a result of which there was considerable improvement in the hearing of the *left* ear, but no improvement in the right. As the suggestive effect of the operation would be likely to influence the right ear more than the left, although in other cases immediate recovery took place in both ears, it would seem

probable that the deafness on the right side was organic and therefore not capable of improvement by suggestion, and that of the left was hysterical and therefore curable by suggestion. This view corresponded with the fact that the wound of the skull extended to within an inch of the right mastoid but did not involve the left side at all, so that the right internal ear might easily have been severely damaged by the concussion. During the next few weeks further improvement occurred in the left side, but no improvement on the right. That the two ears were not affected in the same manner was now proved by a more careful performance of the rotation test: on rotating the patient ten times clockwise the nystagmus lasted for 35 seconds, being at first very rapid and then slow; but rotation in the opposite direction only caused the second part of the reaction observed in the first test, the initial rapid nystagmus being absent and the reaction consisting only of very slow movements which lasted 20 seconds, the average normal duration being 25 seconds. As it would be impossible to influence these reactions by suggestion, it is clear that the difference indicates that the two ears were affected in a different manner, the deafness being either organic in one only, or if organic in both, one being much more affected than the other.

Pathogenesis.—Hearing necessitates listening; inattention during a dull sermon results in total deafness to the sermon. In hysterical deafness the patient is so convinced that he cannot hear that he does not listen. Although the sound vibrations reach the ear in the normal way, they do not give rise to the slightest auditory sensation because of this inattention. The synapses at one or more of the cell-stations in the auditory path to the cerebral cortex must therefore be unswitched, probably as a result of retraction of the dendrons. In absolute hysterical deafness the auditory motor reflex, which is a function of the mid-brain, may be abolished or greatly diminished. One of the unswitched synapses must therefore be below the mid-brain and either in the auditory nucleus or less probably in one of the intermediate cell-stations—the superior olive or the nucleus of the lateral fillet.

The persistence of the deafness during hypnosis and natural sleep shows that when the inattention of hysterical deafness has lasted for a considerable period, the unswitching of the synapses is more profound than that which normally occurs during deep sleep, in which the synapses can always be forced by a loud noise.

When recovery takes place gradually, sounds are generally heard before words can be recognised, and a patient will hear a gramophone before he can recognise the tune. A word may have to be repeated several times before it is understood; even then, as Gordon Wilson has pointed out, there may be considerable delay in answering a question, as the patient hesitates at first to attach a meaning to the word, although he hears it. If asked to repeat a sentence he may only repeat the first or the last word, although he knows that others, which he did not properly grasp, accompanied it. In a case recorded by James Collier, the patient, who was a very intelligent man, was completely mute and appeared to be deaf. In the course of a written conversation he was asked if he heard anything when spoken to. In reply he wrote: "I can hear your voice quite well, sir, but I cannot gather anything from it." He subsequently recovered completely.

Prognosis.—Cases seen at an early stage recover rapidly if they are assured that their condition is not a serious one. On the other hand, if left untreated without any encouragement, the patient will become more and more convinced that he is permanently deaf. One of the worst cases I have seen was that of a man who was told by an aurist that his case was hopeless, as he had already been deaf for four months without any improvement occurring. Prolonged treatment by hypnotic suggestion was required to cure him. If he had been told that the kind of deafness from which he suffered never lasted for more than four months and that he would certainly be well in a week, rapid recovery would have resulted.

Treatment.—Uncomplicated deafness is the most difficult of all hysterical symptoms to treat by the usual methods; mutism is one of the easiest. It is, therefore, fortunate that most cases of hysterical deafness are associated with mutism, as the return of speech in a deaf-mute generally convinces him that he can also hear, especially if he has been told that this will be the case: he listens as soon as he finds he can speak, and the unswitched synapses are once more switched on.

I have only seen four cases of absolute hysterical deafness without mutism, and in one of them mutism was originally present, but spontaneous recovery had occurred some months earlier. All had had various forms of treatment before we saw

them. Simple encouragement and the promise of recovery had proved unavailing, and when we repeated the promise to a man, who was still completely deaf after eight months, he not unnaturally received our promise with considerable scepticism. We tried the effect of making very loud noises of different kinds near the patients' ears in the hope that they might break through the resistance, and that when they were heard re-education with other sounds could be begun. But this method, which is sometimes successful in the partially deaf, was unavailing, as the deafness was absolute, and no noise, however loud, was heard. On many occasions the patients themselves shouted as loudly as they could through the chest-piece of a binaural stethoscope, the ear-piece of which was in their own ears, and at other times the chest-piece was put on an iron plate, which was then hammered, but they heard nothing. Etherisation had been tried without success before we saw them. Various forms of suggestion were tried: one patient had a great belief in electricity, but although we encouraged him to believe it would cure him, it had no effect. Two of the patients were hypnotised on a number of occasions without difficulty, but this proved useless, as the deafness persisted, so that they could not obey any commands such as other hypnotised individuals obey immediately. Suggestions of recovery, which are effective in patients suffering from incomplete hysterical deafness, consequently gave no result. Other loud noises and familiar tunes from a gramophone placed in contact with the ear during hypnosis had no more effect than shouting.

As all treatment had failed, we told the first of our patients, who had been completely deaf since he was blown up ten months earlier, that if he did not get well in the next week, we would be compelled to advise him to undergo an operation, which we had wished to avoid if possible, but which was quite certain to cure him. He was no better at the end of the week, and was eager to have the operation. He was given enough ether to make him excited, and two small cuts were then made behind his ear; a hammer was banged on a sheet of iron during the "operation." The moment after the incision had been made, the patient jumped off the table with his hearing restored. The second patient was unwilling at first to undergo an operation, but finally consented, as he was naturally impressed by the recovery of the first patient,

who was in the same ward and had been deaf for a much longer period. The same "operation" was performed and complete recovery occurred in exactly the same way. The joy shown by both patients on their recovery whilst in the theatre and on return to the ward was so obviously unfeigned that there could be no possible doubt about the genuineness of the deafness; at one time we had been rather doubtful about the second patient, but fuller investigation had convinced us more than a fortnight before that he was not malingering.

Hysterical Deafness following Shell-Shock cured by "Operation." —Lance-Corp. M., 26 years old, was blown up by a shell on the 29th August, 1916. He became completely blind, deaf and dumb, although he did not lose consciousness. His sight returned the following day. On reaching England he was able to read and write, and he talked in his sleep. In spite of treatment with encouragement, electricity and etherisation, no further improvement occurred until one night in November, when he woke up and asked the Sister for a drink. After this he was able to talk normally, but the deafness remained.

He was transferred to the neurological section at Netley on the 21st March, 1917, seven months after the onset of deafness. He was found to be completely deaf both to air and bone conduction, though he could feel the vibration of a tuning-fork on his mastoid. A loud noise just behind his head caused a slight tremor of his hands, blinking, and dilatation of the pupils, although he heard nothing; a slighter reaction was produced on the second and third occasion when the noise was repeated; after this it disappeared completely and did not return again.

The functional activity of the vestibular nerve and semicircular canals was then investigated by the rotation test, and was found to be unaffected, as the normal nystagmus and giddiness were produced. As experience has shown that organic disease or injury of sufficient intensity to produce severe cochlear deafness is invariably accompanied by loss of vestibular activity, it was clear that the internal ear was free from organic changes. This was rendered still more probable by the fact that immediately after the explosion the deafness was associated with mutism, which is always hysterical when it develops after shell-shock. The patient was hypnotised by being made to stare at a lens for fifteen seconds, but the deafness persisted; he could not be made to respond to any suggestion, as he was unable to hear, and a loud noise produced no auditory motor reflex, the pupils as well as the eyelids remaining fixed. The deafness also

persisted during natural sleep, as it was found impossible to wake the patient by shouting "fire" and by banging a poker against a coal-scuttle within a few inches of his head, and no reflex contraction of his eyelids was observed. In the morning he had no recollection that anything unusual had occurred during the night. Suggestion with the aid of electricity, in which he had great belief, and attempts to re-educate the sense of hearing with various noises completely failed, but he was suddenly and completely cured by a "fake" operation on his ear on April 20th to his intense delight, as he had recently become extremely depressed at the absence of any sign of improvement after 7½ months. His hearing was accurately tested the next day, and it was found that it was perfectly normal both to air and bone conduction, and the auditory motor reflexes had returned. He was discharged to duty three weeks later, feeling perfectly fit. He visited the hospital on June 29th a few days before he left for France; his hearing was normal and he was well in every way.

Tinnitus and Auditory Hallucinations.—Tinnitus may be associated with hysterical deafness and may continue after the disappearance of the latter, but it also occurs independently.

Tinnitus resulting from True Shell-Shock.—A man admitted for symptoms resulting from the explosion of a high-explosive shell complained when he regained consciousness of constant noises in his head, which never ceased and greatly distressed him. His hearing was normal, but I found a thick hair growing in the external meatus close to the drum on each side; it touched the drum on the side in which the noises were loudest and the opposite wall of the meatus on the other side. The removal of the hair was immediately followed by disappearance of the tinnitus. The hairs must have been present for years, but until the irritability of his nervous system became exaggerated by the shell-shock they had caused no trouble. An alternative explanation is that the hair was driven in so as to touch the drum by the force of the explosion.

Tinnitus may result in auditory hallucination. The patient imagines he hears shells coming towards him and bursting, or he may hear the whizz of bullets or the blowing of whistles. Hallucinations of this kind are not uncommon in the absence of tinnitus and can generally be cured by hypnosis.

CHAPTER IX

DISORDERS OF VISION

Functional Asthenopia—Migraine.—An error of refraction, which had previously caused no trouble, may give rise to severe headaches or actual migraine in a man exhausted as the result of active service, or in a man suffering from concussion as a result of shell-shock. In both conditions the activity of the ciliary muscles and the extrinsic muscles of the eyes is impaired, like that of the other muscles of the body. In severe cases of shell-shock the patient may be unable to read for weeks owing to difficulty in accommodating. But even a slight degree of asthenopia is important if there is an error of refraction, although this may never before have caused any trouble. At first light is intolerable and the patient makes no effort to use his eyes, but after a time he begins to look at his surroundings, and this alone may be sufficient to cause headache and giddiness. Later he tries to read; the difficulties of near vision are now added to those of distant vision, and the neurasthenic symptoms, especially headache, become aggravated.

The refraction of all soldiers suffering from headache as a result of neurasthenia or shell-shock should therefore be tested, if rapid improvement does not occur; when there is reason to suppose that asthenopia is an important factor, the eyes should be examined under atropine whilst the patient is still in bed. Glasses must be worn all day at first; they should be given up for distant vision and subsequently for near vision when complete recovery has occurred.

Hysterical Blindness.—Just as one must listen in order to hear, so one must look in order to see, and a man who for any reason is convinced that he is blind fails to see because he does not look. That psychical blindness may occur in this way is a matter

of common experience, as an individual who is deeply engrossed in his thoughts becomes totally blind to what is going on around him.

Whilst a man is still semi-conscious or dazed as a result of shell-shock, he is sometimes more or less completely blind. Vision generally returns as he regains consciousness, but if there is anything which draws his attention to his eyes the blindness may persist as a result of auto-suggestion. In two out of the nine cases I have seen of hysterical amblyopia, an error of refraction, which was previously ignored, was present; it increased the loss of function caused by shell-shock to such an extent that the patient's attention was drawn to his eyes, and by a process of auto-suggestion the loss of vision which would otherwise have quickly disappeared, was perpetuated.

Hysterical Blindness following Shell-Shock in an Officer with Extreme Hypermetropia.—An officer, aged 20, had never used his left eye owing to an extreme degree of hypermetropia. When the other eye was covered he could only see very indistinctly with it, but in spite of this he managed to pass the medical examination on entering the army. He was hit on the left side of the head by the butt end of a rifle in June and was unconscious for a few minutes. When he regained consciousness he at once noticed that he could not see at all with his left eye, although he had hitherto been in the habit of neglecting the blurred image he saw with it. On August 10th he received a slight wound to his left thigh, but continued on duty. The wound had not completely healed and was still somewhat painful when, on August 23rd, he was blown up by a high-explosive shell. On regaining consciousness he found himself being carried on a stretcher. The pain from his old wound drew his attention to his left leg, and he thought he would be unable to walk. When he reached the hospital ship he found that this was the case, although there was no new injury to the leg. He also complained of severe pain above the left eye, which he kept covered by a shade, as he found that the least light greatly increased the headache; when the shade was removed he was unable to open the eye at all. For some time he was in an extremely excited condition, and he slept very badly owing to nightmares. His eye was kept covered by a shade during his journey home from the Dardanelles. On his arrival in England it was found that beyond the hypermetropia his left eye was normal, although he was quite unable to see anything with it.

He was hypnotised on four occasions. After the first he slept better, the nightmares ceasing and the headache being less severe. On the second occasion, whilst still asleep, the shade was removed from his eye, and he did not discover until half an hour after he woke up that it was no longer present, although up to that time he said that the least light caused extreme discomfort and spasm of the eyelid. After the third treatment he found that he could see almost as well with his left eye as before he was hit on the head. Meanwhile, he was still unable to walk without crutches, although the wound to the leg had completely healed, and there was no physical cause to account for this. He was hypnotised again and it was suggested to him that he would be able to walk quite well; complete recovery followed.

In three other cases sand was blown into the men's eyes from the sand-bags of the parapet on which the shell exploded, and the irritation it caused drew their attention to their eyes and resulted in blepharospasm and amblyopia, which persisted long after every trace of conjunctivitis had disappeared.

Hysterical Blindness and Blepharospasm after Shell-Shock with Initial Conjunctivitis.—The patient had an attack of blindness following conjunctivitis, which was caused by a sandstorm in Egypt early in 1915. He recovered from this in ten weeks, and six weeks later went to the Dardanelles. On July 12th a shell struck a sand-bag immediately in front of him and the sand flew into his eyes. He did not lose consciousness, but his sight gradually became more and more deficient until at the end of ten days he was only just able to distinguish light from darkness. His vision had not improved when he was seen on September 17th, and he had severe blepharospasm; a few pieces of grit were still embedded in the conjunctivæ, although there was no inflammation.

The patient was easily hypnotised, and considerable improvement occurred during the next three days. He was hypnotised on three more occasions, and he could see perfectly well by September 30th. He still had some photophobia; he wore dark glasses and constantly blinked when they were removed. He was hypnotised again, and it was suggested to him that the photophobia and blinking would now cease. The result was completely successful, as all symptoms had disappeared by October 4th, although no attempt had been made to remove the grit from the conjunctivæ.

In one case the blindness was at first organic, the result of concussion of the visual centres in the occipital lobes. When the

slight organic charges caused by the concussion disappeared, the blindness was perpetuated by auto-suggestion and remained until it was cured by suggestion three months later.

Partial Hysterical Blindness, following Organic Blindness caused by Occipital Concussion and associated with Hysterical Deafness.—Pte. W., aged 22, was wounded over the right occipital region on the 7th June, 1917. He was unconscious for five days and was then trephined. On admission to Netley on July 6th, 1917, he was completely deaf in both ears, but as the vestibular reactions on rotation were normal, the deafness was regarded as hysterical (p. 117). It was noticed that he had difficulty in seeing and that he held anything he wished to read low down on the right side, although he volunteered no complaint about this, and only spoke about his deafness. On further examination it was found that he was totally blind, except in the right lower quadrant, the blindness being what might be expected to result from the wound over the lower part of the right occipital lobe, as it was near the middle line, so that the left lobe might also have been injured to a less extent. An attempt was made at the end of August to cure the hysterical deafness by a pseudo-operation, the patient being told that a cut behind his ear would certainly restore his hearing. Nothing was said to him about his blindness, which was regarded as organic. The "operation" resulted in immediate improvement in his hearing, as it was now possible to carry on a conversation with him by shouting. Quite unexpectedly it was found that his vision was now absolutely normal, the blindness having been cured by the suggestive effort of the "operation." It must, therefore, have been due to perpetuation by auto-suggestion of the organic blindness, which was caused by concussion rather than destruction of the occipital lobe.

One of my cases was the direct result of unconscious medical suggestion.

Hysterical Blindness due to Unconscious Medical Suggestion.—A man received a wound which grazed the left mastoid process and occipital bone. He became totally deaf on this side, but was otherwise well. As it was thought that a wound in this neighbourhood might have injured his visual centre, he was sent to an oculist for further examination. A prolonged investigation was carried out with a perimeter, at the end of which the patient was almost completely blind in his left eye, the right eye remaining unaffected. He would certainly have discovered such an extreme degree of blindness himself if it had been present before the examination, and its nature was what a layman might expect—blindness of the eye on the side of the injury,

though the latter could only have caused loss of the right field of vision.

My remaining case was due to the irritation caused by the fumes from a lachrymatory shell producing blepharospasm, which was perpetuated by auto-suggestion after the irritation had disappeared, and which also gave rise to the idea of deficient vision.

Hysterical Amblyopia and Blepharospasm following Irritation by a Lachrymatory Shell.—Pte. B., aged 44, was gassed in May, 1916, by a lachrymatory shell. Next day he was able to carry on, but he constantly blinked and his vision became somewhat defective. The blinking and defective vision continued, but he did not go into hospital until April, 1917. The thorough examination of his eyes which was repeated in three different hospitals appears to have led by suggestion to severer blindness, and the blepharospasm became worse. The case was diagnosed as disseminated sclerosis and subsequently as cerebellar tumour on account of the swaying gait, which was, however, simply the result of defective vision. On admission to Netley in September, 1917, his vision was 3/60 in both eyes; he was constantly blinking, and had a staggering gait. Suggestion with the aid of faradism applied to his eyelids caused the blinking to stop, and his vision and gait became normal for the first time for sixteen months. The next day the blinking had returned, but he again improved as a result of further suggestion, and a week later he was discharged to duty.

Symptoms.—The eyes are kept closed, and the lids frequently "flutter" or "twinkle," even in a subdued light. On attempting to force the lids open the patient resists by contracting his orbicularis muscle; when this is overcome to a sufficient extent to see the globes, they are found to be directed as far upwards as possible, and the patient is unable to look downwards. The pupils react normally to light, and the optic discs show no change. The patient complains of pain and photophobia, and is much fatigued after being examined. He does not move about like a blind man, but avoids hurting himself, though he does not relax the groping action of people with extremely defective sight. Judged by every test, he is psychically blind, and there is no difficulty in differentiating him from a malingerer, as he passes through long periods of real mental distress.

Treatment.—Vision may return spontaneously or as a result of some emotion. In one case reported to me by Dr. A. Wightwick

recovery occurred when a cat jumped on the patient, and the sight of a Canadian soldier returned when the ship in which he was going to Canada was torpedoed. But in some cases the blindness would probably be permanent if its hysterical nature were not recognised and active treatment given. In a group of soldiers suffering from hysterical blindness and blepharospasm seen with Captain A. W. Ormond, treatment by encouragement, persuasion, rest, tonics, deprivations, such as abstention from tobacco, confinement to bed or isolation, and counter-irritation have been tried without success. Suggestion during hypnosis, however, proved entirely successful in every case, and one may therefore conclude that when simple encouragement and persuasion have not resulted in recovery within a week of the onset of the blindness, suggestion under hypnosis should be practised without further delay. There is indeed no reason why it should not be used the first or second day, as the sooner recovery from such a symptom occurs the sooner will the patient be fit to return to duty.

Additional Cases of Hysterical Blindness following Shell-Shock and cured by Hypnotism.

Case 1.—The patient, aged 22, was looking over a parapet on July 18th, 1915, when a shell struck the sand-bags in front of him. He remembers the sand being thrown up into his eyes, after which he fell back and knocked his head. He was rendered unconscious for twenty-four hours. His first impression on regaining consciousness was extreme irritation in his eyes, and his eyelids were constantly quivering. He tried to open them, but found he could not do so. His mind thus became concentrated on his eyes, and owing to the confusion, which is common among the uneducated between inability to open the eyes and blindness, he became obsessed with the idea that he was blind and that he would never be able to open his eyes or see again. The impairment of other functions, which was doubtless present at first, remained unnoticed in this greater trouble, except for some loss of hearing, which quickly disappeared. The condition of his eyes had not altered when I first saw him with Captain Ormond on September 17th. He was quite blind, and there was a constant flicker of his eyelids, which were kept almost closed. On forcibly opening his eyes they were found to be turned so far upwards that it was difficult to see even the iris. A few fragments of sand were still embedded in the conjunctiva but not in the cornea; there

was no inflammation present. The inability to open the eyes and the idea of blindness were thus perpetuated by auto-suggestion, and persisted long after the inflammation caused by the dust had disappeared.

The patient was easily hypnotised, and whilst asleep he was told that he would be able to see when he woke up. The moment he awoke the suggestion was repeated very forcibly, and his eyes were held open. He cried out that he could see, tears ran down his cheeks, and he fell on his knees in gratitude, as he had thought that he was permanently blind and believed that his sight had been restored by a miracle. When seen again on September 20th the external appearance of his eyes was normal, and he said that he was able to see as well as he had ever done. Captain Ormond, however, found that there was some opacity of the vitreous of the left eye, secondary to hæmorrhage from a retinal vessel; this was doubtless a result of injury at the time of the explosion. There had been no return of symptoms, and the patient was well in every way when I last saw him on September 30th. His vision was 6/6 in the right eye and 6/36 in the left.

Case 2.—The patient, aged 20, was rendered unconscious for a few minutes as a result of the explosion of a shell near him on August 21st in the Dardanelles. Some of the powder was blown into his eyes, which were very sore when he regained consciousness, although he was still able to see quite well. During the next twenty-four hours his vision became more and more impaired. The powder was removed from his eyes when he was taken on board the hospital ship, and his eyes were bandaged. After a few days he thought he would be able to see quite well if the bandages were removed, but the medical officer in charge told him it would be dangerous to do this. During the voyage home he was not allowed to remove the bandages, and he became more and more convinced that this must be because the medical officer thought he was blind. The bandages had not been removed when he was admitted into the hospital on September 25th. On removing them he was found to be in exactly the same condition as the preceding patients. He could distinguish light from darkness, but was unable to see anything, and he kept his eyes turned up and his eyelids closed and constantly twitching.

On September 27th he was hypnotised by Captain Ormond, after which he found that he could see quite well, but the light still worried him and the blinking continued, though to a diminished extent. He rapidly recovered, and was soon able to return to duty.

Case 3.—The patient, aged 29, was knocked over by a

high-explosive shell in the Dardanelles and remained unconscious for a considerable time. On coming round he found he could only distinguish light from darkness; there was no smarting of the eyes, but he constantly blinked. He had a slight headache, but was otherwise well. He began to improve about September 10th, so that he could recognise shadows passing in front of his eyes, but no further improvement occurred until he was hypnotised on September 18th. There was very slight improvement as the result of suggestion at the time, but when seen on the 20th he said that he was beginning to recognise objects, and the blinking was less marked than before, but he still kept his eyeballs turned upwards and his eyes almost closed. He was hypnotised again on September 20th and 25th, and when seen on the 30th his sight was quite normal and the blinking had completely ceased. Subsequent retinoscopic examination revealed the presence of a considerable degree of myopic astigmatism in the right eye and mixed astigmatism in the left.

Case 4.—The patient was signalling from a gun limber on April 28th when he was blown up and remained unconscious for six days. There was no external wound, but on regaining consciousness he found that he was blind, except that he could just distinguish light from darkness; he was also completely deaf and was unable to speak. He regained his speech in June after a fortnight's treatment by hypnotism at Plymouth, but his sight and hearing remained unaltered.

When I saw him on September 17th he could only be made to hear by shouting down an ear-trumpet; he kept his eyelids almost closed and constantly twitching with the eyeballs turned upwards. He was extremely depressed, as he had been told by an aurist that he would never regain his hearing, as it was said that nerve deafness of such long duration could not improve, although the drums were intact. He concluded that the blindness would also be permanent. Moreover he was much worried with domestic troubles. It was not easy to hypnotise him, as he was unable to see, and all suggestions had to be shouted down his trumpet, but Captain Ormond succeeded at the first attempt. When seen on September 20th he said that his sight was distinctly better, and he was able with difficulty to open his eyes. He was much more cheerful, particularly when we told him that his hearing would also return, as his auditory nerves were no more organically diseased than his optic nerves. On September 30th there was some further improvement, as he could see everything in the outer part of his left field of vision fairly well. In November the patient was given an anæsthetic, and suggestion

was tried during the stage of semi-consciousness with marked success, as on the following day he opened his eyes voluntarily. His hearing gradually improved, and by March, 1916, he had recovered completely.

Hysterical Anosmia and Ageusia.—I have heard of no case in which a soldier has spontaneously complained of loss of the sense of smell or taste in the absence of organic injury, but a few cases have been reported in which such loss was discovered on examination. I have no doubt that in every instance it was produced by suggestion when the examination was made, as these symptoms can be very readily produced by injudicious questions.

CHAPTER X

HYSTERICAL PAIN

I HAVE seen a number of cases in which pain, caused by some organic condition, such as an injury to a nerve, has continued with great severity after the removal of the cause. The patient's nervous system was profoundly depressed in every case, and it seems probable that the pain was due to auto-suggestion. In the slighter cases cheerful surroundings and plenty of people to talk to are needed rather than the quiet and isolation which are necessary for severe cases. An officer, from whom Sir Alfred Fripp had removed a bullet in the immediate neighbourhood of the sciatic nerve, still complained of excruciating pain along its course. We discussed the question of injecting the nerve with eucaine or saline solution so as to render it temporarily anæsthetic, but nothing of the kind was required, as he improved directly he was removed from a private room to a large and lively ward.

For intractable cases suggestion may prove of value. An officer, who complained of constant severe pain in his back, where he had been bruised by a bullet, which had passed through his coat without penetrating his skin, was rapidly cured by means of the high-frequency current, which acted, I expect, as much by suggestion as by any direct analgesic influence.

In some cases in which pain caused by injury to a nerve-root, especially in the cauda equina, cannot be relieved by operation, I have found hypnosis of great value; it often diminishes the longing for morphia which generally develops, and in some cases it has seemed that the patient groaned and cried out in order to have morphia, when the pain was no longer present or was only present in his imagination, as complete disappearance of pain and of the desire for morphia followed suggestion under hypnosis repeated on some six or seven occasions.

The severest and most intractable pain, which is primarily organic in origin, but is later partly or entirely hysterical, results from injuries to the median nerve. The condition was admirably described at the time of the American Civil War by Weir Mitchell, who called it *causalgia* (καῦσος, burning heat, and ἄλγος, pain) on account of the burning character of the pain, which is only relieved by constant application of rags soaked in cold water. It is most severe in the hand, and is increased by excitement and the slightest external stimulus. The following is a typical example, which is no worse than many others which have been recorded, as the pain has led in several instances to suicide.

Hysterical Pain following Injury to the Median Nerve (Causalgia).—Pte. M., aged 22, was wounded in the right arm in July, 1916. He was taken prisoner and kept in Germany until December, when he was sent to Switzerland, and he reached Netley in August, 1917. Severe pain in the hand began immediately after he was wounded and has continued without intermission ever since. He was badly treated in Germany and was tied down in order to have massage and electricity. The pain was confined to the right hand, except for a much less severe pain in the other hand. The least noise, excitement, or movement greatly increased it, and the patient could not bear to be touched in any part of his body, as this always aggravated the pain. The only relief he could obtain was by applying a rag soaked in cold water. He could only wash in cold water, and he chose the coldest corner of the ward for his bed; he would eat and drink nothing hot or even warm, as both external and internal heat gave him pain in the hand. It was impossible to make any examination, as he was terrified at the idea of being touched. His general condition rapidly improved with strict isolation and hypnosis, but the pain continued. The wound was explored by Captain Joll on September 19th, and the nerve was found almost obliterated by fibrous tissue, although in most cases of causalgia the lesion of the nerve is comparatively trivial. Two inches had to be resected before normal nerve tissue was reached, and the cut ends were then brought together. In spite of this the pain persisted as severely as ever, and the patient still maintained that he could only obtain relief by cold applications to his hand, although complete anæsthesia in the distribution of the median nerve must have been present. It was clear that the pain, which must have been originally due to irritation of the nerve, must finally have been entirely psychical in origin.

The perpetuation of the pain by auto-suggestion was particularly likely to occur in this man, who had been rendered abnormally suggestible not only by exhaustion following prolonged severe pain, but also by the moral effect of the ill-treatment he had suffered whilst a prisoner in Germany.

CHAPTER XI

THE WAR AND ORGANIC NERVOUS DISEASE

THERE is no doubt that organic nervous diseases are aggravated by factors, such as fatigue and worry, which are often erroneously supposed to lead only to functional disorders, and in years to come we shall probably see cases of paralysis agitans, disseminated sclerosis, and other organic nervous diseases, in which the war will prove to have been the exciting cause. I have not, however, seen a single soldier suffering from paralysis agitans or disseminated sclerosis; the numerous cases sent home with the latter diagnosis have all proved to be hysterical, with the exception of a few men with cerebro-spinal syphilis. Bruce has recorded the case of a soldier, who had had symptoms of disseminated sclerosis twelve years before, but then had a complete remission; the symptoms returned suddenly as a result of severe wetting during a long route march. I have already referred to the various syphilitic diseases of the nervous system and to epilepsy.

Among the numerous cases of sciatica I have seen, a large proportion occurred in soldiers who had suffered from it before the war, the return of symptoms being due to exposure and fatigue. Exaggeration and voluntary prolongation of incapacity are particularly common in men who have had sciatica. Malingering can be easily detected by the absence of the physical signs, some of which are invariably present in true sciatic neuritis. The ankle-jerk in my experience is always diminished on the affected side, and in severe cases it is absent. The comparison should be made with the patient kneeling symmetrically on a cushioned couch or chair, after complete relaxation has been shown to be present by squeezing the calves and seeing that the foot is plantar flexed. In most cases pain is produced on extension of the leg on the flexed thigh (Lasègue's sign), and on the adducted thigh (Bonnet's sign), and the knee is flexed on the affected side when the patient stands and bends his trunk forward (Neri's sign).

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PART II

INFECTIVE DISORDERS

"THE health of the troops has been most satisfactory, and during the period to which this dispatch refers (May 19th to December 23rd, 1916) there has been an almost complete absence of wastage due to disease of a preventable nature."
—SIR DOUGLAS HAIG.

CHAPTER XII

DYSENTERY

DYSENTERY is the name applied since the days of Hippocrates to a condition in which frequent stools containing blood and mucus are passed and tenesmus is present. The discovery of the *Entamœba histolytica* and of the *Bacillus dysenteriæ* resulted in the differentiation of amœbic and bacillary dysentery, which are now known to be distinct in their geographical distribution, morbid anatomy, and to some extent in their symptoms, and to require distinct treatment. The modern tendency has been to restrict the term "dysentery" to infective colitis caused by these organisms, even if no blood and mucus are passed and no tenesmus is present. It is, however, more logical to include all forms of infective colitis, in which blood and mucus may be passed and tenesmus may occur, as the disease produced by certain other protozoa closely resembles amœbic dysentery, and there is a greater similarity between the morbid anatomy of the ulcerative colitis, which occurs sporadically in England, and chronic bacillary dysentery than between bacillary and amœbic dysentery.

(i) Amœbic Dysentery

Amœbic dysentery is common in tropical and sub-tropical countries. It had never occurred in epidemic form in Europe

until the summer of 1915, when nearly every soldier in the British Army at Gallipoli suffered from it, and a large proportion of the thousand sick men, who were daily removed from the Peninsula during August and September, had amœbic dysentery. It was less common in October, and the cold and rain in the great gale at the end of November were quickly followed by the disappearance of the epidemic. The disease was probably brought to Gallipoli from Egypt, where Wenyon and O'Connor found that at least 13·5 per cent. of healthy natives are carriers of the infection, as it never became common among the French troops at Sedd-el-Bahr, though it was common among the Turkish prisoners taken on the Peninsula. A comparatively small number of cases occurred at Salonica in the summer of 1916 and 1917, mostly in troops who had been in Gallipoli or Egypt; they formed, according to Graham, less than three per cent. of the total cases of dysentery.

Amœbic dysentery first appeared in France in September, 1915, and though it is still rare, the number of cases has steadily increased both among British and French troops. The infection was introduced by carriers from India, Morocco, and Senegal; the arrival of troops from Gallipoli led to a fresh source of infection in 1916, and the disease has now spread to men who have never seen service out of France. Thus on investigating the stools of soldiers sent from France as convalescent from dysentery, Inman and Lillie found that cysts of *E. histolytica* were present in about ten per cent. of the English cases and twenty per cent. of the Australian cases, although only one-third had previously been in a country where amœbic dysentery is endemic.

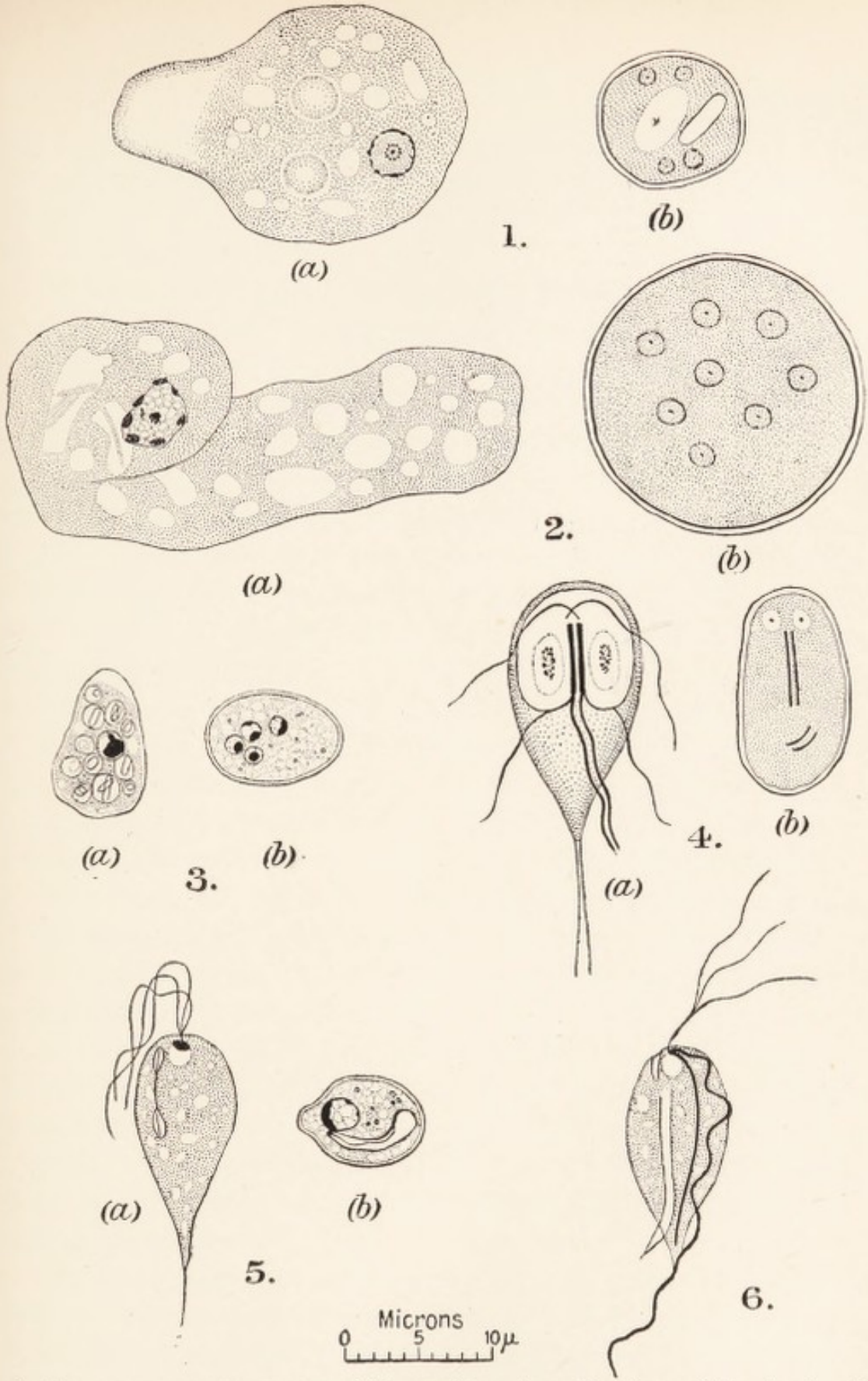
Parasitic amœbæ—entamœbæ—are distinct from the non-parasitic amœbæ of the soil and water in their morphological characters. They have not yet been cultivated artificially, and they become parasites in the intestine when given by mouth, whereas the non-parasitic amœbæ can be cultivated, but spontaneously disappear from the fæces within a week of being swallowed.

There are three varieties of entamœbæ, which are structurally distinct; two—the *Entamœba coli* (Fig. IV., 2) and the recently isolated *Entamœba nana* (Fig. IV., 3)—are harmless parasites, found in the stools of many normal individuals, and the other

—*Entamœba histolytica*, or *E. dysenteriæ* (Fig. IV., 1)—is the cause of amœbic dysentery. The *Entamœba coli* produces no lesions when given to kittens by mouth, but the *Entamœba histolytica* produces characteristic ulcers in the colon and abscesses in the liver, which contain the pathogenic amœbæ but no bacteria.

Different forms of the entamœba of dysentery have been described. Wenyon has now definitely proved that the large form (15 to 30μ in diameter), which invades the tissues, and the *E. minuta* (10 to 20μ), which lives like the *E. coli* on the surface of the mucous membrane, are really two forms of a single organism, the *E. histolytica*. The large form is the only one found in the stools of acute dysentery; its size is about the same as that of *E. coli*, but it is more refractile and greenish, with a clearer distinction between ectoplasm and endoplasm, its pseudopodia being formed of highly refractile ectoplasm alone, its nucleus is smaller and less easy to detect, and its movements are more active; it may contain many red blood corpuscles, though none are found in *E. coli* or *E. nana*, which is very small (6–12μ) and sluggish in its movements. When the acute symptoms abate the *E. minuta* replaces the large form. Both forms reproduce by simple division; some cease to multiply, become spherical and encyst themselves by secreting a transparent shell. The cyst is completely passive and is excreted in the fæces. Its single round nucleus divides into two and then into four. The four nuclei, on account of which it has been described as the *E. tetragena*, distinguish it from the cyst of *E. coli*, which contains eight or sixteen nuclei, and is generally larger (11 to 34μ instead of 7 to 14μ, but occasionally even 20μ) and less refractile. The difference between the cysts is of great importance, as the active stage of the minute form of *E. histolytica* closely resembles that of *E. coli*. The cysts of *E. nana* have one, two, or four nuclei, and are about the same size as the smaller cysts of *E. histolytica* (7 to 10μ), but they are oval instead of round and do not contain chromatoid bodies, like those seen in *E. histolytica* cysts when examined in saline solution.

The active forms of *E. histolytica* die too quickly outside the body to take any great part in spreading the disease. Amœbic cysts survive many days in moist fæces and in water; the infection can therefore be carried in particles of fæces, which



1 ENTAMŒBA HISTOLYTICA : (*a*) large tissue-invading form with ectoplasmic pseudopodia and containing two red blood corpuscles ; (*b*) encysted form with four nuclei, chromidial body and vacuoli.

2 ENTAMŒBA COLI : (*a*) large entamœba ; (*b*) encysted form.

3 ENTAMŒBA NANA : (*a*) showing nuclei and numerous vacuoles containing ingested bacteria ; (*b*) quadrinucleate cyst.

4 LAMBLIA INTESTINALIS : (*a*) surface view showing sucking disc, two nuclei and eight flagella ; (*b*) encysted form with two nuclei.

5 TETRAMITUS (OR CHILOMASTIX) MESNILI : (*a*) free form ; (*b*) lemon-shaped cyst, with nucleus, remains of buccal structures and volutin grains.

6 TRICHOMONAS INTESTINALIS.

FIG. IV. INTESTINAL PROTOZOA. Nos. 1, 2, 4, 5 (*a*), and 6 after Wenyon ; Nos. 3 and 5 (*b*) after Dobell.

are moist inside though dry outside, and pieces of paper or leaves contaminated with moist fæces, and in contaminated drinking water. As the cysts are rapidly killed by drying, they cannot be carried in dust.

Wenyon and O'Connor's investigations have shown that flies are much the most important means of spreading the disease. They ingest fæces, which they begin to excrete within half an hour, and in twenty-four hours the excretion is almost complete. If the fæces contain amœbic cysts, the latter can be found during the whole of this period in the intestines of the fly. As a fly generally defæcates whenever it ingests food, it deposits fæces containing amœbic cysts on any human food upon which it settles, if it has had access to infective fæces in the preceding twenty-four hours. A considerable number of house-flies caught by Wenyon and O'Connor in different parts of Alexandria, including one caught in a cookhouse, deposited fæces containing cysts of *E. histolytica* and *E. coli*. The infection is rarely if ever carried by the fly on its legs, body, or proboscis, as it cleans itself after leaving the fæces, and the traces still left dry so rapidly that no amœbic cysts could survive long enough to be deposited on food.

The stools of convalescent and contact carriers are a more fertile source of infection than those of patients already suffering from dysentery, as the encysted amœba is present in largest quantities in the formed stools of patients, who have recovered from dysentery—convalescent carriers, and of individuals who have never had definite dysenteric symptoms, the disease having been latent or the submucous tissue having entirely escaped invasion. Fatal dysentery can be produced in cats by feeding them on the cyst-containing fæces of healthy carriers, who have had no symptoms of dysentery.

Owing to the extreme frequency of defæcation, soiling of the patient's clothes and skin is common, and hospital attendants may infect themselves, if they are careless about washing their hands. Defective personal hygiene is a frequent cause of the spread of the disease in camps, as the hands are likely to be soiled with infective material in the latrines, and if they are not thoroughly washed, the food becomes contaminated.

The cyst wall is unaffected by the gastric juice, but is digested by the pancreatic juice. Each of its four nuclei develops

into an active amœba, which burrows from the lumen of the colon through the mucous membrane into the submucous tissue.

Infection may at once lead to an attack of dysentery, or the individual may become a carrier and either remain healthy though still a carrier for six months or more, or he may eventually develop dysentery. Among 1979 healthy British troops, whose stools were examined by Wenyon and O'Connor in Alexandria during 1916, 106 or 5·3 per cent. were found to be carriers of cysts of *E. histolytica*, although only 16 had had attacks of slight diarrhœa or abdominal pain. As traces of mucus were often present, it is probable that a mild degree of catarrhal colitis existed in spite of the absence of symptoms.

Morbid Anatomy.—The essential lesion in amœbic dysentery is an inflammatory round-celled infiltration of the submucous tissue of the colon, caused by the invasion of the amœbæ. The proximal part of the colon is first involved, and even when the whole colon later becomes affected, the disease is generally most advanced in the cæcum. The appendix is occasionally affected, but the ileum is always spared.

Small raised red dots first appear as a result of congestion of the mucous membrane over an area of submucous infiltration; the centre of the congested area soon becomes necrotic and yellow, and the necrotic tissue finally disappears, leaving a spreading ulcer with an overhanging margin and the infiltrated submucous tissue as its base, or a flask-shaped cavity containing pus. The affected areas are greatly thickened and are raised above the adjacent mucous membrane, which remains comparatively healthy; even in the severest cases areas of normal mucous membrane remain, especially in the distal half of the colon. In the worst cases black sloughs of necrosed mucous membrane, which may be several inches in diameter, separate, and are passed in the stools, or are found after death attached here and there to the edge of a large ulcer. In these cases the amœbæ pass between the muscle fibres to the subserous coat, and the round-celled infiltration extends to the muscular coat and peritoneum, which is covered with purulent fibrinous patches.

In very chronic cases and in the process of healing the ulcers become slightly depressed, round or oval in shape; they finally leave dark rounded scars.

The amœbæ may invade the small submucous veins, by which they travel to the liver, where they give rise to inflammation and suppuration (*vide* Chapter XIII). In rare cases abscesses also form in the brain and spleen. I saw two cases in Salonica, in which all the symptoms of cerebral abscess were present; both patients had had dysentery at Gallipoli, and rapid and complete recovery followed treatment by means of emetine injections. It seems probable that they were suffering from amœbic encephalitis, corresponding with the hepatitis which precedes the actual formation of an abscess in the liver.

Symptoms.—The incubation period is short. Crean records the case of a soldier who landed in Alexandria from England at 4.30 p.m. on a Friday. He had never been abroad before. He at once marched to Mustapha, where there were several cases of amœbic dysentery. At 9 a.m. on Sunday—forty hours after his arrival—he passed blood and mucus, which contained amœbæ.

The onset of amœbic dysentery may be acute, but in a considerable proportion of cases the disease begins as chronic diarrhœa or as diarrhœa alternating with constipation. In acute cases ten to twenty or even more stools are passed a day. When the disease has fully developed, blood and mucus are almost always present in the stools, which consist of a few drachms of greenish yellow or dirty brown mucus, or a grey muco-purulent mass, suspended in semi-fæculent or serous fluid, which is often blood-stained; the blood is either intimately mixed with the stool or is present in streaks. In other cases large separate fragments of mucus stained bright red are seen. At least one stool should be inspected every day. Additional information can often be obtained by diluting it with water and allowing it to stand a couple of minutes, when the mucus and sloughs sink to the bottom; the fluid is then poured off with most of the fæces, and the residue is washed repeatedly until no fæcal matter is left.

If a microscopical examination is made within half an hour of the passage of a stool, active amœbæ can generally be found without difficulty in the blood-stained mucus, but they may also be present in mucus without obvious blood and in watery stools free from blood and mucus. The amœbæ are generally scattered among numerous pus cells and often numerous red corpuscles with some epithelial cells.

Griping pains, which are worst shortly before and during defæcation, are always present to a greater or less extent across the lower part of the abdomen. Tenesmus, the painful bearing down and straining sensation which occurs during and for some time after defæcation, is common, but it is only severe if the rectum is ulcerated.

The thickened colon can often be felt, especially in the right and to a less extent in the left iliac fossa, as a very tender tumour; when the cæcum is chiefly involved, appendicitis may be simulated, but the tenderness is found to extend over the whole cæcum and ascending colon instead of being confined to the appendix itself.

Constitutional symptoms are generally slight and, apart from complications, fever is either completely absent or low and intermittent even in acute cases. Leucocytosis is generally present, and there is a relative increase in the mononuclear cells; in severe cases the number of leucocytes per c.mm. often exceeds 20,000 and sometimes even 30,000.

In the fulminating gangrenous type of amœbic dysentery the patient passes very numerous stools, which often consist of blood with a little mucus but no fæces. When extensive gangrene occurs, the colon may become paralysed, and only a few stools containing black sloughs are passed. Abdominal pain is severe, and palpation shows that parts of the colon are greatly thickened and extremely tender. In such cases perforation and general or local peritonitis may occur.

Mild and unrecognised amœbic dysentery is a not uncommon precursor of hepatic abscess; a history of slight diarrhœa without the passage of blood, often alternating with constipation and sometimes accompanied by pain in the right side of the abdomen, is obtained. The comparatively mild symptoms are probably due to the lesion being confined to the cæcum and ascending colon, which may be tender and slightly thickened. In such cases amœbæ are difficult to find in the stools, unless frequent search is made for a little blood-stained mucus. The diarrhœa, which was common among the troops at Gallipoli, was probably in many instances a mild form of amœbic dysentery. The disease may even be completely latent, ulceration being found post-mortem in patients dying from amœbic abscess of the liver or from some intercurrent disease.

In chronic amœbic dysentery, which should never occur with efficient treatment, amœbic cysts are passed, and symptoms may continue for months or years, often with long intermissions; emaciation, anæmia, and general asthenia result, and the patient ultimately dies from exhaustion or hepatic complications.

Several cases of sciatic neuritis and a smaller number of neuritis affecting other nerves were observed in association with the outbreak of amœbic dysentery in the Mediterranean Forces.

Diagnosis.—In any neighbourhood in which dysentery is known to occur, every case of diarrhœa should be regarded as a possible case of dysentery, and if blood or mucus or both are passed or if tenesmus is well marked, it should be regarded as a probable case of dysentery. Other causes of similar symptoms, the most important of which is a growth of the pelvic colon or rectum, should not be forgotten; I found an inoperable growth in the rectum of a governess sent home from Persia for what was supposed to be chronic dysentery, and I saw a case in Lemnos, in which a man with a similar growth had been treated for some weeks with emetine. A careful inquiry into the history and in doubtful cases a rectal examination should prevent such a mistake being made.

If symptoms of appendicitis occur in a patient, in whom there is a possibility that amœbic dysentery is present, the stools should be examined for amœbæ, and the effect of emetine injections should be tried before advising operation, except in severe cases, in which the delay caused by failure of the treatment might prove dangerous.

The clinical differentiation of amœbic and bacillary dysentery will be discussed after the symptoms of the latter have been described. A definite diagnosis can only be made by the discovery of the *Entamœba histolytica* in the stools. Various features have already been mentioned, which distinguish the *Entamœba coli* from the *Entamœba histolytica* both in the active and encysted states. But although an amœba with refractile ectoplasm clearly differentiated from the granular or vacuolated endoplasm, an indistinct nucleus, and active movement is generally the *E. histolytica*, its characteristics vary so much that it may be quite indistinguishable from the *E. coli*, in which the distinction between ectoplasm and endoplasm is not present, the nuclei are distinct,

and the movements are very sluggish. The inclusion of red corpuscles is, however, conclusive evidence in favour of *E. histolytica*, and Wenyon and O'Connor suggest that no acute case of dysentery should be diagnosed as amœbic unless amœbæ containing red corpuscles are found, as they are almost invariably present, or characteristic cysts containing one, two, or four nuclei are present in association with amœbæ, the general characteristics of which are suggestive of *E. histolytica*.

In extensive epidemics in war time, when it is impossible to examine the stools of every patient microscopically, treatment with emetine should at once be instituted in cases arising in a district in which the disease is known to have occurred; rapid improvement can be regarded as almost conclusive proof of the amœbic origin of the dysentery.

Prognosis.—If emetine is promptly given, death should hardly ever occur. When treatment is delayed, the prognosis is less good and the actual dysentery is likely to be followed by chronic colitis, which may persist for many months. Incontinence of fæces, persistent vomiting, hiccup and the passage of sloughs are the most serious symptoms.

The amœbic dysentery at Gallipoli "lowered vitality more than it destroyed; it was the universal sickness, not the occasional death, that mattered." It was, in fact, one of the deciding factors in the failure of the campaign.

Prophylaxis.—Neither chlorination nor the addition of acid sodium sulphate tablets to water destroys encysted amœbæ. All drinking water should therefore be boiled when amœbic dysentery is prevalent, and no raw vegetables in the form of salads, and no raw fruit unless it is carefully peeled should be eaten. In severe epidemics a gelatine capsule or salol-coated pill containing gr. i of emetine bismuth iodide which some individuals can take without any digestive disturbance, or a smaller dose if this cannot be tolerated, should be taken after food once a day in order to destroy any amœbæ which may be swallowed, before they have time to penetrate the mucous membrane of the colon. Every case of diarrhœa lasting for more than twenty-four hours should be regarded as possibly due to dysentery and treated in the manner described in the note at the end of this chapter. Dysentery wards should be fly-screened, and the stools should be disinfected at once

and destroyed as quickly as possible. A patient who has had dysentery should not be allowed to return to his unit until he can take a full Army ration without getting diarrhœa, and his fæces have contained no blood, mucus, or amœbic cysts for a fortnight. He should still be given emetine after his apparent recovery, as described in the section on treatment.

The fæces of all troops should be passed into dust- and fly-proof receptacles containing cresol, and whenever possible they should be incinerated ; failing this they should be deeply buried. However thoroughly this is done, flies will continue to carry infection from the fæces of native carriers. Camps and hospitals should therefore be situated as far away as possible from native quarters, and natives found defæcating in the open near a camp should be punished. Large and efficient fly-traps should be kept at the entrance to cook-houses, messes, and canteens, all of which should be protected by fly-proof netting. As in spite of these precautions flies are certain to enter, all food should be kept covered. Special attention should be paid to the health of cooks, and no man should be allowed to have anything to do with the preparation of food if he is suffering from diarrhœa. Before leaving their wards, medical officers, nurses, and orderlies, who are looking after dysentery cases, should wash their hands with cresol (1 in 10), which Wenyon and O'Connor have shown is the best antiseptic for destroying amœbic cysts.

Treatment.—In both amœbic and bacillary dysentery the patient should be kept warm in bed until the diarrhœa ceases. The diet required is the same for both infections. It should be entirely fluid at first, and in very acute cases nothing but barley water, albumin water or whey should be allowed for twenty-four hours. With this exception it should consist in the early stages chiefly of milk, citrated by the addition of two grains of sodium citrate to each ounce. Milk soured by a pure culture of lactic acid bacilli, yoghurt, or the similar Egyptian preparation called laban zebady, is very useful after the acute symptoms have abated. as it renders the conditions in the colon unfavourable for the development of dysenteric and putrefactive organisms. Arrow-root and cornflour made with water and sweetened can be given from the onset ; when improvement occurs they can be made with milk, and custard, purées of potatoes, peas or lentils, and

boiled rice, to which milk and sugar can be added, may also be given. Meat extracts should be avoided. When the stools begin to be formed the diet can be gradually increased.

Directly the diagnosis has been made by the discovery of the *Entamœba histolytica* in the stools, gr. i of emetine hydrochloride should be injected subcutaneously, and gr. i injected once daily for the next twelve days. This produces no more disturbance and is less trouble to the patient and the nursing staff than two injections of gr. ½ a day. In very severe cases gr. ½ to i dissolved in 5 c.c. of normal saline solution should be injected intravenously, and in somewhat less severe cases gr. i can be given subcutaneously twice a day for as long as may be necessary, but there should be an interval of a few days after each period of twelve days. The amœbæ, blood and mucus generally disappear from the stools between one and six days after the first injection. In the rare cases of amœbic dysentery, in which emetine fails more or less completely, a mixed infection is certainly present, and antidysenteric serum should be given in addition to the emetine. It is unnecessary to persevere with emetine until the stools are solid, as after the blood and mucus have disappeared the stools may remain loose for a time owing to the post-dysenteric and non-specific colitis still present. They may rapidly become solid on discontinuing the emetine, which apparently acts as a mild intestinal irritant. If any blood or mucus reappears in the stools, gr. i of emetine hydrochloride should be injected every night for six nights ; it is unusual for more than one such relapse to occur. In the very exceptional cases in which emetine injections cause vomiting, this may be prevented by the simultaneous injection of a small dose of morphia.

Dale found that repeated injections of emetine in cats had a cumulative action upon the heart. In very exceptional cases emetine has seemed to cause shortness of breath and irregularity of the pulse in patients who were not kept in bed, but these symptoms always disappeared after a few days' rest. Cases have been recorded of death from cardiac failure from emetine poisoning, but in all probability death was really due to the intestinal toxæmia. Pain and stiffness of the legs have also been said to follow the use of emetine, but this is only temporary, and there is no evidence that genuine neuritis ever occurs.

As amœbic cysts are often still present in the fæces of patients who have apparently recovered from an attack of dysentery, it has been supposed that active amœbæ may develop from the cysts and cause a recurrence of symptoms if the patient's general health is depressed or his colon is irritated, and that a chronic infection of this sort may give rise to amœbic hepatitis or abscess of the liver. Wenyon and O'Connor have, however, shown that this is not the case, as cysts do not liberate active amœbæ anywhere but in the small intestine. But the presence of cysts in the fæces indicates that free amœbæ must be present in the colon, and it is these that are a danger to the patient. From the patient's point of view it is the cyst-forming amœbæ rather than the cysts themselves which require destruction, just as it is the ankylostoma which produces the ova found in the stools in cases of ankylostomiasis and not the ova themselves which must be destroyed. It is therefore of comparatively small importance that amœbic cysts are very resistent to emetine, as if the drug is properly administered the amœbæ which produce the cysts are destroyed, and the cysts disappear from the fæces. The frequently expressed view that some cases of infection with *E. histolytica* do not respond to treatment because only the resistant cysts are present in the intestine is thus incorrect. It is only the cysts in the fæces, after they have been passed, which urgently require destruction in order to prevent the spread of the disease.

It is exceedingly difficult to eradicate the infection in chronic carriers by means of emetine injections. The cyst-producing amœbæ appear to be shut off from the circulation and are, therefore, more readily destroyed when the drug is given by mouth. In order to avoid the emetic action of ipecacuanha Major Warden, I.M.S., prepared an iodide of the alkaloids of ipecacuanha and mercury in 1891, and Captain Tull Walsh used it with great success in the treatment of acute dysentery. In 1915 Du Mez recommended a similar preparation, emetine-bismuth-iodide, which Dale introduced into England in 1916. It is insoluble in water and in gastric juice, but slowly decomposes in the alkaline juices of the intestine with liberation of emetine and precipitation of bismuth sulphide. One grain of emetine-bismuth-iodide is given in cachets or gelatin capsules three times a day after meals for two or three weeks; compressed and keratin-coated

tablets are less useful, as they are often passed unchanged in the stools and they are more liable to cause vomiting. This daily dose is equivalent to one grain of emetine hydrochloride and one drachm of ipecacuanha. Nausea and diarrhœa are generally produced, and it is therefore important to see that the patient really takes the drug. Vomiting may also occur, but the dose should not be reduced, as tolerance is often quickly established, especially if the patient makes an effort to restrain the desire to vomit. If vomiting persists, it can generally be overcome by giving m. x of chlorodyne half an hour before the drug is taken; failing this, the patient should remain in bed until the course of treatment is completed, and a mustard plaster should be applied to the epigastrium. The drug was given by Dobell to carriers of *Entamœba histolytica* cysts, all of whom had had a course of about a dozen daily injections of gr. i emetine hydrochloride without getting rid of the infection. In each case the cysts disappeared and did not reappear in the next few weeks. In three similar control cases no improvement followed a second course of twelve emetine injections; two of the three cases were subsequently given emetine-bismuth-iodide with the result that the cysts disappeared from the stools.

When pain is excessive, especially if it causes insomnia, and when very severe diarrhœa persists in spite of treatment and is leading to exhaustion, which would probably be fatal if it continued, a hypodermic injection of a quarter to half a grain of morphia should be given and repeated, if required, every four hours, with the addition of a hundredth of a grain of atropine. I have found charcoal very effective in diminishing the frequency of defæcation and in deodorising offensive watery stools: at the same time flatulence, which is often excessive and is the chief cause of colicky pain, is greatly reduced. Half an ounce of charcoal to a teacupful of sweetened arrowroot makes a palatable food; it should be given three times a day in the more acute stages, and a single dose should be given the last thing at night as long as the stools remain soft.

Hot hip baths are useful for tenesmus; when the latter is severe and persistent, the individual ulcers should be swabbed with a saturated solution of silver nitrate through a proctoscope.

If excessive diarrhœa results in collapse, three or four pints

of hypertonic saline solution, as recommended by Rogers for cholera, should be injected intravenously : two drachms of sodium chloride are dissolved in each pint of water. In less urgent cases, normal saline solution (gr. 90 to the pint) should be injected subcutaneously. According to French observers the collapse is sometimes due to suprarenal insufficiency ; in such cases, which can be recognized by the very low blood-pressure, the subcutaneous injection of seven minims of adrenalin chloride (1 in 1000) every three or four hours is of the greatest use.

Since the treatment of amœbic dysentery has become so much more effective as a result of using emetine subcutaneously, neither appendicostomy nor any other operation ought ever to be necessary.

(ii) Bacillary Dysentery

From what is now known about its geographical distribution it is highly probable that the dysentery, which has always been common in armies during war-time, was bacillary and not amœbic. Out of the 30,000 English soldiers who fought in the Crimea 7883 suffered from dysentery, and of these 2143 died ; in the South African War there were 38,108 cases with 1342 deaths. In the Federal army during the American Civil War there were 285,000 cases, as many as 21 per thousand troops being attacked in 1864.

Bacillary dysentery made its first appearance in the early weeks of the war in East Prussia and Galicia and among von Kluck's " ill-fed and tired " soldiers on their march towards Paris. Since then it has occurred on every front whenever the weather has been hot, but there has been no serious epidemic in France or Flanders. It was the common form of dysentery in the British Mediterranean Forces until June, 1915 ; during July, August and September a large majority of the cases were amœbic, but with the colder weather after October bacillary dysentery became once more the prevailing type, though its frequency gradually diminished, until at the end of the year there was very little dysentery of either kind. Bacillary dysentery was common, but amœbic dysentery comparatively rare, in the summer of 1916 among both British and French troops at Salonica and in the Army in Mesopotamia. Serious epidemics have broken out in various parts of

Germany and Austria since the beginning of 1917; 3802 cases with 550 deaths were reported in Prussia during the third week of September. The disease appears to have been spread by soldiers returned from the front.

The bacillus of dysentery was discovered by Shiga during an epidemic in Japan in 1898; varieties of the organism, differing in their cultural properties, were isolated by Flexner and Strong in 1900, and by Hiss and Russell—the Y-bacillus which is very similar to Flexner's—in 1903. The bacillary dysentery in the Mediterranean Forces and on the Eastern front was chiefly caused by Shiga's bacillus, and that on the Western front by Flexner's and the Y-bacillus, although in the summer of 1916 isolated outbreaks of Shiga infection were recorded, probably as a result of infection brought by troops who had previously fought on Gallipoli. In some cases bacilli have been found which differ slightly from all previously isolated varieties.

The disease is spread in exactly the same way as amœbic dysentery, and the *B. dysenteriæ* has been found in the excreta of flies up to three days after they had fed on infected stools. The stools of convalescent patients may still contain the bacillus for as long as a year after infection. So-called "contact carriers" have generally had mild diarrhœa, the nature of which has not been recognised, as there is no doubt that the *B. dysenteriæ* can cause slight catarrhal colitis, in which loose stools containing no blood and little or no mucus are passed.

Morbid Anatomy.—In acute bacillary dysentery all or nearly all of the colon and the last one to three feet of the ileum are involved. In chronic cases the changes are generally confined to the lower half of the colon.

The disease begins as an acute inflammation of the whole mucous membrane with areas of coagulation necrosis, which later give rise to superficial ulcers, the submucous tissue and the muscular and peritoneal coats escaping more or less completely. In a typical specimen dark greenish grey necrotic areas and irregular superficial ulcers are scattered over the inflamed and thickened mucous membrane. When the disease becomes chronic, the necrotic areas are completely replaced by ulcers, which may be so extensive that only scattered islets of thickened mucous membrane remain.

Symptoms.—The incubation period is between one and seven days. The disease generally begins acutely with griping abdominal pain ; this is soon followed by the passage of fluid stools containing mucus, which may be blood-stained from the onset. In the early stages between six and thirty stools, many of which consist entirely of mucus mixed with pus and blood, are passed in the twenty-four hours. When the rectum is involved tenesmus occurs, in addition to the abdominal pain which generally precedes defæcation ; it may be so persistent that the patient has a constant desire to open his bowels and is unwilling to leave the bed-pan. In severe cases there is often incontinence of fæces. The anus may become inflamed, excoriated, and painful. Drinking, eating, and every movement intensify the desire to defæcate. Micturition may also be frequent and painful. The intestines are very rarely thickened, and there is not much tenderness, but the iliac colon is often palpable as a firm, rather tender contracted cord. Vomiting is very rare, except in fulminating cases, in which collapse may occur and death ensue from suprarenal insufficiency, the suprarenal glands showing congestion and necrosis at the autopsy.

The patient rapidly becomes exhausted, and a bluish-red flush often appears on the cheeks. Moderate fever is generally present, and the temperature may rise as high as 105°. The pulse is rapid and weak.

In slight cases the acute symptoms do not last more than two or three days ; in severer cases improvement only begins after a week or fortnight, and convalescence is slow owing to the great weakness which results from the diarrhœa and toxæmia. The disease sometimes becomes chronic ; diarrhœa persists with the passage of blood and mucus or of abundant thick mucus with little or no blood, and emaciation, weakness, and anæmia become progressively greater. In most cases the patient ultimately recovers, but death may result from exhaustion.

Arthritis involving one or more of the large joints may occur. It is common in some epidemics, such as that which attacked the British troops in the South African War, but it has been comparatively rare in the present war, except in a localised epidemic in the French Army during 1916. The symptoms generally do not develop until the diarrhœa has ceased for an interval varying

between a few days and three weeks. Their severity does not vary with that of the original attack, and arthritis is not uncommon after an attack which was so slight that its nature was not recognised at the time, and the patient may not even have gone into hospital for it. The possibility of a dysenteric origin should therefore be considered in every case of arthritis when bacillary dysentery is prevalent. The knee is most often affected, but several joints may be involved simultaneously. There is a large effusion, but less pain, tenderness, and redness than in acute rheumatism, the disease bearing more resemblance to gonoccocal arthritis. A considerable amount of muscular atrophy often occurs. Complete recovery without suppuration always takes place, but sometimes not until several months have elapsed. Fever is common, but the heart is never affected. The effusion into the joint is sterile, but contains numerous polymorphonuclear cells. I have also seen two cases in which an attack of dysentery brought on acute gout in men who were already subject to the disease. It is necessary to distinguish dysenteric arthritis from the arthritis which may follow the injection of antidysenteric serum. This is often accompanied by other symptoms of serum disease, such as urticaria and headache, and generally begins between the fifth and tenth day after injection.

An individual, who has previously been subject to hæmorrhoids, is very liable to be troubled with bleeding and prolapse after an attack of dysentery ; the possibility of this source of hæmorrhage, when the stools have become solid, should be borne in mind.

Diagnosis.—The only conclusive evidence of the presence of bacillary dysentery is the discovery in the fæces of the bacillus, with its characteristic cultural properties and its agglutination with specific immune sera. A piece of mucus free from fæces should be picked out of the stool and sent at once for examination, as the organism can be much more easily isolated from such a specimen than from the actual fæces. A definite result cannot be obtained in less than three days, but a presumptive diagnosis can often be made in thirty-six hours.

Wenyon and O'Connor have, however, pointed out that the macroscopic and still more the microscopic appearance of the stools of bacillary dysentery are so characteristic that a probable

diagnosis can be made without waiting for the bacteriological report. Glairy white or yellowish mucus streaked with bright red blood is passed alone or with a little fæcal matter between the patches of mucus. Under the microscope red corpuscles are seen scattered over a ground of pus-cells, mixed with a few larger round mononuclear cells and a still smaller number of very large cells, which have occasionally been mistaken by inexperienced observers for amœbæ. All these cells are phagocytic, and they can be found in the submucous tissue of the colon after death. The characteristic stools of the acute attack are followed by the passage of brown liquid fæces, which contain similar cells to those found in the mucus. When the stools become formed, the fæces are at first coated with mucus, which contains numerous elongated cells derived from the columnar epithelium of the mucous membrane, a few of which are sometimes present during the acute stage.

Amœbic stools are generally less copious and watery than in bacillary dysentery. The blood and mucus are generally darker, the blood being often brown or even black, and the mucus transparent and dark brown. The mucus is more often mixed with fæces, and in some cases the stools consist simply of unformed fæces impregnated with mucus. Apart from the amœbæ there is nothing characteristic about the microscopical appearance of the stools, the cells being smaller in number and more variable in character than those found in bacillary dysentery.

The blood of patients suffering from dysentery never agglutinates the bacillus before the seventh and sometimes not until the twentieth day, after which the reaction lasts for three months or more. It thus develops too late to be of much practical value, for specific treatment should be instituted at once in order to be successful. With Shiga's bacillus agglutination should be obtained in a dilution of 1 in 50, and with Flexner's with 1 in 200 or even 300 before infection can be regarded as certain; although a positive result is then of great value, a negative result is of comparatively little importance. Each strain of bacillus used for agglutination tests should be tested against normal individuals before being employed; thus the Flexner bacillus commonly used during 1916 in French laboratories was agglutinated by normal sera and still more often by sera of typhoid and paratyphoid patients up to a dilution of 1 in 300.

Bacillary dysentery differs from amœbic dysentery in the more acute onset, higher fever and severer toxæmic symptoms; tenesmus is more frequent and thickening of the colon is much less common; tenderness is less marked, and when present is generally greater on the left side than the right. Fisher lays great stress on the bluish-red flush on the cheeks in early bacillary cases, as he never saw it in amœbic dysentery. Hepatitis and hepatic abscess never occur, and local and general peritonitis are very rare. There is no leucocytosis, a greater number of leucocytes than 15,000 per c.mm. pointing strongly to amœbic dysentery.

In the epidemic in the Mediterranean Forces in 1915 the clinical picture was much confused by the frequent association of amœbic with bacillary dysentery. The possibility of such an association should be considered, whenever a case of apparently amœbic or bacillary dysentery does not respond to treatment with emetine or anti-dysenteric serum respectively. In other cases dysentery was associated with paratyphoid fever and with infective jaundice.

In rare cases the onset is so acute and the stools are so watery and profuse that the disease may be mistaken for cholera, until large quantities of mucus with or without blood are also passed and the diagnosis becomes clear. Most cases of so-called malarial dysentery are really due to mixed infection. In all but 2 of 68 cases investigated at Salonica, in which malaria was associated with hæmorrhagic diarrhœa, Graham found dysentery organisms, and microscopic examination of the stools showed none of the characteristic features of bacillary dysentery. The two exceptions were probably genuine examples of malarial colitis. In cases of this sort seen by Castellani the spleen was enlarged, and recovery followed treatment with quinine when anti-dysenteric drugs had failed.

Prognosis.—Some epidemics appear to be due to a much more virulent strain of the bacillus than others, the mortality being occasionally as high as 40 per cent. Shiga's bacillus generally gives rise to a more severe and protracted form of dysentery than Flexner's bacillus. With the treatment described below the mortality is now often no more than 2 per cent. Graham reported that in a large series of cases treated in the Toronto General Hospital at Salonica between June, 1916, and May, 1917, the

mortality was less than $\frac{1}{3}$ per cent.; in 200 severe cases, in which over 20 stools containing blood and mucus were passed per day and from which a dysentery bacillus was isolated, the death-rate was only 1 per cent.

Prophylaxis.—Vaccination against dysentery has not been much employed owing to the very severe local reaction, which follows the injection of killed cultures of the organism. The reaction is, however, said to be less severe if the bacilli are sensitised with an immune serum or treated with eusol, and it is probable that in the future a considerable degree of protection will be obtained by the use of polyvalent vaccines, made whenever possible from strains isolated in the locality in which the vaccine is to be used. Apart from this, the general sanitary measures, as described for the prophylaxis of amœbic dysentery, are the only means of preventing the spread of the disease among armies in war-time.

In extensive epidemics it is impossible to deal with the carrier problem, but in small epidemics convalescent carriers should be kept under supervision until three negative examinations of the stools have been made at intervals of a week following the last positive result. An examination of the stools for occult blood—traces which can be recognised by chemical means though they do not colour the fæces—is also useful, as it shows whether any actual ulceration is still present. The necessity for some such test, which would help to prevent a man being sent to duty before he had really recovered, was well shown in a convalescent patient, who was passing normally formed stools; as a little blood was still occasionally present, I examined him with the sigmoidoscope and found numerous small superficial and readily bleeding ulcers, the intervening mucous membrane being abnormally thick and red. Some mucus removed directly from an ulcer contained Shiga's bacillus.

Treatment.—The specific treatment of bacillary dysentery with anti-dysenteric serum instead of emetine, the saline treatment, and the local treatment with enemata require special description, but otherwise the treatment is the same as for amœbic dysentery.

As the intestinal lesions are due not to the local action of the bacilli but to the action of their toxins, which are absorbed from

the colon and cause necrosis of the mucous membrane on re-excretion, and as the general symptoms, including such complications as arthritis, are also due to the absorption of toxins, the rational treatment is to delay their absorption and get rid of them from the bowel as rapidly and completely as possible, and to neutralise them after absorption by means of specific anti-toxins. The former indication is met by treatment with saline aperients, and the latter by the use of anti-dysenteric serum.

Saline Treatment.—Saline aperients have been used for dysentery since the seventeenth century; they were successfully employed in the American Civil War and in the South African War, but were found to be of no value by many Anglo-Indian physicians. This difference of opinion is now known to be due to the fact that the treatment is only of value in bacillary dysentery,

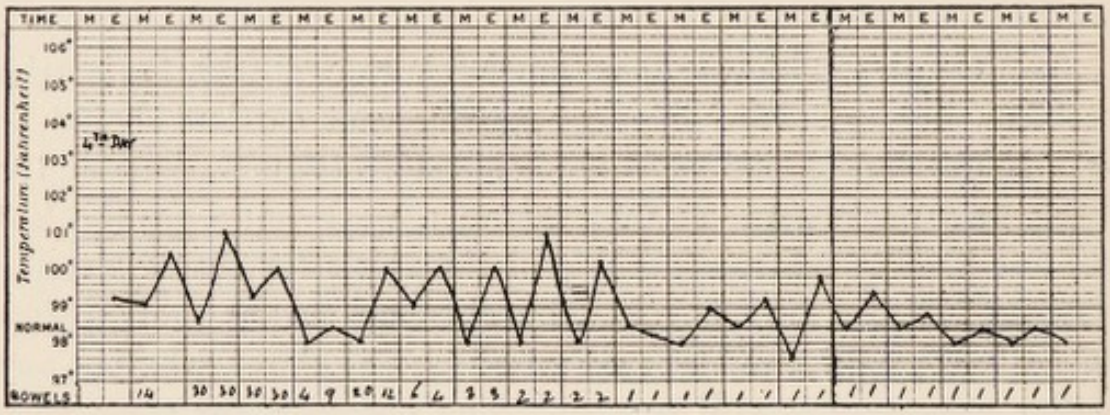


CHART I.

Bacillary dysentery treated with salts but no serum.

which is less common than amœbic dysentery in most parts of India.

Half an ounce of sodium sulphate in saturated solution should be given at the onset. This is followed by drachm doses every four hours during the first day and then every six hours until the dysenteric character of the stools disappears. If the patient is not seen on the first day the large initial dose should be omitted, and if the excessive action of the bowels has already resulted in exhaustion, no saline aperient should be given at all. This treatment not only exerts a favourable influence on the character of the stools, but it also relieves tenesmus. In mild cases the saline treatment often results in rapid recovery (Chart I.), but there is considerable risk that the patient will remain a carrier, who is

likely to have a relapse with any accidental irritation of his bowels and is at the same time a source of infection for other members of his unit. For this reason the serum should be given in mild as well as severe cases if a sufficient quantity is available.

Serum Treatment.—As the serum of horses immunised against one variety of dysentery bacillus is only useful in the treatment of dysentery due to infection with this variety, a polyvalent serum prepared by injecting horses with all available strains of the dysentery bacillus should be used; a serum of this kind is made by the Lister Institute. Its valency has recently been increased by the addition of a number of strains given me in Salonica by Captain Elworthy, and its value could be still further increased if bacteriologists would send home strains isolated in different parts of the world. In severe cases the serum should

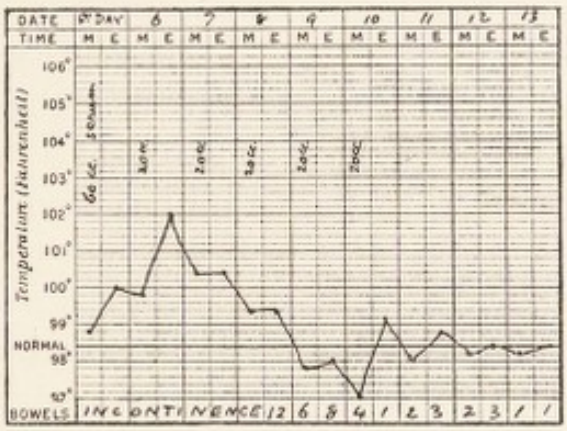


CHART II.

Bacillary dysentery treated with intravenous injections of serum.

be given as soon as the patient is seen, if the dysentery is probably bacillary in origin; in mild cases the injection may be postponed until bacteriological confirmation is obtained.

The serum should be injected intravenously, as the antitoxin is then immediately available to neutralise the toxin, whereas if subcutaneous injection is employed, a delay of some days occurs before the whole of the serum is absorbed; serum given by intramuscular injection is absorbed rather more rapidly than when given subcutaneously.

In asthmatics and in patients who have on any previous occasion received dysentery, diphtheria or tetanus antitoxin or other serum the danger of anaphylaxis can be overcome by a preliminary subcutaneous injection of $\frac{1}{10}$ c.c. of the serum half

an hour before the first large injection, and by giving the latter into a muscle instead of into a vein. The danger of anaphylaxis is not merely theoretical : in July, 1916, a man died on a hospital ship a few minutes after 20 c.c. of antidysenteric serum had been injected intravenously ; he had had three previous injections without ill effect, the last having been at Salonica 24 days earlier. He felt " funny " and became pale during the injection ; his whole body then became deeply flushed. In a few minutes the flushing was replaced by extreme cyanosis, his pulse became weaker, and he died in about a quarter of an hour.

In mild cases 40 c.c. of serum are sufficient, but in very severe cases as much as 100 c.c. should be injected at one time : 60 c.c. given on the first day, followed by 20 c.c. on the two following days, is an appropriate dose for cases of moderate severity. If necessary, however, the dose may be repeated daily for ten or more days. When hæmorrhage is severe, the serum has the additional advantage of increasing the coagulability of the blood ; no doubt it is this property which explains the good effects occasionally observed when the serum has been given in amœbic dysentery, but in such cases ordinary horse serum would be equally efficacious.

According to Rist the injection of 60 c.c. of serum causes the pain of dysenteric arthritis, which is unaffected by salicylates, to disappear in twenty-four hours, and if the injection is repeated for three or four days all symptoms disappear.

Technique of Intravenous Injection.—The serum should be warmed by standing the bottle in water at 100° F. If a deposit is present, the serum should be drawn off in such a way that the deposit is left with the last few drops in the bottle. The injection should be made with a sharp needle into one of the large veins of the elbow, after the skin has been cleaned and painted with iodine, and the veins have been made prominent by tying a bandage round the arm or by an assistant grasping the arm with his hand. The serum is drawn into the syringe, which should not be completely filled, and all air bubbles should then be removed ; the needle is inserted into the vein and a little blood is withdrawn into the syringe in order to be sure that the needle is within the vein : the bandage is then removed and the serum slowly injected. If a larger quantity has to be introduced than

the syringe can hold, it is disconnected, the needle being left in position and the finger placed upon the vein whilst an assistant refills the syringe. In severe cases a funnel instead of a syringe should be attached to the needle, and the serum, together with two or three pints of saline solution, should be run in by gravity.

Local Treatment.—Astringent and antiseptic drugs given by the mouth are of little or no use, as they become too greatly diluted by the time they reach the colon, if given in doses sufficiently small to pass through the stomach and intestines without damaging them, and when they reach the affected part of the colon they are washed along at too great a speed to have any effect. The only method of treating the bowel locally is by enemata, which are extremely valuable in the chronic colitis and chronic diarrhœa, which frequently follow acute amœbic and bacillary dysentery, especially if specific treatment has not been given sufficiently early and energetically. I have found tannic acid (gr. iv to the ounce) the most useful of the numerous drugs which have been recommended, with the exception of silver-gelatose [albargin] (gr. i to the ounce), which is, however, at present unobtainable. Injections should not be used in acute cases: quite apart from the difficulty in administering them caused by the great irritability of the rectum, which makes it impossible to retain fluid sufficiently long to be of any value, there is actual danger in severe cases; I know of one case in which the injection caused sudden violent abdominal pain owing to perforation of an ulcer, and death from general peritonitis followed three days later. One and a half pints of the fluid at 100° F. should be slowly run into the rectum from a funnel or douche-can, held not more than one and a half feet above the patient, the tube being introduced only just beyond the anal sphincter. The patient may lie on his left side, but the cæcum is reached most rapidly if he afterwards assumes the knee-elbow position for a few minutes. The injection should be given soon after the bowels have been opened, and the fluid should be retained for as nearly half an hour as possible, though it cannot as a rule be retained for more than a few minutes at first. The treatment should be repeated daily or on alternate days between three and five times; the series can, if necessary, be repeated after a week's interval.

(iii) Flagellate Diarrhœa

Lamblia intestinalis (Fig. IV., 4), *Trichomonas intestinalis* (Fig. IV., 6), and *Tetramitus mesnili* (Fig. IV., 5) are flagellate organisms, which were present in the stools of a considerable number of British soldiers, who contracted diarrhœa and dysentery in Egypt, Gallipoli, Lemnos, and Salonica, but they have also been found in men suffering from no intestinal disorder.

Lamblia inhabits the upper part of the small intestine, where it may become encysted; the cyst is passed in the fæces and may then gain access to the intestine of another individual, in which the active lamblia develops. Numerous cysts are frequently found in the stools of healthy individuals living in the tropics, so that their discovery in a case of diarrhœa or dysentery does not prove that they are the cause of the symptoms; in almost all the cases of dysentery in the Mediterranean Expeditionary Force in which they were present, the *E. histolytica* or *B. dysenteriæ* was also found when the fæces were examined during the early stages. Possibly the changes in the intestinal contents caused by dysentery favour the multiplication of lamblia. Sometimes, however, recurrent attacks of abdominal discomfort and diarrhœa with the passage of large quantities of yellowish mucus containing enormous numbers of active lamblia occur; in such cases the organism is probably the cause of the symptoms. The diarrhœa, which may persist in cases of dysentery after treatment with emetine or antidysenteric serum, is probably sometimes due to additional infection with lamblia, which is not cured by these means.

Trichomonas is an inhabitant of the colon. It has never been seen in an encysted condition and differs from other intestinal protozoa by surviving in fæces for a week or more in a spherical but unencysted form, which becomes active again on warming. Its discovery has less significance than that of lamblia. It is rarely found in any but soft or liquid stools, but its presence is probably in most cases accidental, its absence from normal fæces being due to the fact that there is no encysted form, such as that which makes the presence of a lamblia infection recognisable in the absence of diarrhœa. All the cases of dysentery in which trichomonas has been found were bacillary or amœbic, and there is no evidence that such a condition as trichomonas dysentery

exists. When, however, very large numbers are present in soft or fluid stools it may perhaps be the cause of the diarrhœa. It was found by Escomel in the drinking water during an epidemic of diarrhœa at Arequipa, a town at the foot of the Peruvian Andes, and the purification of the water was followed by the disappearance of the epidemic. It was also found in the scum which had settled on the drinking water in a tank at Lemnos in 1915.

So far as is known tetramitus is an entirely harmless parasite. It is only of importance because it has often been mistaken for trichomonas, but it has a characteristic cyst, by means of which it can be easily recognised.

Treatment.—Emetine injections and emetine-bismuth-iodide by mouth have no effect on lamblia ; turpentine (m. xv), methylene blue (gr. iii), and beta-naphthol (gr. xv) with bismuth salicylate (gr. xx), cyllin, and liquid paraffin have all been recommended for the treatment of lamblia infection, but by making more thorough examinations of the fæces for a longer period than had previously been done, Dobell and Lowe found that none of these drugs was capable of curing the infection. Wenyon and O'Connor, however, showed that gr. xx of bismuth salicylate, which was just as effective alone as combined with beta-naphthol, caused the lamblia to disappear from the stools and the diarrhœa and passage of mucus to cease, for as long as it was given three times a day. As the drug produces no ill effects, it should be given continuously for long periods in order to prevent the reappearance of the parasites. Their reappearance is probably due to their gaining access to the lumen of the tubular intestinal glands, where they are protected from the action of the bismuth salicylate.

The treatment of trichomonas is identical with that of amœbic dysentery, but emetine acts less promptly.

(iv) Ciliate Dysentery

Balantidium coli, a ciliate and much the largest intestinal protozoon, is a common inhabitant of the intestine of pigs, even in countries such as France, in which human infection is almost or quite unknown. It escapes in an encysted form and may then gain access to the human colon. It has been found in the stools

of normal individuals and in rare cases of dysentery in Russia, Norway, Sweden, and America. It was present in a few cases of dysentery occurring in British troops in Egypt in 1915. A fatal case was observed in a Serbian soldier in France, and it has also occurred among German troops. It produces lesions in the colon and liver identical with those produced by the *Entamœba histolytica*.

Symptoms.—The symptoms are similar to those of amœbic dysentery, but fewer stools are passed, and anæmia is more marked, owing to the hæmolytic action of a toxin produced by the parasite in addition to the loss of blood in the stools. There is no leucocytosis.

Treatment.—Emetine is useless, but the administration of thymol causes numerous dead balantidia to be passed, and in mild cases the symptoms may disappear. Two cachets, each containing gr. x of thymol, are given every hour for four hours; two hours after the last dose a drachm of sodium sulphate is given. Alcohol and all fat-containing food must be avoided until the aperient has acted; otherwise thymol, which is dissolved by alcohol and fat, may be absorbed and give rise to serious toxic symptoms. Rectal injections of tannic acid (gr. iv to the ounce) should also be given, as this drug destroys any balantidia which are free in the lumen of the bowel.

(v) Note on the Treatment of Diarrhœa during Dysentery Epidemics

As an attack of diarrhœa may be due to irritation of the bowels by improper food or by sand, an aperient, preferably an ounce of castor oil with Tinct. Opii mx, should be given at once: this may also get rid of the earliest infection with specific organisms. The patient should keep as quiet as possible and have the lightest and most digestible diet available. If amœbic dysentery is prevalent, a grain of emetine hydrochloride should at once be injected, even if no blood or mucus is passed, as the diarrhœa may be the first sign of amœbic infection. If improvement occurs, but the diarrhœa does not cease completely, an additional dose of half a grain should be injected on the two following days. Some opium preparation may be given if the slight diarrhœa and colic

continue, but the dose should be insufficient to cause constipation. If in spite of this treatment the diarrhœa persists for a fortnight sufficiently to cause general unfitness, or for a week if at all severe, the patient should be sent into hospital.

If at the onset of the diarrhœa or during the preliminary treatment any blood or mucus is passed, the temperature is raised to 100° or more, or tenesmus or severe colic occurs, the patient should be kept warm and at rest, and a grain of emetine should be injected when amœbic dysentery is the prevalent form, the dose being repeated daily for three days. If marked improvement does not occur, or if the symptoms are severe from the onset, the patient should be sent to hospital at once. If the prevalent type of dysentery is bacillary, and amœbic dysentery is either absent entirely or rare, no time should be lost in trying emetine, but the patient should be sent to hospital for treatment with anti-dysenteric serum after receiving a preliminary dose of half an ounce of sodium sulphate.

For efficient early treatment it is thus essential that the type of dysentery prevalent in each part of the Army should be made known as widely as possible, and that the discovery of a change in type should be promptly notified to all regimental medical officers, as well as to hospitals which have no bacteriological laboratory of their own, as it is quite impossible for pathological examinations to be made of the fæces in more than a small proportion of cases during the first few days of the illness.

(vi) Colitis and Irritability of the Colon following Dysentery

Patients who have recovered from an acute attack of dysentery frequently remain unfit for a considerable period, which may even extend to years. The symptoms are due to the chronic colitis, which may follow either amœbic or bacillary dysentery after the specific infection has died out, but the possibility that amœbic cysts or even dysentery bacilli may still be present can only be excluded by frequent expert examinations of the stools. In most cases the patient suffers from alternating attacks of constipation and diarrhœa, the latter often being brought on by aperients taken for the relief of the former, or it may follow an

indiscretion in diet or exposure to cold. During the periods of constipation, hard scybala coated with mucus are passed. The diarrhœa is accompanied by colic, which is often severe; the stinking fluid fæces contain much undigested food, often with mucus and occasionally a little bright-red blood. The diarrhœa may only last for a few hours, or it may continue for two or three days, the attacks being separated by intervals of several weeks or months. Sometimes chronic diarrhœa is present, especially after amœbic dysentery.

The patient has little appetite and cannot regain his former weight. He complains of constant abdominal discomfort. Slight tenderness is often present, especially over the iliac colon, which can generally be felt as a firmly contracted cord, which contains scybala when constipation is present. The liver is tender and may be slightly enlarged in many of the cases in which the original infection was amœbic. The tongue is dirty and the patient complains of discomfort and fulness immediately after meals. There is no fever, but the pulse is often rapid and symptoms of "soldier's heart" may be present. The patient gets quickly tired and may complain of backache. All the symptoms are aggravated by overwork. In one case the attacks of diarrhœa were immediately preceded by fainting; in another an attack of asthma, from which the patient had suffered for many years, was always a warning that diarrhœa would follow. In both cases the absorption of toxins from the fluid fæces led to symptoms before sufficient fæces had reached the rectum to produce the desire to defæcate.

Sometimes the attacks of diarrhœa cease to occur, but intractable constipation remains and the general symptoms persist, though in a lessened degree. In spite of the widespread ulceration in both forms of dysentery, and in spite of its great depth in amœbic cases, I have neither seen nor heard of any case in which the constipation was due to the development of a stricture.

Dr. T. G. Moorhead observed a number of cases in Egypt, most of which came from Gallipoli, in which severe abdominal distension developed from four to eight months after the patient had apparently recovered from an attack of dysentery. They complained of a feeling of fulness in the abdomen, with dyspnœa and general dyspeptic symptoms. The bowels were regular, and nothing

abnormal was found except enormous tympanitic distension of the abdomen, which was sometimes so great that ascites was simulated, and in two cases an attempt had been made to tap the abdomen before Moorhead saw the patients. The swelling became still more obvious when the patient stood up. Nothing abnormal except slight general stasis was found on X-ray examination. Moorhead believed that the distension was due to general intestinal atony, and corresponding with this view, the only treatment which he found of any use was abdominal massage. As no obvious dilatation was seen with the X-rays, it is also possible that the distension was not due to any change in the bowels, but to spasm of the diaphragm, which I have occasionally observed in chronic gastric ulcer and which has been found by French observers in soldiers with chronic colitis. A man invalided from Salonica with gastritis and diarrhœa developed enormous distension of his abdomen, which was the size of a seven-months' pregnancy. The X-rays showed that it was caused by spasm of the diaphragm, and it gradually became smaller as the result of vigorous manipulation and physical exercises.

Prevention and treatment.—Except in mild cases of dysentery of very short duration, it is advisable for the patient to live for some months after his recovery in a country with a warm and equable climate, where the disease is not endemic, and he should wear a cholera belt. He should take no green vegetables, except as purées, no salads nor pickles, and no fruit skins nor pips, whether raw, cooked or in jam, for at least two months after the symptoms have disappeared. As alcoholism predisposes to hepatic complications in amœbic dysentery, no alcohol should be allowed for at least six months, and then only a small quantity at lunch and dinner. Smoking is only permissible in strict moderation. The patient should eat very slowly, and attention should be paid to the teeth in order to prevent secondary infection of the bowel. There is no evidence, however, that the amœba found in many cases of pyorrhœa alveolaris has any connection with dysentery, though morphologically it resembles the *Entamœba histolytica*.

The bowels should be kept regular by sodium sulphate taken every morning or by paraffin taken twice a day, the dose being just sufficient to produce a single stool or at most two stools. Vegetable aperients must be avoided with the exception of rhubarb,

which Waddell recommend strongly for the treatment of post-dysenteric diarrhœa. Not less than 4 to 6 drachms of the tincture are given as a single dose, which is repeated once or twice after intervals of 48 hours. If the diarrhœa persists a few doses of bismuth, hydrochloric acid and opium are then generally effective.

The stools should be examined for amœbic cysts and for dysentery or paratyphoid bacilli. If cysts are found, or if the liver is tender, even in the absence of amœbic cysts, a grain of emetine hydrochloride should be injected subcutaneously every morning and a grain of emetine-bismuth-iodide should be taken by mouth every evening for ten days, or for longer if hepatic tenderness persists. In the rare cases in which dysenteric bacilli are found, 20 c.c. of antidysenteric serum should be given daily for a week, precautions being taken to avoid anaphylaxis if injections were given during the acute stage. A persistent infection with the *Bacillus dysenteriæ* or *Bacillus paratyphosus* should be treated by autogenous vaccines.

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CHAPTER XIII

AMŒBIC HEPATITIS AND HEPATIC ABSCESS

Etiology.—Amœbic hepatitis and amœbic abscesses of the liver are invariably secondary to amœbic dysentery. In 98 per cent. of fatal cases Rogers either obtained a history of dysentery or found amœbic ulcers or scars of ulcers in the large intestine. The patient had had dysentery or was still suffering from it in 72 per cent. of cases admitted into his wards with amœbic abscesses, and in an additional 14 per cent. there was a history of diarrhœa, which was doubtless a mild form of amœbic dysentery; in the remaining cases, in which no intestinal symptoms were recorded, amœbic ulcers had probably been present in the cæcum or ascending colon, situations in which they do not necessarily give rise to any symptoms. Dysentery may precede the development of an abscess by months or even years, and when hepatitis occurs during an attack of dysentery, the latter generally becomes less severe as the inflammation in the liver progresses.

Chronic alcoholism is an important predisposing cause, but it is not essential, as the disease may occur in teetotallers. Exposure to sudden changes of temperature may bring on an attack, but it is important to avoid mistaking a rigor at the onset for the cause of the illness.

Considering the large number of cases of amœbic dysentery, which occurred in the Mediterranean Expeditionary Force, remarkably few soldiers have up to now developed amœbic hepatitis or hepatic abscess. This is partly due to the thorough treatment with emetine which the majority received. But the treatment was not always continued for a sufficient period, especially at the beginning of the epidemic, and a considerable number of slight cases must have escaped treatment altogether, so that there can be little doubt that hepatitis and hepatic abscess will frequently occur

during the next few months and even during the next few years among men who have served in Gallipoli or Egypt. A visit to the military hospitals in Malta with Colonel Sir A. E. Garrod in June, 1916, convinced me that hepatitis, if systematically looked for, will be found to be a more frequent cause of chronic ill-health than has hitherto been recognised. A number of cases of amœbic abscess have occurred in French soldiers, who have never been abroad, since amœbic dysentery appeared in France in September, 1915.

Pathology.—The amœbæ, which collect in the thrombosed veins at the base of dysenteric ulcers, pass to the liver by the portal vein and give rise to thrombosis in the portal radicles. The circulation is thus obstructed and necrosis of the surrounding tissue occurs ; at the same time pus is secreted as a result of the irritant action of the amœbæ on the liver tissue. The wall of the abscess is at first formed by necrotic liver tissue, but the cavity gradually becomes limited by a fibrous capsule produced by the inflammatory reaction.

In 70 per cent. of cases a single abscess is present ; in nearly half of the other cases there are two abscesses, in a quarter there are three, and in the remainder there are four or more. A single abscess is found five times as often in the right as in the left lobe of the liver.

The pus generally contains amœbæ, but no bacteria ; in chronic cases all the amœbæ may have died, but they can still be found in the material obtained by scraping the wall of the abscess. In rare cases an amœbic abscess becomes spontaneously sterile, and its dried remains have been found at a post-mortem examination years afterwards. When an hepatic abscess reaches the surface, adhesions form between the liver and the adjoining structures, so that the contents of the abscess may burst into the lung, stomach, or bowel, or open externally without infecting the serous cavities.

Symptoms.—The symptoms vary so greatly in different cases that it is convenient to describe separately the fulminating variety, in which the whole liver is riddled with small collections of pus with no fibrous tissue separating them from the surrounding liver substance, the common subacute form, and the very chronic form, but every gradation occurs between these three types. As most

cases of the subacute type, including some with severe symptoms, subside completely with emetine injections, it is clear that in the early stages acute hepatitis without actual suppuration is present. The symptoms of subacute amœbic hepatitis and those of hepatic abscess can therefore be described together.

(i) *Fulminating Multiple Abscesses of the Liver.*—About one-tenth of the cases are of this type. There is always a definite history of dysentery, which is often still present when the hepatitis develops. The liver rapidly increases in size ; it is very painful and extremely tender. Slight jaundice may be present. The temperature is high with rapid remissions, the rise being often accompanied by rigors and the fall with copious sweating. Leucocytosis is well marked. Death generally occurs between six and eighteen days from the onset of symptoms.

(ii) *Subacute Hepatitis and Subacute Abscess of the Liver.*—Discomfort and a sense of weight are felt in the right hypochondrium in the slighter cases, but in the more severe ones the pain may be so great that the patient is hardly able to move, and he is often unable to lie on his left side owing to the dragging pain caused by the change in position. Pain may also be referred to the right shoulder and occasionally to the right arm, especially when the upper part of the liver is involved. When the left lobe is involved the pain may extend to the left side. The liver is enlarged and tender, but the rigidity of the abdominal muscles may be so great that it is impossible to feel its edge. Slight jaundice is occasionally present in the severer cases. The appetite is impaired or lost, and the patient rapidly becomes weak and emaciated.

The temperature is generally remittent, varying between 100° in the morning and 103° or 104° in the evening. In severe cases it remains high with only small remissions, but in more chronic cases, especially when the abscess bulges through the capsule of the liver and the tension within its cavity consequently falls, the fever is less marked ; it may then be low, continued, or relapsing, and may finally disappear. Copious sweating is common in the more severe cases. Moderate leucocytosis is always present, but there is less increase in the proportion of polymorphonuclear cells than in ordinary septic infections, the percentage being generally between 70 and 80.

The upper part of the liver is most frequently involved. The

main increase in dulness is then in the upward direction, but the lower border is also abnormally low. The lower ribs bulge and the intercostal spaces become wider; the skin may become œdematous, even in acute hepatitis without suppuration. At an early stage the breath sounds become feeble and the percussion note impaired at the right base; the X-rays show that the right side of the diaphragm is abnormally high, and when suppuration occurs its movements are diminished and finally cease completely. If the diaphragm is perforated by an abscess of the liver, a pulmonary abscess or less frequently an empyema develops. In the former case large quantities of thick reddish pus are expectorated, and spontaneous cure often results; the sputum differs from that brought up in other forms of pulmonary abscess in being odourless. The base of the lung becomes dull to percussion, and the vesicular murmur and voice sounds are much diminished and may disappear completely. The X-rays show a dense shadow in the lung, which cannot be clearly differentiated from the shadow of the liver.

An abscess in the anterior and lower part of the right lobe produces a tender tumour in the right side of the epigastrium; the lower ribs often become prominent and the lower border of the liver can either be felt or is found by percussion to be displaced downwards. A rub may be heard when an abscess reaches the surface of the liver, but it disappears on the formation of adhesions with the anterior abdominal wall. In advanced cases the skin becomes œdematous, fluctuation is present, and the abscess may open externally just below the costal margin. Less frequently the abscess reaches the under surface of the liver, when it may rupture into the duodenum or hepatic flexure of the colon; pus is then passed by rectum and the tumour suddenly diminishes in size; the general condition of the patient immediately improves, the temperature falls and spontaneous recovery may follow. In a case I saw at Salonica the rupture of a hitherto unsuspected abscess into the lung and intestine at the same time led to rapid recovery.

An abscess in the left lobe of the liver generally gives rise to a tender tumour in the epigastrium; if it becomes adherent to the anterior abdominal wall, definite fluctuation is obtained. An abscess bulging from the lower or upper surface of the left lobe

is less easily diagnosed ; it may escape recognition till it ruptures respectively into the stomach, when the characteristic thick reddish pus is vomited, or into the left lung or pericardium.

In some cases the X-rays show a localised increased density in the hepatic shadow, especially if the abscess is in the left lobe or if it is large and surrounded by a thick fibrous capsule. Localised subdiaphragmatic, subhepatic and retroperitoneal abscesses are uncommon complications, and general peritonitis rarely develops.

(iii) *Chronic Hepatitis and Chronic Abscess of the Liver.*—After a period of alternating diarrhœa and constipation, sometimes with a definite history of dysentery, the patient feels ill, his appetite is poor, he loses weight and strength, and his temperature may rise to about 100° every night, but I have seen several cases in which no fever occurred. The area of hepatic dulness is increased, especially upwards, and the lower border may be felt below the costal margin. There is often a dull dragging sensation in the right hypochondrium ; there may be little or no tenderness, but in cases of chronic hepatitis without any localised abscess, the whole of the liver below the costal margin is definitely tender on palpation. The blood changes and the X-ray appearances are the same as with a subacute abscess. The nature of the disease may remain unrecognised until rupture into the lung, stomach, or bowel gives rise to characteristic symptoms.

Cirrhosis of the Liver.—According to Rogers the long-continued slight amœbic infection of the liver which may result from very chronic dysentery can eventually produce cirrhosis, which is for this reason very common in Calcutta and Egypt. Soldiers, who have had amœbic dysentery and have not been treated by emetine injections with sufficient thoroughness, will therefore be particularly liable to develop cirrhosis of the liver, if they indulge too freely in alcohol. In the future it will be wise to inquire whether men suffering from cirrhosis were in the Mediterranean Expeditionary Force, and if the answer is in the affirmative a course of emetine injections should be given.

Diagnosis.—The possibility of amœbic hepatitis or hepatic abscess should be considered whenever a soldier, who has recently served in India, Egypt, or Gallipoli, is suffering from progressive deterioration in health with more or less pyrexia, especially if

the latter is remittent and accompanied by chills and sweats, and if obscure abdominal symptoms are present. A history of dysentery or simple diarrhœa, occurring in India or Egypt, or in Gallipoli during the summer and early autumn of 1915, would strengthen the suspicion, which would be further increased if no emetine or only one or two injections had been given, but the diagnosis would not be excluded in the absence of such a history. The most characteristic local symptoms are progressive enlargement of the liver, pain in the region of the right hypochondrium and in the right shoulder, and tenderness over the liver.

The condition is most commonly confused with malaria; but the rise of temperature is generally in the evening instead of during the day, and the liver is enlarged out of proportion to the spleen instead of *vice versâ*, leucocytosis is present with a relative increase of polymorphonuclear cells and only 2 to 4 per cent. large mononuclear cells, in contrast with a normal or subnormal count with 15 to 20 per cent. large mononuclear cells; the malarial plasmodium cannot be found in the blood, and emetine gives good results whilst quinine fails to influence the pyrexia.

A localised tumour in the liver in a man who has been exposed to amœbic infection is generally an amœbic abscess; it is elastic or fluctuating, unlike the hard solid tumour formed by a gumma and a growth, which moreover are unaccompanied by marked pyrexia and leucocytosis. A suppurating hydatid cyst is a much less probable diagnosis in such an individual, and in most cases eosinophilia is present, though it is absent in amœbic abscess.

Cases of basal pneumonia or pleurisy, especially on the right side, occurring in a soldier who has recently served in India or the Eastern Mediterranean, should suggest the possibility of an amœbic origin if anomalous symptoms are present, such as a gradual onset and prolonged course, absence of the characteristic rapid respiration, and irregular pyrexia with repeated chills.

Prognosis.—Until 1907 hepatic abscess was the second commonest cause of death in the British Army in India, but the incidence of the disease has become much smaller since amœbic dysentery has been treated by emetine injections, and the mortality has been greatly reduced since the necessity for operation has become comparatively rare, owing to the frequency with which

treatment is instituted in the pre-suppurative stage. The prognosis is always rendered worse if an open operation is necessary. It is best if the abscess is single, as multiple abscesses are difficult to locate and their evacuation may be impossible, especially in the rare fulminating cases. Abscesses which open into the lung, stomach, or bowel often get well spontaneously. The presence of active dysentery renders the prognosis worse, as further abscesses may develop and the patient is simultaneously weakened by the diarrhœa and loss of blood.

Treatment.—In acute hepatitis without suppuration very rapid improvement follows the subcutaneous injection of emetine hydrochloride in the same dosage as recommended for the treatment of amœbic dysentery: the hepatic tenderness diminishes within eight hours of the first injection, and the temperature may fall to normal in twenty-four hours. As there are no definite signs which distinguish hepatitis without suppuration from hepatic abscess, unless a definite tumour is present, no local treatment should be adopted until emetine injections have been given daily for a week without producing any improvement. The observations of Rogers and of Armand Delille and his colleagues prove that even a small abscess may be completely absorbed without aspiration, after the amœbæ in its walls have been killed by means of subcutaneous injections of emetine.

Hepatic abscesses were at one time often treated with a moderate degree of success by repeated aspiration. This treatment was gradually superseded by drainage after an open operation, but the results were not very good owing to the liability to secondary infection of the previously sterile abscess with pyogenic organisms. The most satisfactory treatment is that advocated by Rogers, in which the abscess cavity is evacuated by aspiration, and emetine is injected subcutaneously in order to kill the amœbæ in the abscess wall and in any ulcers which are still present in the colon. I have seen very large abscesses cured in this way after a single aspiration. If the pus reaccumulates, a grain of emetine hydrochloride dissolved in an ounce of water is injected into the abscess cavity after it has been aspirated. Unless the situation of the abscess is already evident, repeated exploration under an anæsthetic is generally necessary before aspiration can be carried out. The results with this treatment are so remarkably successful,

whilst the open operation is so frequently followed by prolonged convalescence or death, that it is astonishing to read Colonel C. H. Fagge's opinion that Rogers' treatment is out of date and only of historical interest.

Only in the comparatively rare cases, in which bacteriological examination of the pus obtained by aspiration shows that the abscess is already infected with bacteria, is there any indication for an open operation and drainage.

When an abscess has ruptured into the lung, stomach, or bowel, subcutaneous emetine injections should be continued until the temperature has remained normal for a week. Recovery generally takes place without aspiration or drainage becoming necessary.

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CHAPTER XIV

TRENCH FEVER

After all examples of the well-defined acute infections had been separated from the cases of fever occurring in the British armies in France and in Salonica, a considerable number remained in which the diagnosis was doubtful. A few were probably due to infection with the *B. paratyphosus* or even the *B. typhosus*, although all bacteriological examinations had proved negative, but I do not think that these should amount to more than 5 per cent. of the cases in which bacteriological confirmation is obtained. The large majority are most frequently diagnosed as "pyrexia of unknown origin," influenza, and rheumatic fever, the first of these being the only diagnosis which is indisputably correct. A small number of cases of true influenza with the characteristic catarrhal and general symptoms undoubtedly occur, although an attempt is rarely made to confirm the diagnosis bacteriologically; but an epidemic of very severe purulent bronchitis, which was proved to be due to the *B. influenzæ*, occurred at one of the bases in Northern France during the winter of 1916–17. Rheumatic fever is rare; Herringham only saw five cases in France between October, 1914, and October, 1915, though it subsequently became rather more common, and I only saw a single definite case in Salonica; in the few cases so diagnosed, in which the pain is actually in the joints and not in the muscles or tendons, the arthritis is more often gonococcal than rheumatic. We are thus left with a number of cases of pyrexia of unknown origin. In this chapter two varieties of a well-defined febrile disease are described, which account for a considerable proportion of these cases.

In the early summer of 1915 Major J. H. P. Graham drew attention to a type of fever occurring in the British Army in

France, in which two periods of pyrexia were separated by a normal interval. Similar cases were recognised with increasing frequency, and the disease soon became widely known as " trench fever." In November, 1915, Captain G. H. Hunt and Major A. C. Rankin described thirty cases of the same type of trench fever, and a still fuller account was published in February, 1916, by Captain J. W. McNee, Lieutenant A. Renshaw, and Captain E. H. Brunt, in which for the first time two distinct clinical types were distinguished.

The disease was only observed among officers and men living near the trenches, and in the personnel of hospitals, especially among orderlies of wards in which there were patients suffering from the disease. No cases occurred among ammunition columns, ordnance, and headquarters troops. It was for this reason that the name " trench fever " was adopted, though actual residence in the trenches themselves was certainly not an essential factor, and Hunt and McNee, in their most recent communication, state that cases have lately been met with further from the front. This was also the experience of Captain J. Muir between June, 1915, and February, 1916. Quite recently Macgregor observed a case in an orderly in a hospital in England ; he had never been abroad, but was attending cases from France.

According to General Sir Wilmot Herringham, literally thousands of cases of the first type occurred among the troops in France and Flanders between the end of April and October, 1915 ; it was comparatively rare in the following winter, but increased again in the spring of 1916. It was not recognised among French troops until May, 1916 ; since then a few cases have been described by Beauchant and by Boidin. It did not occur in Gallipoli and was not definitely recognised in Salonica until April, 1916. The second type, which was rare in France and Flanders until November, 1915, when it became more common than the first, has been very prevalent in Salonica since December, 1915. It has also occurred since May, 1916, in Mesopotamia. At first cases had to be recorded as " P.U.O." (pyrexia of unknown origin), or under the head of influenza or some other equally incorrect name, even when their nature had been recognised. For this reason it became customary to call the disease " pyrexia of unknown origin (A)," or shortly, P.U.O. (A), as this designation did not

introduce into the official records an unauthorised name, such as "periodic one day fever" or "Salonica fever," which had been used by some medical officers. The term "trench fever" was not used, as besides being an unauthorised name, the disease was common in Salonica at a time when no men were living in the trenches, and cases occurred in ammunition columns several miles from the trenches as well as in front-line troops and in hospital orderlies. When, however, in March, 1916, we recognised that the disease was identical with the long form of trench fever described by McNee, Renshaw, and Brunt, and when a month later the common short form of trench fever seen in France and Flanders became prevalent in Salonica, it seemed wise to adopt this name, which has now been officially recognised.

A few cases with symptoms intermediate between the two types of trench fever have been observed in France and in Salonica, and the initial symptoms are very similar, but the temperature chart is so different in typical cases, and the second class occurred in such large numbers without the first in the winter months, although it was very rare in comparison with the first during the summer of 1915 in France, that it cannot yet be regarded as finally proved that the two diseases are really due to the same infection, as is generally believed by British observers in France.

My attention was first drawn to the disease in Salonica at the beginning of January, 1916, by Lieut.-Colonel D. J. McGavin, Major D. S. Wylie, and Major H. T. D. Acland, of No. 1 New Zealand Stationary Hospital. At first it was not widely recognised, but as medical officers became more familiar with its characteristics, it became clear that it was extremely common, especially in certain units.

With the exception of one man, who contracted the disease whilst in hospital for another disease, all of the cases observed in Salonica up to March, 1916, belonged to two divisions which had been several months in France. None had been in Gallipoli or Serbia. The short form of trench fever had occurred in several of the affected units whilst they were in France. It is highly probable that these divisions brought the infection with them. In March, 1916, the periodic form of trench fever began to occur in a division which had been in Serbia, but not in France; some of the men had been in Gallipoli, but most had come straight to

Salonica from England. In April groups of cases of both forms of trench fever appeared in units belonging to other divisions, which had been free from the disease since their arrival at the end of 1915. The infection in the later cases was probably conveyed by men coming in drafts from France. The disease also developed among the personnel of some of the hospitals, to which the patients were sent. It has not been recognised among the French troops in Salonica.

Trench fever has occurred among Austrian troops in the Tyrol and German troops on both the eastern and western fronts; the short form was described in the *Wiener klinischer Wochenschrift* as gaiter fever, and the periodic form in the *Münchener medizinischer Wochenschrift* as five-day fever.

Pathology.—From the first all investigators agreed that trench fever was not an aberrant form of some other condition, such as paratyphoid fever, which it may closely simulate during the first pyrexial period, true relapsing fever, which it resembles in so far as the fever is of a characteristic relapsing type, and malaria, which is sometimes simulated by the shorter and sharper pyrexial attacks.

Reports have been published from time to time, in which various bacteria are said to have been isolated from the blood in cases of trench fever, but all of these observations remained unconfirmed.

McNee and Renshaw found that trench fever could be transmitted to healthy soldiers by the intramuscular and intravenous injection of the blood of men suffering from the disease. Injection of the washed red corpuscles had the same effect, but the plasma and serum were not infective. These observations, together with the striking periodic character of the fever in the long type of case and the increase in the proportion of large mononuclear leucocytes, which is often present, suggested a protozoal rather than a bacterial origin. This view seemed at first to receive support, when Riemer found a single spirochæte after prolonged search in one out of numerous preparations made from the blood during the height of the fever; he stated that he subsequently obtained cultures of the spirochæte by incubating 1 c.c. of the blood for seven days with 5 c.c. of human serum. All attempts by British observers failed, however, to confirm this discovery,

but Nankivell and Sundell have isolated a spirochæte in the urine of twelve out of eighteen cases of trench fever. As recent observations have shown how frequently spirochætes are present in the normal urethra (*vide* p. 233), and as they examined the urine in only eight control cases (none of which contained a spirochæte), much more evidence will be required before it is possible to conclude that trench fever is of spirochætal origin.

In the summer of 1917 Captain C. P. Symonds discovered what he thought might prove to be the intra-corpuscular parasite of trench fever, but circumstances prevented him from completing his investigations. His unpublished observations appeared to be confirmed by Pappenheimer, Vermilye, and Mueller of the American Medical Service, who announced in September, 1917, that they had discovered a piroplasma in the blood of 9 out of 150 patients suffering from trench fever. The supposed organism was a small, circular body, which was found lying upon the red corpuscles or free in the plasma. In a later communication, however, Pappenheimer states that further investigations proved that the body, which he and his colleagues had described, is in all probability not a living organism and is not related in any way to the production of trench fevers.

Captain Lyn Dimond has recently discovered a body, which he believed to be a hæmogregarine, in the venous blood and the blood from liver, spleen, and lung punctures in over a dozen cases of trench fever. But his detailed account of its life-history has been severely criticised by a number of protozoologists. Captain H. Henry, having examined some of Dimond's preparations in London, found that when the same technique was used in France, bodies identical with those illustrated in Dimond's first four figures were found not only in the blood of patients with trench fever, but also in that of healthy individuals and in the distilled water used for laking the blood. A scraping from the candle of a Berkefeld filter, through which some of the distilled water had been passed, yielded very active flagellates, some of which ingest bacteria and die, giving the appearance of the so-called merozoites, shown in Dimond's sixth figure. There can then be no doubt that the "hæmogregarine," which Dimond believed to be the cause of trench fever, is really a flagellate protozoon present in impure distilled water.

One attack does not seem to protect against reinfection. I saw a man with the long type of trench fever in April, 1916, in Salonica ; he had been seen by Captain McNee in a typical attack in September, 1915, while in France, and had been quite well in the interval. It is possible, however, that there was no reinfection, the original infection having remained latent between the two attacks.

Method of Propagation.—There is no nasal, pharyngeal, or bronchial catarrh, and, except for constipation, gastro-intestinal symptoms, though occasionally well marked, are generally completely absent. It is probable, therefore, that the disease is not conveyed by the respiratory secreta or by the fæces, but through the intermediation of some insect. The occurrence of the long form of the disease during the winter months shows that the infection can be conveyed in the absence of mosquitoes and other flying insects ; though Herringham found mosquitoes in France throughout the winter, there were certainly none in Salonica. Fleas have been scarce in both countries, and the men themselves rarely complain of them. Almost all patients admitted that they were lice infested up to the time of their entry into hospital, so that it is quite possible that the disease is conveyed by lice. A hospital orderly, who had been free from lice since his arrival in Salonica, had to carry the kit of a number of new patients suffering from trench fever on May 2nd, 1916. The clothes were swarming with lice, and the same evening he found some in his own clothes. He got rid of them in the course of a few days, and on May 20th an attack of trench fever began. He was not employed in the wards, he never came in contact with any patients suffering from the disease, and he was the first case of trench fever in the personnel of the hospital to which he was attached.

The incidence of trench fever is least in the cleanest battalions and the cleanest companies and platoons of battalions, and it is least in the divisions which have the best facilities for bathing. In some units a successful campaign against lice has been immediately followed by a great diminution in the incidence of trench fever, and the campaign against lice in the whole Salonica Army in the spring and early summer of 1916 was followed by the almost complete disappearance of the disease. Captain A. L. Urquhart

developed the short form of trench fever after allowing the lice from a patient with this form of the disease to bite him, and the same result was obtained more recently by Davies and Weldon in one out of two attempts. Captain McNee tells me that his observations in France have led him to agree with the conclusion I came to in Salonica—that the disease is spread by lice.

Cold, wet, and fatigue appear to be exciting causes in a man who has become infected, but has so far had no symptoms ; thus Captain Hay noticed that almost all cases in his regiment in Salonica began two or three days after they had been wet through. Captain J. Muir has laid stress upon the great importance of fatigue in rendering men more liable to infection. Statistics of the incidence of the fever in a division in France between April, 1915, and February, 1916, showed that it was always highest during a period of stress. Thus it was much greater during periods of great activity in the trenches than after withdrawal into rest areas. This was not due simply to the removal from the trenches, as on one occasion each brigade of the division underwent very strenuous training whilst out of the trenches, and this was followed in each case by a rapid increase in the number of cases observed. Captain E. R. Grieveson, however, believes that exposure to cold and wet only act indirectly by inducing men to share blankets and sleep closer together, so that infected lice pass from one to another with greater ease. Over-fatigue may also act indirectly, as official methods of destroying lice are likely to be postponed in times of stress, and the men themselves are too tired to give up the necessary hour a day to destroy their own lice.

In many instances a group of men sleeping in the same tent or dug-out have been affected. Grieveson found that all but a very few of the numerous cases occurring in his battalion could be traced to small groups of men sleeping together. Some patients appear to be carriers, who do not lose the infection completely for several months, but have recurrences from time to time, during each of which they may infect an additional number of men. A sergeant, who had been in good health whilst in France between December, 1914, and November, 1915, developed the periodic form of trench fever early in December, 1915, directly after he

left France for Salonica; in the following four months he was in hospital five times for a week or more, though he was perfectly well in the intervals. Every time he returned to his unit he became lice infested again, and he appeared to infect most of the men with whom he came in contact, about forty men of his company, including six sergeants, having been taken ill with trench fever between January and March; one of the sergeants had wrestled with him, another had danced with him, a corporal slept next to him, and a private sat next to him for some lectures.

Incubation Period.—As a result of observations in Salonica on cases arising in hospital, when a patient had been admitted for some other condition, I came to the conclusion that the incubation period is between 15 and 25 days; in the case of the hospital orderly already described it was probably 18 days, and in the experimental infection with lice observed by Davies and Weldon the incubation period was 16 days. Quite independently Hunt and McNee in France concluded that it was between 14 and 24 days, and Grieveson that it was about 14 days. The following four cases are typical of those which led to my estimate of the incubation period.

(1) Sergeant B., R.E., was admitted for rhinitis on December 27th, 1915, into a ward in which there were at the time two patients suffering from the periodic form of trench fever, no other cases of which have yet been observed in his unit. On January 1st, 1916, he was moved into another ward, in which there were and have since been no such cases. When convalescent from the rhinitis, which had been accompanied by no pyrexia or pains in the head, back, or legs, he suddenly became ill on the evening of January 24th; his temperature in the morning was normal, but at 6 p.m. it was 104°. This proved to be the first pyrexial period of a typical attack of the periodic form of trench fever. It is probable that the infection was contracted from the other cases in the ward between December 27th and January 1st, between 23 and 27 days before the onset of symptoms.

(2) Private W. went to France at the beginning of the war with the 1st —— Regiment. He was wounded in January, 1915, and was in England until the end of 1915, when he came to Salonica, joining the 2nd —— Regiment, which had come there

from France in November, on January 13th, 1916. A few days after he arrived he became lice infested. On January 21st he went to a field ambulance and then to a casualty clearing station with a hydrocele; he was transferred to a stationary hospital on February 6th. On February 12th his temperature rose and a typical attack of the periodic form of trench fever began. His clothes were disinfected when he entered the casualty clearing station, and he had no more trouble with lice after his admission there. It is probable that he contracted the disease whilst with his regiment, *i.e.* between 24 and 16 days before the onset; as he was not lice infested until he had been with his regiment some days, the period was probably about three weeks.

(3) An officer was admitted under my care for shell-shock. He began to develop a typical attack of trench fever on October 27th. A brother officer, who shared a dug-out with him, had returned to the front after an attack of trench fever about September 20th. The patient was much troubled with lice and only became free from them when he changed his clothes at a clearing station on October 3rd. He was probably infected by his brother officer, in which case the incubation period must have been at least 24 days.

(4) A fourth patient was admitted into hospital for quinsy. He was in a ward, in which there were no other cases of trench fever, but he developed the disease 14 days after admission. He had probably contracted it whilst still with his regiment, in which at least one case had already occurred, so that the incubation period was over a fortnight.

Symptoms.—The disease generally begins suddenly without any premonitory symptoms, but a feeling of malaise occasionally precedes the attack for a day or two. The patient complains of severe headache, especially frontal and behind the eyes, and this is rapidly followed by pain in the lower part of the back and on the second or third day in the legs. Pain in the neck is occasionally observed; in two cases mentioned by Hunt and McNee pain and stiffness in this region were so severe that a lumbar puncture was performed in order to exclude meningitis, and I also saw two cases in Salonica in which this was done. The patient generally shivers, but there is never a definite rigor; he is occasionally flushed and often sweats profusely. The bowels

are regular or constipated, and there is no nasal or bronchial catarrh; the appetite is lost, the tongue is moist and often slightly furred, and occasionally mild pharyngitis is present. Herpes labialis has occurred in a few cases, but less frequently in the long than in the short form of the disease. There is no rash.

The onset is sometimes extremely abrupt; the patient suddenly feels giddy, his legs give way under him and he shivers; he may be very short of breath, and occasionally complains of a pain in his left side. He has to fall out if on parade or marching, and has often great difficulty in returning to camp without assistance.

In a few cases, in which constipation is generally present, there is some abdominal pain with slight distension and tenderness, and there may be nausea and vomiting at the onset. Four out of my first fifty cases of the periodic type were sent to hospital diagnosed as appendicitis; in one a normal appendix was removed, and a second would have been operated upon had he not refused. The abdominal symptoms rapidly disappeared, and in the relapses they were less prominent than the other symptoms.

When the pain in the legs is severe, there may be some cutaneous hyperæsthesia over the shins. The shins are always tender, even if the patient complains of no pain in the legs, but tenderness appears to be most marked in groups of cases and at certain times. Graham did not mention it, and it was not observed in the earliest cases in Salonica. The pressure of puttees may be enough to cause pain, and the patient often removes them in order to relieve his discomfort. In several cases the periosteum of the tibia has seemed to me to be rough and thickened, as it pits slightly on pressure, although no pitting of the subcutaneous tissue was present. The tenderness is most marked over the lower half of the shins and may be very severe, a comparatively slight pressure causing the patient to cry out, and the pain produced may last for hours. A less degree of tenderness is often present in the tendons behind the knee, and occasionally in the ligamentum patellæ and along the course of the femur. In one case observed by Captain J. A. Gunn there was marked tenderness of the ulna on both sides. There is little or no tenderness of the

calves or other muscles. The knee and ankle jerks are normal, and there is no evidence of neuritis, though a considerable degree of muscular atrophy may occur.

In the first attack the spleen is sometimes palpable or is found to be enlarged on percussion, and there may be some tenderness in the left hypochondrium. Although this was certainly the case in Salonica, Herringham, Hunt, and McNee never found any splenic enlargement in the cases they observed in France in 1915; on the other hand, the committee recently appointed to investigate trench fever report that in 35 out of 91 cases seen in July, August, and September, 1917, the spleen was palpable, and Boidin observed slight enlargement in the eight cases he saw in French soldiers in 1916.

Leucocytosis is often, but not always present during the

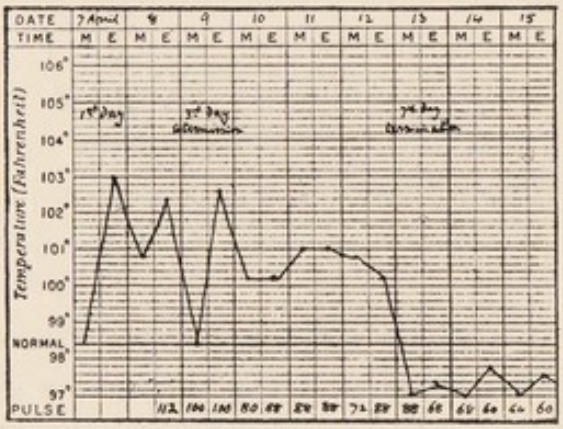

CHART III.

Short form of trench fever.

pyrexial attacks; the count varied between 4,700 and 22,000 per c.mm. in 35 cases, mostly of the short type, examined in France (Hunt and Rankin; McNee, Renshaw, and Brunt); in many of the cases examined both in France and in Salonica there was a relative increase in the large mononuclear cells (Elworthy, Urquhart). Polychromatophil cells above the normal size with well-marked punctate basophilia were observed by McNee, Renshaw, and Brunt in France; but Elworthy, working in Salonica, came to the conclusion that they only occurred in the later stages of the more severe cases. The percentage of hæmoglobin is generally about 80, though the number of red corpuscles is undiminished.

In the *short form of trench fever* the temperature rises rapidly

to between 102° and 104°, but the pulse rate is only slightly increased. On the third or fourth day the temperature suddenly falls—generally to normal or subnormal, but there is no corresponding improvement in the symptoms. After an interval of a few hours it rises again, and then after another two to five days it falls to normal; on this occasion there is immediate relief to all of the symptoms (Chart III.). In some cases the remission on the third or fourth day does not occur, the temperature remaining raised for about a week. There is often a single relapse after an interval varying from a few hours to ten days, but generally less than four days; the temperature rises to 100° or 101° for 24 or 48 hours, during which the symptoms return with diminished severity (Chart IV.). The patient is generally fit for duty almost immediately after the temperature falls again. Many cases have

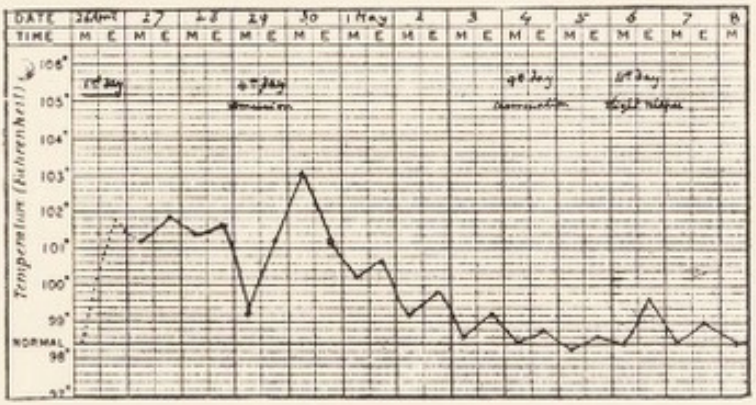

CHART IV.
Short form of trench fever.

been kept under observation by Hunt and McNee for weeks or months after the fall of temperature without any return of fever or other symptoms, so that there could be no question of additional relapses occurring after the patient had been discharged from hospital.

In the *long* or *periodic type of trench fever* the temperature rises to between 101° and 104° on the first evening. The initial attack is variable in duration; the temperature may be normal the first morning, high in the evening, normal the second morning, and rather less high the second evening than the first, after which it remains down. In other cases the first attack may last as long as four or five days, the temperature being always lower in the morning than the preceding and following evening, the

highest temperature being reached on the second or third day; in one case it reached 105·8° on the third evening, though it was normal the previous and following mornings. The pulse is generally accelerated in proportion with the temperature, but at first it may be considerably faster. With the fall of temperature at the end of the initial attack all the symptoms disappear and the patient is often sent back to duty.

After being well for two to ten days he complains of a return of headache and pain in the legs, which culminate at night; the temperature rises in the evening to a point which is generally a little lower than the highest temperature in the first attack. The temperature falls to normal or nearly normal the next morning, and either remains down or rises to a less extent the second evening, thereafter to remain normal. The general symptoms are much less severe than in the first attack, and the acceleration of the pulse is less marked, but the pain in the legs and tenderness of the shins are generally greater, and they may not disappear completely in the interval between the second and third attacks, though the headache, which generally remains the most prominent symptom during the attack, is never present in the apyrexial periods. The pain in the legs is sometimes extreme and may prevent sleep; in other cases it is comparatively slight and the patient looks and feels remarkably well, considering that he has a temperature of 101° or more.

Recurrences follow periodically, the maximum temperature being always reached in the evening (Chart V.). The intervals between the attacks are fairly constant in each case, but it varies in different cases between four and eight days, five being the most common interval. Each succeeding attack is generally milder than its predecessor and the temperature is rather lower, but in severe cases the patient feels weaker in the later intervals, and the pain and tenderness of the legs are more persistent. The later attacks may be of such short duration that the rise in temperature is not recorded at all if it is only taken twice a day (Chart VI.). On the afternoon and the evening of the day on which the attack is expected the temperature should therefore be taken every two hours, especially if there is any pain in the head or legs, as in most cases the patient knows from his sensations that there is going to be a relapse,

even before the temperature rises. The temperature is sometimes

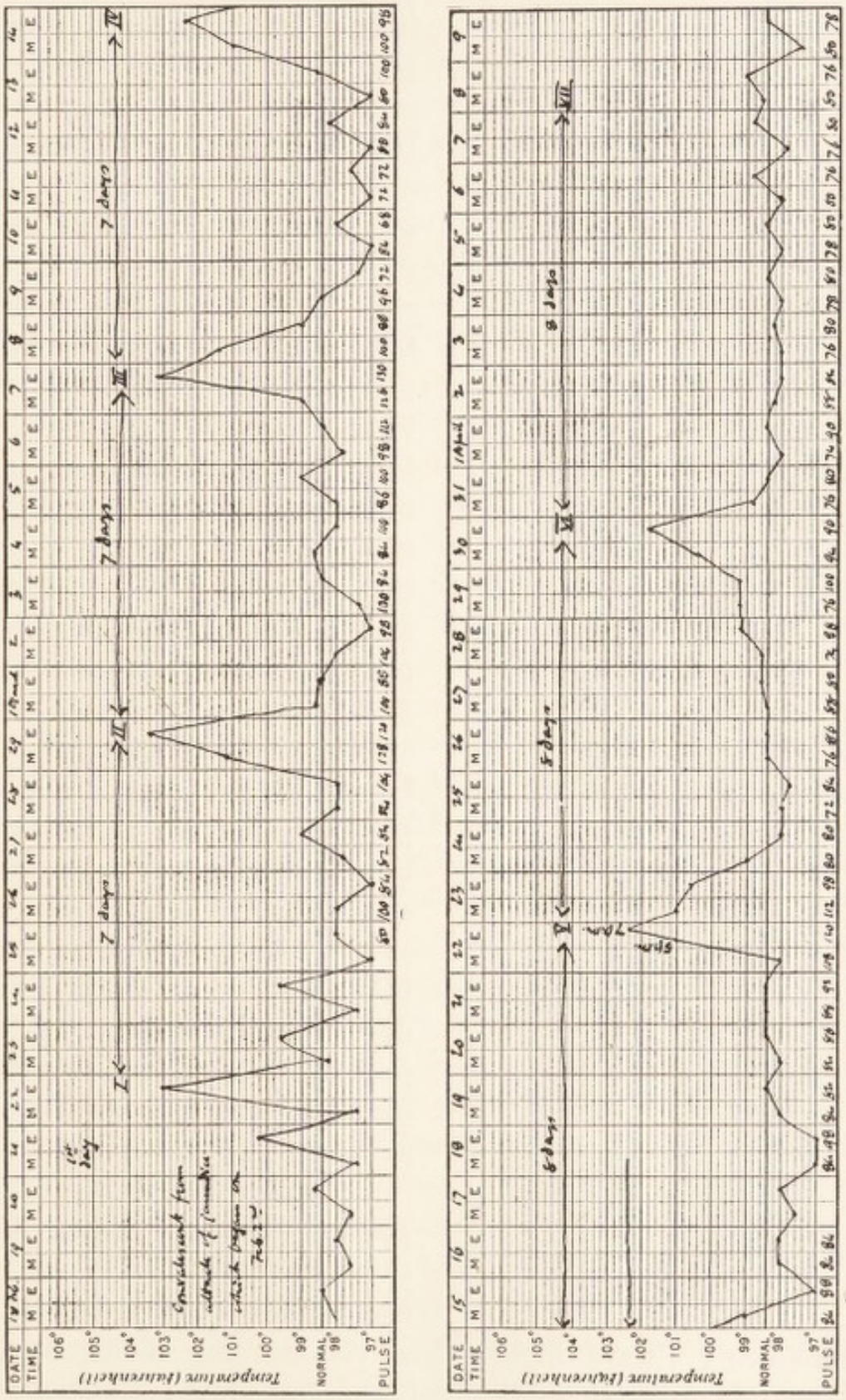

CHART V.

Periodic form of trench fever, developing whilst the patient was in hospital.

only raised for three or four hours: in one case, for example, the morning temperature was 98°; at 5.30 p.m. it was 99°, at

6.30 p.m. 100°, and at 8 p.m. 101°; at 9 and 10 p.m. and at 8 a.m. the following morning it was 98·4°. In another case it was 97·6° at 5 p.m., though the patient had had a headache since the morning, but 101·2° at 8 p.m., 102·4° at 10 p.m., 101·4° at 2 a.m., 100·2° at 6 a.m., and 98° at 8 a.m., so the morning and evening chart showed no rise, as the temperature in the ward was taken at 8 a.m. and 5 p.m. This liability for the rise in temperature to escape recognition accounts for the fact that it may appear from the chart that an attack has been missed, the interval between two of the later attacks being double that between the earlier ones: a headache may have been felt and a rise in the pulse rate recorded halfway between the attacks. In one case the third relapse was of exceptional duration and severity; this may have been due to a relapse having been really missed, as the apyrexial period which preceded it was of double length.

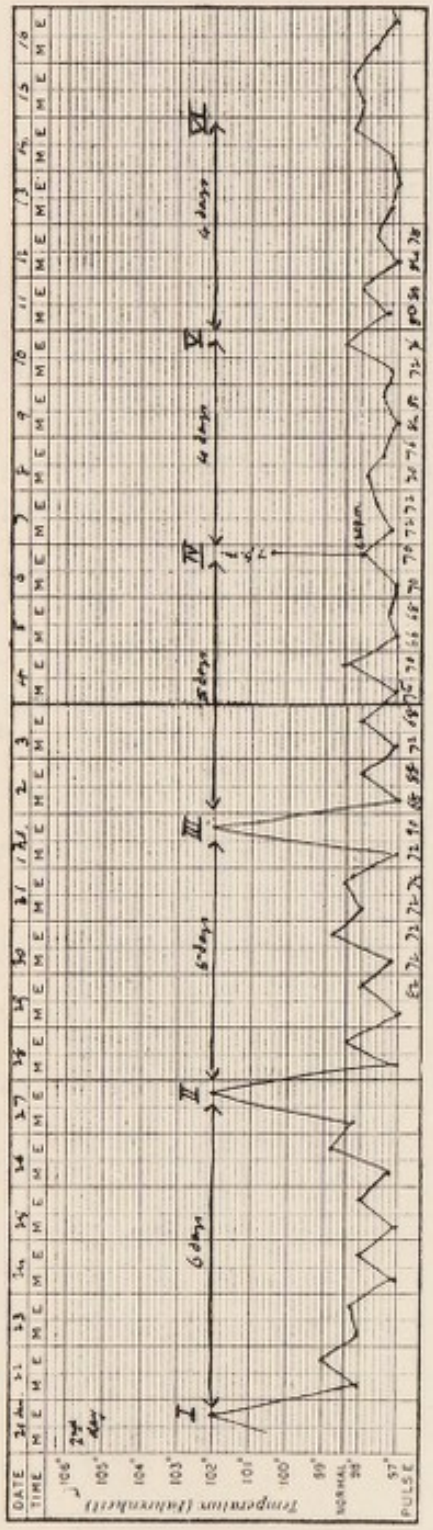

CHART VI.

Periodic form of trench fever; the fourth rise of temperature only occurred after the evening temperature had been recorded.

In a few cases the temperature remains raised for three or even four days in each attack, the evening temperature being always higher than the morning temperature, which may be normal on the first and last days; the highest point is generally reached the second evening.

Diagnosis.—The diagnosis can only be made with certainty

from a study of the temperature chart, but the association of pyrexia with tender shins is very suggestive of trench fever already in the first attack. Painful and tender shins have, however, occasionally been observed in the Salonica Army in the apparent absence of fever, and the unsatisfactory name of "trench shin" has sometimes been used to describe such cases. It is, however, not improbable that slight initial pyrexia whilst the patient was still at duty escaped notice. Some of the cases regarded as examples of the short form of trench fever are probably really periodic cases, as there is no doubt that the later bouts of pyrexia are often entirely missed owing to the short time they last, the patient having meanwhile gone back to duty, or if in hospital the evening temperature may have been taken at 5 p.m., although the rise only began at 7 p.m. or later. Several medical officers, who were very familiar with the early stages of the disease, only recognised the periodic rise of temperature after their attention had been specially drawn to its occurrence, as their patients had returned to duty after the first or second attack and had not "gone sick" for the later and comparatively slight recurrences.

The majority of cases were at first diagnosed as *influenza*, though it was generally recognised by medical officers that they were not identical with the familiar forms of that disease. Thus there is never any nasal or bronchial catarrh, the patient rarely appears or feels seriously ill, except sometimes during the first two days of the first attack, and respiratory complications never occur. The periodic return of pain and pyrexia and the pain and tenderness of the shins are quite characteristic, and prevent confusion with influenza except at the onset.

The possibility of *malaria* must always be considered, and a blood film should be examined for the malarial plasmodium before making a definite diagnosis in cases of doubt, especially if the patient has previously had malaria, or when it is prevalent, as was the case during the summer in Salonica. The differential leucocyte count is of no assistance, as there is a relative increase in large mononuclear cells in both diseases. The longer intervals between the attacks, their invariable occurrence in the evening instead of at various times of the day, the absence of true rigors and the failure of quinine given by mouth to modify the course

of the illness are distinguishing features of trench fever. Several old soldiers at first thought that they were suffering from malaria, but they subsequently realised that the disease must be different, as they never before had had severe pain and tenderness in their shins.

A few cases of true *relapsing fever* occurred in British as well as Indian troops at Gallipoli. The disease was actually first described by Hippocrates in the Greek island of Thasos, and it has been endemic in the neighbourhood of Salonica since it was introduced during the Balkan Wars. It was common in the Serbian Army in 1915, and sporadic cases have occurred ever since. Portaculis has recently described an epidemic of about 800 cases in the Greek National Army, and a few cases have occurred among British and French soldiers in Salonica. Buchanan reported a formidable epidemic among the Egyptian labour corps in April, 1916; 200 British troops in Egypt were attacked at the same time. The disease has been remarkably mild, the mortality being only about 2 per cent. The initial pyrexial period was generally longer than in trench fever, varying between five and seven days, and the maximum temperature was often over 104°. In a large majority of cases only a single relapse, lasting between three and five days, occurred after an interval of from eight to ten days. Headache was severe, and pain was present in the muscles and the joints of the legs, but not specially in the shins. Slight jaundice, slight enlargement of the liver, and albuminuria were often present. The spirochæte was found without difficulty in the blood during the pyrexial stage.

Prognosis.—There have been no fatal cases and the patient never appears seriously ill, except occasionally for a very short time in the first attack.

Until the commencement of the hot weather in Salonica at the end of May no complications had been observed, with the exception of phlebitis of the femoral vein in one case and slight jaundice in two cases, but the latter at any rate was probably accidental. Grieveson observed tachycardia in most of his cases in France, and with the onset of the hot weather in Salonica it was found that trench fever was often accompanied by a moderate degree of cardiac dilatation, which resulted in the development of "soldier's heart" if the patient returned to full duty too soon.

Endocarditis has never occurred. Hunt and McNee have not observed albuminuria, but Herringham found a trace of albumin, which soon disappeared, in a few cases. I saw one similar case in Salonica and another in a man invalided from France.

The total duration of the periodic type of trench fever from the onset to the end of the last attack is generally between four and six weeks, but some cases appear to abort, and in a few others attacks may recur for several months, the patient remaining quite well in the intervals. I saw a sergeant with the periodic form of trench fever in January, 1916, in Salonica; he had had similar attacks at intervals since August, 1915, when he first became ill in France. In most cases the patient rapidly gets strong again after an attack and is generally fit for duty after the second period of pyrexia, though he may have to rest for a few hours when the later attacks occur. Sometimes, however, great exhaustion follows and convalescence is slow.

Prophylaxis.—As the disease is probably conveyed by lice, which become infected by biting a patient during an attack, every effort should be made to keep troops free from them. All cases of trench fever should either be sent to hospital or isolated, and the patient's clothes and bedding should be specially disinfected, as well as that of all men who have recently slept near him. After the initial or the second attack a man is often able to return to duty; it is very important that he should be kept under observation, and if he again becomes verminous his clothes and bedding should again be disinfected; men who are still having attacks or have recently recovered should sleep together, isolated from the other men in their unit, but there is no reason why they should not work with them.

Treatment.—No treatment has yet been found which prevents the periodic return of attacks or which is really effective in overcoming the pain. It is generally agreed that in the first attack considerable relief occurs if the tendency to constipation is prevented by aperients. Acetyl-salicylic acid is the most effective analgesic drug. Quinine given by mouth has very little effect, but in some cases I found that the subcutaneous injection of ten grains of the bihydrochloride at the height of an attack, especially if repeated in three hours, appeared to prevent the occurrence of relapses. Salvarsan and antimony have proved useless.

Numerous local applications, both hot and cold, have been used for the painful shins; some of them have appeared to do good in certain cases, but the most frequently successful seems to have been a cold compress of saturated magnesium sulphate solution, which was first recommended by Captain D. S. Harvey. In slight cases gentle massage has given temporary relief, and I have seen a number of cases in which Captain W. R. Reynell has given galvanic baths for the legs with frequent reversals of the current with unexpectedly good results. Captain A. J. H. Miles obtained excellent results with iodine and sodium salicylate ionisation for ten minutes on the first day and sodium chloride ionisation on the second day, the negative electrode being used in both cases, followed by faradisation for the muscles of the legs for the next few days. In a few cases in Salonica the periosteum was incised, but when this was done on one side only, improvement occurred with equal rapidity on the opposite side. Even if the results had been more promising, I should regard the operation as quite unjustifiable, as the pain always disappears spontaneously in the course of a few weeks and often quite rapidly, even when it is exceptionally severe.

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CHAPTER XV

PARATYPHOID FEVER

PARATYPHOID fever is a septicæmia caused by infection with the *Bacillus paratyphosus A* and *Bacillus paratyphosus B*, and in rare instances by other allied organisms. In morphological and cultural properties these bacilli closely resemble the *B. typhosus*, but they differ so decidedly from the latter and from each other in their reactions with certain media and in their behaviour with specific immune sera that they must be regarded as distinct and specific organisms.

Paratyphoid fever was first recognised clinically by Achard and Bensaude of Paris in 1896. Though paratyphoid B is probably more common in England than has hitherto been supposed, it is very rare compared with typhoid fever among the uninoculated general public, and it has never been observed in India; its chief source in France appears to have been the civil population close to the front and especially the Belgian refugees. Some French observers, however, believe that the reoccupation after the battle of the Marne of territory, which had been fouled by paratyphoid carriers among the German invaders, was the primary source of the epidemic. Paratyphoid A is well known in India, though it had never been recognised in Europe until the present war; the majority of the earlier cases which occurred in France were among troops from India or men who had been attached to such troops. From its subsequent widespread distribution, however, it seems probable that the disease must have been endemic, though hitherto unrecognised, in the Mediterranean area and perhaps also in France and Flanders.

Up to November 1st, 1916, 1684 cases of typhoid fever, 2534 of paratyphoid fever, and 353 indefinite cases, making a total of 4571 cases of the enteric group, had occurred among the British

troops in France and Flanders. The contrast between this record and the 60,000 cases with 8227 deaths in the much smaller army which fought in the South African War is remarkable. The number of cases of paratyphoid B in France formed at first three-quarters but later only two-thirds of the total of A and B. The incidence of the disease steadily diminished after the first four months in spite of the increasing size of the army, and in November, 1916, there were only thirty cases of typhoid and ninety-two of paratyphoid fever in hospital in France—the lowest number since October, 1914. Paratyphoid fever was considerably more prevalent in Gallipoli and Lemnos, where it was probably ten times as common as typhoid fever; according to statistics published by Martin and Upjohn, about 5700 cases of enteric fever occurred among the 96,683 medical casualties, which occurred in the 300,000 men who fought in this area up to the middle of December, 1915, and of these about 93 per cent. were paratyphoid and only 7 per cent. typhoid fever. Paratyphoid B was the prevalent type up to the end of October, when paratyphoid A became more common, and by December it had almost entirely replaced paratyphoid B. Paratyphoid A was also very much more common than paratyphoid B in Mesopotamia and in Salonica, where the total incidence was small.

Etiology.—Infection with paratyphoid fever occurs in the same way as with typhoid fever, the primary source being a patient suffering from the disease or a carrier who has had the disease, as the *B. paratyphosus* may still be present in the fæces a year after the illness, water or food becoming contaminated by his excreta directly, or more often indirectly by flies and dust. The sputum has been found to contain the organism in cases complicated by broncho-pneumonia; it must therefore be regarded as a possible source of infection for nurses, orderlies, and other patients in the ward.

Morbid Anatomy.—There does not appear to be anything about the morbid anatomy of the paratyphoid fevers, which distinguishes them from typhoid fever or from each other. There is perhaps a tendency both in paratyphoid fever A and B for the colon to be more widely involved compared with the small intestine than in typhoid fever, as ulcers are often present from the cæcum to the splenic flexure, and I have even seen them in the rectum,

but this is not a constant feature, for in two cases the small intestine was alone infected and in a third—a case of paratyphoid A—no ulcers were present in any part of the inflamed intestines. Carles refers to six similar cases recorded by French observers, and to one in which the intestines were not even inflamed.

Symptoms.—The symptoms of paratyphoid fever A and B are so much alike that they are best described together. The incubation period is between ten and twenty days. The onset is generally more or less acute. The patient always complains of headache, often of abdominal discomfort, and sometimes of diarrhœa; the diarrhœa soon ceases, but the headache persists and pains in the back and limbs develop, so that by the fourth day he generally feels too weak and ill to continue with his duties. In some cases severe headache, abdominal pain with or without diarrhœa, and repeated shivering attacks develop so acutely that in a few hours the patient is obviously ill. Epistaxis, so slight that the patient generally does not mention it unless specially asked, was a common early symptom in France, but was comparatively rare at Lemnos and Salonica.

The headache may be sufficiently severe to prevent sleep the first few nights, after which it gradually improves. Diffuse abdominal discomfort is common; it rarely amounts to pain and generally only lasts two or three days, but in very acute cases it may be severe and accompanied by vomiting. The tongue is furred, and in severe cases it is dry and brown; if an entirely fluid diet is given, it remains coated except at the tip and edges. I have, however, generally found that if the patient takes biscuits and other dry food, which entail chewing and promote the flow of saliva, the tongue remains clean and moist throughout the illness, just as it does under similar conditions in typhoid fever. The slight diarrhœa, which occurs in more than half of the cases at the onset, is not severe, a small number of loose stools being passed for a few days, after which constipation is almost always present.

The abdomen may be normal in appearance, but it is more often full as in typhoid fever. There is generally no tenderness apart from that of the spleen. The spleen is palpable in at least two-thirds of the cases, and in my experience it is found to be

enlarged by percussion in all of the others. The enlargement is felt curiously far forward under the left rectus muscle, even when it is not felt further to the left. It is often much firmer than in typhoid fever and may remain palpable for some days after the temperature has fallen to normal. It is generally tender in the early stages and may give rise to spontaneous pain, which is increased by deep inspiration and coughing, and is occasionally so severe that the onset of pleurisy is suspected. Even when it cannot be felt, pain is produced by deep palpation under the outer part of the left costal margin. The liver is not enlarged ; the gall-bladder is occasionally tender owing to the presence of cholecystitis, which may give rise to no other symptoms.

In addition to the headache and abdominal discomfort, the patient often complains of pains all over the body, especially in the back and limbs and sometimes in the joints, which are not, however, swollen. Slight bronchitis is often present during the first ten days, and may give rise to a troublesome cough, which is also frequently a result of pharyngitis. Though at first the patient generally has the heavy, inert appearance so often seen in typhoid fever, in most cases he looks and feels comparatively well by the second week, and he almost always feels still better at the end of the third week, even if the temperature is still raised. Only in a small proportion of cases does the toxic condition with mental dulness, characteristic of severe typhoid fever, develop.

In 75 per cent. of cases spots appear between the sixth and twelfth days. They come in crops, which last three or four days and are sometimes still visible after the temperature is normal. They are often larger, more irregular in shape, more raised and of a deeper red colour than in typhoid fever, and are sometimes remarkably profuse. They may not fade completely on pressure and sometimes leave a faint pigmented mark after they disappear. They occur most frequently over the lower ribs in front, on the flanks, and on the back of the shoulders, and are often arranged in groups.

The temperature rapidly rises at the onset, usually reaching its maximum in 48 hours. The morning and evening chart is of a typically spiky character with daily variations of at least two degrees ; it is generally remittent, ranging between 99° and

102° during the second week, but it may be intermittent. Continued fever is very unusual. In many cases the temperature does not reach 103°, and it is rarely higher than 104°. The duration of fever varies between one and eight weeks; in about half of the cases it is twenty days or less (Charts VII. and VIII.). It generally terminates rapidly by lysis, which is often complete in

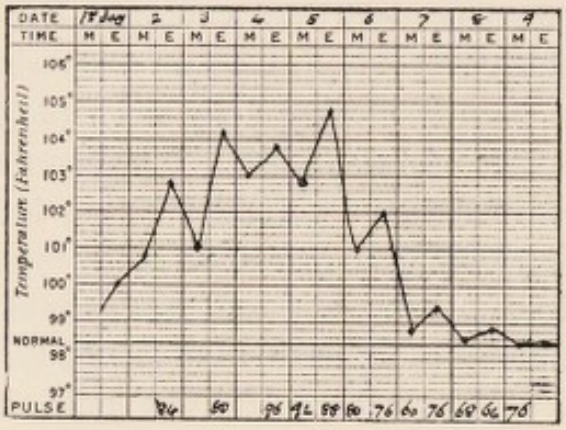

CHART VII.

Short case of paratyphoid fever A.

two days. It may be followed by a sudden rise of temperature lasting for one to three days, and this is occasionally repeated once or twice. One, and occasionally two, three, or even four true relapses, lasting six to fifteen days, with a return of pyrexia and often with splenic enlargement and a new crop of spots, occur in about 10 per cent. of cases after between two and eighteen days,

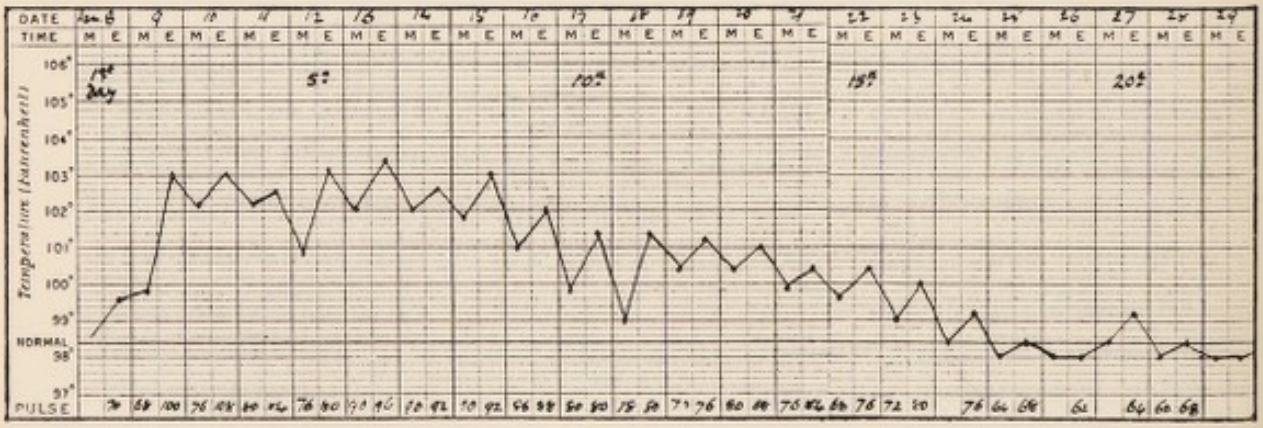

CHART VIII.

Paratyphoid fever B.

but most frequently after eight to ten days of apyrexia (Chart IX.). Rigors may be repeated throughout the illness in addition to the initial shivering attacks. Sweating is much more common than

in typhoid fever, but it generally does not begin until after the first

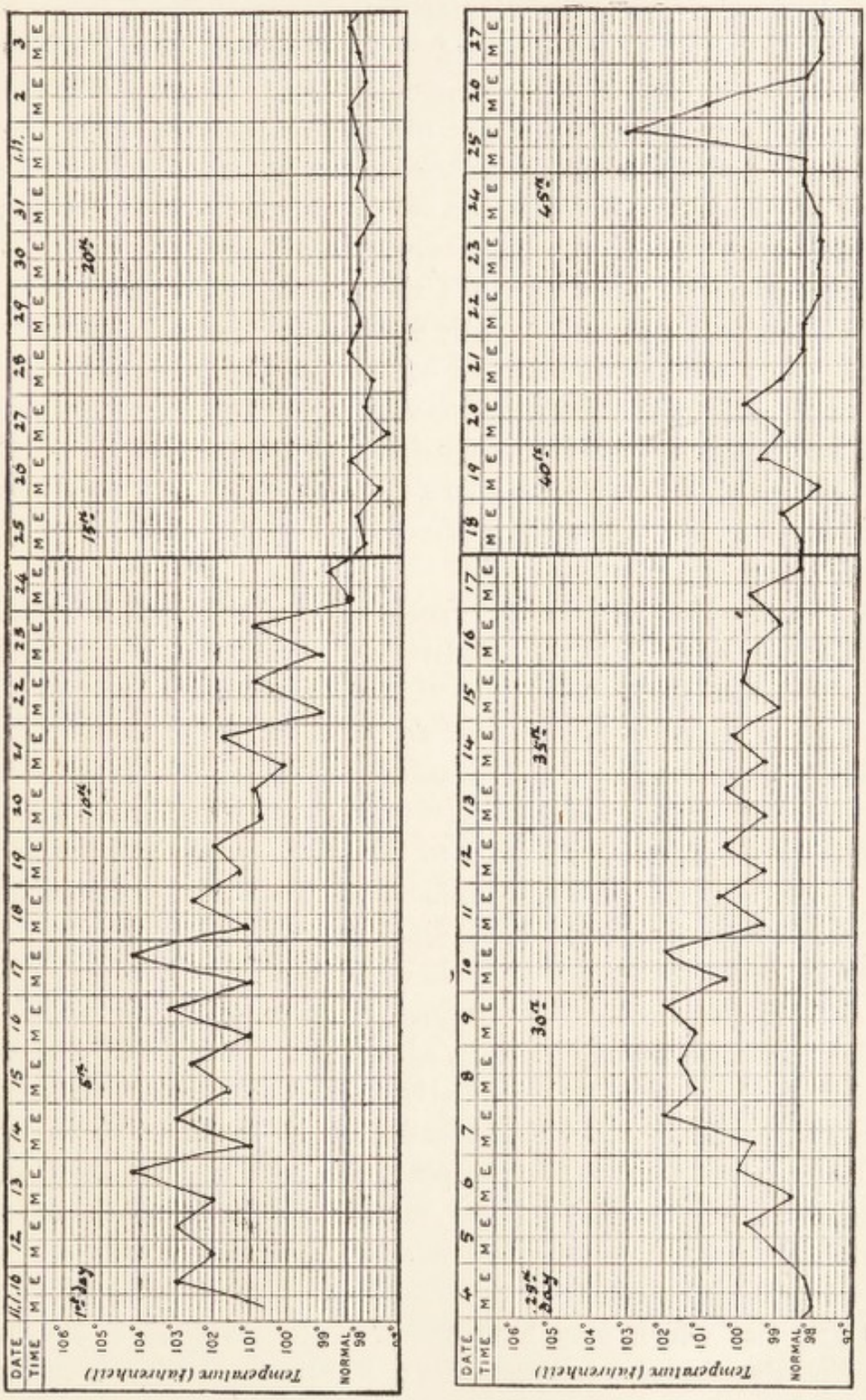

CHART IX.

Paratyphoid A with one long relapse and one short rise of temperature.

few days of fever. It may be so profuse at night that the clothes

have to be frequently changed, and sleep is consequently much disturbed.

A slow pulse is very characteristic of paratyphoid fever, a rate of 70 with a temperature of 102° being common; at the onset, however, the pulse is often quite rapid. The rate sometimes varies considerably without obvious reason, and its variations are independent of variations in temperature. The pulse is very soft and often dicrotic, the systolic blood-pressure varying between 80 and 105 mm. of mercury. Bradycardia may persist after convalescence, but it gives rise to no symptoms. In a number of cases the heart is slightly dilated with a systolic murmur at the apex; slight cyanosis and coldness of the extremities are then observed, and tachycardia may be present and may persist for a considerable period, greatly delaying convalescence. If a patient with this condition returns to duty before the poisoned myocardium has completely recovered, he is very apt to develop a "soldier's heart" (*vide* p. 282).

Marris has shown that the acceleration of the pulse, which normally follows the injection of atropine, is very much diminished in typhoid and paratyphoid fevers, but in no other condition, except for about ten weeks after antityphoid inoculation. The test is of comparatively small value when the heart is rapid and irregular, and in individuals over fifty or men suffering from cardio-sclerosis, as their reaction to the drug is less than normal even in the absence of enteric infection. The acceleration with atropine is often excessive in meningitis and is normal with all other fevers which were tested, including dysentery, malaria, spirochætal jaundice, trench fever, influenza, pneumonia, and appendicitis; it is also normal in cases of soldier's heart. The test is made with the patient lying quietly in bed. The pulse rate is taken every minute until it is steady, about ten minutes being usually required; gr. 1/33 atropine sulphate is then injected hypodermically. As the highest rate is generally attained in about half an hour, twenty-five minutes are allowed to elapse; the pulse rate is then taken again and recorded minute by minute until any rise which may have followed the injection is shown to be passing by the falling pulse; fifteen to twenty minutes may be necessary. If the difference between the average pulse rate before the injection and the highest point in the second

series of rates is more than 20 an enteric infection is very improbable ; if it is 10 or less such an infection is probably present. Readings between 10 and 20 are uncertain and necessitate further observations a few days later. The reaction is most constantly observed during the second week, but it may occur as early as the fifth day, and in relapses as late as the hundredth day, but it is never present after the temperature has fallen to normal.

Hæmorrhage occurs in paratyphoid fever less frequently than in typhoid fever ; it is very rarely severe, but fatal cases have been recorded. Perforation, which had never been observed in paratyphoid fever before the war, occurs in less than 1 per cent. of cases both of paratyphoid A and B, generally in the fourth or fifth week ; the ileum, appendix, and colon are involved with approximately equal frequency. In a third of the cases perforation is preceded by hæmorrhage. A perforation of the ileum eight inches from the ileo-cæcal sphincter was successfully sutured by Nitch in one of Miller's cases. Meteorism is very rare and only occurs in the most severe and toxic cases.

Capillary bronchitis and broncho-pneumonia are comparatively common, especially in bad weather. The latter is a serious complication and in my experience is more frequent in paratyphoid A than B ; it may develop at the onset, so that the case is at first regarded as simple pneumonia. In rare cases the broncho-pneumonia is complicated by a pulmonary abscess ; the *B. paratyphosus* has been isolated from the sputum in pure culture. Miller observed a few cases in which paratyphoid fever appeared to activate latent pulmonary tuberculosis. Pleurisy, empyema, which is generally pneumococcal, infective endocarditis, and pericarditis have been observed in rare cases. Thrombosis may occur in the femoral and saphenous veins, especially of the left side, or in the small subcutaneous branches of the internal saphenous vein over the calf. Miller found that the internal saphenous vein was hard and rigid where it passed in front of the internal malleolus in 95 per cent. of cases, although the vein over the calf was generally unaffected. A similar condition was present in only 30 per cent. of cases in the medical and 20 per cent. in the surgical wards. A case of gangrene of the foot following thrombosis probably explained the occurrence of convulsions accompanied by a rise in temperature

for two days during convalescence in one case I saw, and also the transient hemiplegia which was reported in another case. I have seen one case with mental symptoms : shortly after a profuse hæmorrhage on the twenty-eighth day of a severe attack of paratyphoid B the patient became noisy, suspicious, and depressed ; he was incontinent and difficult to wash and feed. The mental symptoms continued for four weeks after the temperature fell to normal, and then disappeared completely. Pharyngitis, tonsillitis, and simple or ulcerative laryngitis may occur. The latter may be due to perichrondritis and lead to severe dyspnœa, which is only relieved by tracheotomy. Laryngeal ulceration may produce stenosis, in which case all attempts to remove the tracheotomy tube are likely to fail. Transitory deafness is not uncommon and is probably due to mild otitis media. Captain E. A. Peters has operated on four cases of mastoid disease occurring as a complication of paratyphoid fever. In several of the Salonica cases pain was felt in the sole of the foot near the metatarso-phalangeal joints, and there was tenderness in this region, though the joints themselves were not affected ; the pain began towards the end of the illness and generally lasted about a fortnight. Miller observed several cases of arthritis, especially of the knee, two of which were followed by partial ankylosis. In several instances the joint had previously been injured. The condition often relapsed, especially if the joint was strained. In some cases true rheumatic fever complicated paratyphoid fever in men who had previously suffered from rheumatism. A man who had had rheumatic fever as a boy developed synovitis of his left hip when convalescent from paratyphoid fever B ; he was invalided from Egypt as a case of arthritis, but on arrival in England four months after his temperature had become normal the condition was found to be hysterical, fixation of the joint having apparently occurred as a result of auto-suggestion when the synovitis disappeared ; by vigorous persuasion he was made to move his hip and walk, but for a long time he continued to limp.

Slight albuminuria, which disappears when the temperature falls, is common ; occasionally true nephritis with blood and casts in the urine is present, but œdema and other renal symptoms are rarely observed. The blood disappears in a few days and the albumin does not remain much longer. In a fatal case I saw at

Salonica I found the kidneys large and congested with numerous minute abscesses and hæmorrhages under the capsule, but the renal pelvis was normal. As paratyphoid bacilli are often excreted in the urine, it is not surprising that pyelitis, cystitis, and in rare cases pyelonephritis may result. The infection generally begins about the third week, cystitis being in most cases secondary to pyelitis. Webb-Johnson believes that a renal calculus may form as a sequel of paratyphoid pyelitis, corresponding to a case recorded by Davies and Walker Hall, in which ten calculi containing the *B. typhosus* were removed from the kidney of a woman five years after an attack of typhoid fever. Orchitis is an occasional complication; the infection probably spreads from the urinary tract, which is always simultaneously infected; the pus, which generally forms, always contains the *B. paratyphosus*.

The bile is always found to contain the organism after death; this accounts for the occasional development of cholecystitis. Jaundice was a not uncommon complication in Gallipoli, where epidemic jaundice was also very prevalent; its rarity in France, where it occurred in only 1·3 per cent. of enteric cases, and in Salonica, where jaundice was much less common, makes it probable that the association was generally accidental. Suppurative pylephlebitis is a very rare complication and is generally secondary to gangrenous inflammation of the appendix. Splenic abscess is a rare complication and results from infarction; it is almost always fatal, and the specific organism can be isolated from the pus.

Suppurative parotitis was a frequent complication in Mesopotamia in the hot weather, when the vitality of the men was at a low ebb and the hospitals were understaffed. Suppurative myositis, cerebral abscess, periostitis and costal perichondritis, and suppurative peritonitis without perforation have been observed, and I have also seen an abscess of the thyroid gland and an ischio-rectal abscess; in some cases the *B. paratyphosus* has been isolated from the pus. Asthma occurred during convalescence in three of Miller's patients, although they had never before suffered in this way.

Bacteriological Diagnosis.—Although a diagnosis of "enteric fever" can be made with almost complete certainty on clinical evidence alone, the only infallible proof that typhoid or

paratyphoid fever A or B is present is the discovery in the blood, fæces, or urine of the bacillus with its characteristic cultural properties, and its property of agglutinating with the specific immune serum obtained from an animal immunised with the organism in question. In a few cases a bacillus has been isolated with all the cultural properties of *B. paratyphosus,* which does not agglutinate with the specific immune serum of either A or B, but which agglutinates with the patient's own serum; these cases are the result of infection with one or more allied organisms, which might be called *B. paratyphosus C* or *D.* As the bacillæmia is of short duration, beginning about the third day and rarely lasting more than a week, the blood should be taken for examination as soon after the third day as possible, and preferably in the evening when the temperature is generally at its highest. If a relapse occurs in a case in which the organism has not been isolated, hæmoculture should be tried again, as it may then be successful. The *B. paratyphosus* can often be found in the fæces, especially in the early stages when slight diarrhœa is so common; when hæmoculture is negative, an attempt should therefore be made to isolate the organism from the fæces, an aperient being given if constipation is present, and from the urine if this fails.

In all cases coming under observation after the sixth day, and in cases seen earlier, but in which the organism has not been isolated from the blood, the agglutinating reaction of the serum should be tested against *B. typhosus* and *B. paratyphosus A* and *B* (Widal's reaction). If agglutination occurs with a high dilution with one of the paratyphoid organisms, and either not at all or very slightly with the other and with the *B. typhosus,* no further examination is required; this is still true now that universal inoculation against paratyphoid A and B as well as typhoid fever has been adopted. Occasionally agglutination takes place in considerable dilutions, but not in slighter dilutions; thus in a case of paratyphoid B Elworthy obtained no result until a dilution of 1 in 160 was used; if only weaker dilutions had been tested the reaction would thus have been missed. In men who have been inoculated against typhoid fever, agglutination of *B. typhosus* without agglutination of either paratyphoid organism is only important if it is obtained in a dilution of at least 1 in 100. If agglutination is absent or only occurs with feeble dilutions for one

of the three organisms, or if agglutination occurs with more than one, one or more additional examinations must be made at intervals of five or six days. Infection is proved if there is a rise in agglutinating power with one of these organisms, whilst agglutination of the others is absent throughout, remains constant, or diminishes. Absence of agglutination up to the twentieth day, or slight agglutination, which remains unaltered or diminishes and is therefore probably due to previous inoculation, is evidence against infection. Elworthy found, however, that agglutination occasionally disappears and subsequently reappears in a greater dilution than before. Positive agglutination results are of more value than negative results, as in rare instances agglutination never occurs, although the clinical evidence is almost conclusively in favour of a diagnosis of paratyphoid fever; this occurs particularly in very mild cases, and in very severe cases in which there is no reaction. The test should be repeated in such cases just before the patient leaves hospital, as it occasionally becomes positive even in late convalescence; thus Elworthy did not obtain a positive Widal reaction in a mild case of paratyphoid A until fourteen days after the temperature had fallen to normal. According to Dreyer and Walker, the maximum agglutination titre of typhoid and paratyphoid infection occurs between the sixteenth and twenty-fourth day both in inoculated and uninoculated individuals. In some cases the reaction only becomes positive after a relapse. The Oxford standardised cultures and Dreyer's technique, in which the reaction to each of the three organisms is measured quantitatively two or three times at intervals of a few days, are now almost universally used, as in most cases they permit an accurate diagnosis to be made, even in individuals who have received a triple inoculation. But with this method, as in all other serological methods, a diagnosis should not be accepted unless the clinical evidence points to an infection with the enteric group of organisms.

Leucopenia is common, but is less constant than in typhoid fever, especially in the early stages and in mild cases; thus between 8000 and 14,000 leucocytes were present per c.mm. in several cases examined by Elworthy in Salonica. Complications such as broncho-pneumonia give rise to definite leucocytosis. A relative increase in lymphocytes is present in a larger proportion

of cases; Elworthy rarely found less than 36 per cent., even when the total count was as high as 14,000. Such a differential count is not of course specific, as it is quite compatible with various other infections.

Differential Diagnosis.—Paratyphoid fever differs chiefly from typhoid fever in being a much milder infection with a much smaller mortality, but these differences are not present in patients who have received anti-typhoid inoculation. The profound toxic state, which is comparatively common in unmodified typhoid fever, is only seen in exceptional cases. Whereas in typhoid fever the patient tends to become more and more toxic as the disease progresses, in paratyphoid fever he is generally at his worst at the end of the first week, and even in severe cases, which give rise to the greatest anxiety in the early stages, remarkable improvement occurs before the temperature falls, and convalescence is unexpectedly rapid. The onset is more acute, and the temperature rises more rapidly, remains raised for a shorter period, and shows greater daily variations in paratyphoid fever, and there is rarely any period of continued high fever, such as is seen in the second week of ordinary typhoid fever. Shivering at the onset is much more common in paratyphoid than in typhoid fever, and rigors in the course of the illness are not so rare. The spleen is firmer and is generally more tender, and the spots are often larger, more irregular in shape, and more raised. Lastly, paratyphoid fever is very much more common than typhoid fever in individuals who have been inoculated against the latter but not the former.

Many cases are at first diagnosed as *influenza*, especially when the onset is acute, and for a few days it is often quite impossible to distinguish between the two diseases. True influenza has, however, been comparatively rare in the present war, and catarrh of the upper respiratory passages is almost always present. Apart from the bacteriological evidence and Marris's test, leucopenia with relative lymphocytosis points to paratyphoid fever, though its absence does not exclude the diagnosis; the spleen is always either palpable or is found to be enlarged on percussion, but this is sometimes also the case in influenza. The temperature generally falls before the end of the first week in influenza, and the diagnosis is often settled about this time by the appearance of spots.

The onset of both forms of *trench fever* may simulate that of paratyphoid fever, but the course of the illness and the pain and tenderness of the shins quickly indicate the nature of the infection. The temperature chart in prolonged cases of paratyphoid fever may resemble that of *Malta fever;* in doubtful cases the diagnosis can only be settled by blood culture and agglutination reactions.

Birks, Thornley, and Fawcus have recently drawn attention to the frequency of infection with the *Micrococcus tetragenus*. They isolated it in pure culture in 25 out of 100 consecutive cases of obscure fever in France. The onset of the illness is generally acute, but may be insidious. Fever is continuous or intermittent, and its duration is very variable, but often not more than three or four days. Headache is constantly present, and the patient often complains of pain in the lumbar region and the legs. The tibia is sometimes tender, and in three cases the spleen was enlarged and some spots were present. The disease may thus closely simulate paratyphoid fever, from which it is only distinguished by bacteriological means. That the symptoms are actually due to the micrococcus is rendered probable by the fact that the patient's serum agglutinates the organism, and that mice inoculated with it die from septicæmia. Its infectivity is shown by healthy mice contracting the disease from infected mice living in the same cage, and the orderlies in hospital being liable to contract it. Less frequently similar symptoms were caused by infection with pneumococci or streptococci. French observers had already described a group of cases, clinically indistinguishable from paratyphoid or typhoid fever, in which the *Micrococcus tetragenus*, a Gram-positive diplococcus, or the *Bacillus coli* was isolated from the blood in pure culture. In other cases the *B. typhosus* or *paratyphosus* was found at the same time or at a preceding or subsequent examination, or a positive Widal reaction for the *B. typhosus* or *paratyphosus* has been obtained, although one of the former organisms was also isolated from the blood. It is probable, therefore, that these infections are in many cases secondary, the organisms perhaps being absorbed into the blood-stream from the intestinal ulcers, but this is certainly not always the case. Shearman and Moorhead observed a small epidemic in Egypt during the spring of 1916, in which the *B. fæcalis alcaligenes* was isolated from the blood in pure culture, and in which agglutination of the

bacillus occurred with the patients' sera up to a dilution of 1 in 200, but not of *B. typhosus* or *paratyphosus*, while control sera failed to agglutinate in dilutions of 1 in 50. In addition to the twenty-five cases in which the organism was isolated, agglutination was positive in a few clinically similar cases, in which blood culture was performed too late. The patients were sent to hospital as cases of enteric fever; the onset was sudden with slight chill, severe headache, pains in the limbs, nausea, and occasionally vomiting. The temperature fell by lysis after two to five days, and rose again for two or three days after two to five days. There were no symptoms apart from those of fever.

Exceptional cases of paratyphoid fever, in which the onset is very acute with diarrhœa, vomiting and abdominal pain, may closely simulate *food poisoning;* the course of the disease, the presence of spots, the enlargement of the spleen, and the bacteriological examination will settle the diagnosis. When abdominal pain and vomiting occur at the onset, especially if there is tenderness in the right iliac fossa, *appendicitis* may be simulated, but instead of the leucopenia with a relative increase of mononuclear cells seen in paratyphoid fever, leucocytosis with a relative increase of polymorphonuclear cells is present in appendicitis, and in most of these paratyphoid cases the suspicious symptoms rapidly disappear, though the temperature remains raised. As the appendix is often involved in the inflammation and ulceration of the bowel, it may give rise to peritonitis both with and without perforation. In two out of Miller's 500 cases of paratyphoid fever an acutely inflamed appendix had to be removed; in both cases the infection was due to *B. paratyphosus B.* The early joint pains are in rare cases so severe that *rheumatic fever* is suspected; the joints are, however, never swollen, and the temperature does not at once fall to normal with salicylates. In exceptional cases *dysentery, cholera,* or *meningitis* may be so closely simulated that the possibility of paratyphoid fever is not considered, until the bacteriological examination of the stools and cerebro-spinal fluid demonstrate the absence of the specific organisms of these diseases and the presence of the *B. paratyphosus.* The possibility of a double infection with dysentery and paratyphoid fever must also be remembered, as this association was not uncommon in Gallipoli.

When the initial fever is accompanied by shivering followed by profuse sweating *malaria* is closely simulated, but the temperature does not fall with the sweating. The sub-tertian form of malaria may resemble paratyphoid fever very closely. Castellani found that 90 per cent. of the cases in an epidemic of supposed typhoid fever in the Serbian Army in 1915 were really malarial. In such cases the differentiation can only be made by a blood examination, as remissions may be slight, and typical rigors, whilst not uncommon in paratyphoid fever, do not always occur in malaria. The intense headache and high fever in cases of paratyphoid fever beginning acutely often led to a diagnosis of *heat stroke* in Mesopotamia.

It is impossible to distinguish clinically between paratyphoid A and B, and the majority of the features which have at different times been said to be characteristic of one or other condition have not proved to be so on further investigation. This is particularly noticeable when the descriptions of the disease as seen in different areas of the war are compared; I have found that none of the symptoms, which have been said by observers in France to aid in the diagnosis, have been of any value in Lemnos or Salonica. It has been repeatedly stated that paratyphoid A is a much milder disease than paratyphoid B, but this has not been my experience, as many of the most severe cases as well as some of the least severe have been examples of paratyphoid A. The only symptom which has seemed to me to be generally distinctive is the very large and very hard spleen sometimes seen in paratyphoid A.

Prognosis.—The prognosis in paratyphoid fever is very much better than that in typhoid fever unmodified by previous inoculation. The mortality among 2118 bacteriologically proved cases in France up to August 25th, 1916, was only 29 or 1·32 per cent. In the same period 166 out of 1501 cases of typhoid fever died; of these 903 were inoculated and 47 died, and 508 had not been inoculated and 119 died. The mortality was probably rather higher in the Mediterranean Expeditionary Force, though not at Salonica. In Mesopotamia it was over 10 per cent., but this was largely due to the great heat in July, 1916, when 30 out of 39 deaths, mostly due to hyperpyrexia and cardiac failure, occurred in the cases seen by Batt and Feiling. Now that universal inoculation against paratyphoid as well as typhoid fever is being practised,

the severity of the disease has greatly diminished, and the mortality is likely to fall still lower.

The chief causes of death are perforation, broncho-pneumonia, profuse hæmorrhage, and toxæmia, which is also an important factor in many cases of death occurring from other causes. So far as my experience goes, and it agrees with the post-mortem records of Dawson and Whittington, the extreme end of the ileum was most severely ulcerated in cases in which toxæmia was the main or sole cause of death. The ileo-cæcal aperture was so much involved that there appeared to be some obstruction due to swelling of the mucous membrane, and to this might be added spasm or absence of the normal periodic relaxation of the ileo-cæcal sphincter, such as occurs in acute appendicitis. This would result in severe ileal stasis and consequent intestinal intoxication, which would aggravate the toxæmia due to the poisons produced by the *B. paratyphosus*. Death has also resulted from abscess of the spleen, gangrenous cholecystitis, and peritonitis without perforation. I performed the autopsy on a case of paratyphoid A at Salonica, in which death occurred from suppurative nephritis in the fifth week, when all the ulcers in the intestine had healed ; the organism was isolated from the bile obtained from the gall-bladder after death.

The proportion of severely toxic cases is small, and ambulatory cases lasting only a few days with mild pyrexia, slight headache, splenic enlargement, and perhaps spots occur, though their frequency is not known, as many doubtless escape recognition. Even in the worst cases convalescence is comparatively rapid.

The faster the pulse in relation to the temperature the more severe is the infection, a rate of over 100 after the fifth day indicating a case of some severity. Severe bronchitis, much mental clouding, and abdominal distention are the most serious symptoms. The prognosis is much better in warm than in cold weather, when the disease is very liable to be complicated by broncho-pneumonia and relapses are more common, and in very hot weather, when the patient is likely to be in a debilitated condition. In one area occupied by the French Army the mortality was 6·25 per cent. in February, 1915, but almost zero the following spring and summer.

Prophylaxis.—The prophylaxis of paratyphoid fever consists

in recognising and isolating cases as early as possible, preventing contamination of food and water with infective material, and immunising all who may be exposed to infection by anti-paratyphoid inoculation. All suspicious cases should be sent into hospital without delay; familiarity with the clinical features of the disease will lead those in charge to call in the aid of a bacteriologist at an early stage, when diagnosis by blood culture is possible. As soon as the diagnosis is made, the man's unit should be informed, so that his bedding may be disinfected and a search made for the source of infection. There is no real need to isolate cases in special hospital wards, though it may be convenient to do so, especially if inexperienced orderlies are in charge. The fæces and urine should be disinfected immediately and incinerated as quickly as possible; as the sputum may also be infective, it should be disinfected and patients should not be allowed to cough into the air but into paper handkerchiefs, which can be burnt. It is advisable to isolate convalescent patients until their stools and urine are proved to contain no paratyphoid bacilli on three consecutive occasions at intervals of a week, in order to avoid sending carriers back to their units. If it were not for this, a man who has had paratyphoid fever would often be fit for light duty a month after his temperature has fallen to normal. Mild cases, which have escaped recognition, are a far greater danger as a source of infection than convalescent carriers. Their number is likely to increase considerably now that the disease is becoming so much less severe on account of the introduction of mixed typhoid and paratyphoid inoculation.

Inoculation against typhoid fever was first advocated by Sir Almroth Wright and was used on a small scale in the South African War with promising but inconclusive results. In the following years it proved extremely effective in the Indian Army, and there is no doubt that the almost universal inoculation practised in the present war is the explanation of the very small number of cases which have been observed. The official returns given in Parliament on March 1st, 1917, showed that the admission rate for typhoid fever amongst the troops in France, who had not been inoculated, was fifteen times as great as amongst those who had been inoculated and the death rate was seventy times as high.

If a mixed typhoid and paratyphoid vaccine had been used

from the beginning of the war, there would probably have been only 75 cases of paratyphoid fever instead of 2118 among the British troops in France during the first two years of the war, or about 5 per cent. of the number of cases of typhoid fever, as this appears to be the relative frequency of the fevers in the uninoculated civil population. Since the beginning of 1916 a triple vaccine, known as T.A.B., has been the only official vaccine for the British and French Armies and has proved most efficient in preventing paratyphoid as well as typhoid fever. The vaccine consists of 500 millions *B. typhosus*, and 250 millions each of *B. paratyphosus A* and *B*, and the dose is repeated in a week.

Treatment.—Ever since a visit to America ten years ago, when I saw the remarkably good effect of a generous diet in shortening convalescence and in reducing the liability to septic complications without increasing the danger of hæmorrhage or perforation, I have treated all typhoid patients in this way; I have never had occasion to regret it, nor have I ever heard of any ill-result. It is the duty of those in charge of sick soldiers to make them fit for active service as quickly as possible, and I am quite certain that a diet consisting at least of milk puddings, custard, eggs, and bread and butter in addition to milk makes a man fit for duty very much sooner than he would be on a semi-starvation diet of milk alone. Such articles of diet are completely fluid by the time the ulcerated area of the ileum and colon is reached; on the other hand, I have several times seen curds in these parts of the bowel after the death of patients who had been on a purely milk diet. Biscuits, which the patient has to chew, are of the greatest value, as they stimulate the secretion of saliva and keep the tongue moist. I have never seen the so-called typhoid tongue in a patient treated in this way; it is simply a result of a diet which requires no chewing and which calls forth no secretion of saliva. Complications such as parotitis are directly due to a septic condition of the mouth, which can easily be prevented by good nursing and an antiseptic alkaline mouth-wash, which does not contain glycerine, if a suitable diet is given. An abundant supply of fluid should be given, and in severe toxic cases normal saline solution should be administered per rectum by the drop method, or in urgent cases by subcutaneous or intravenous injection. Tepid sponging is

very useful when the temperature is high or the patient is restless or delirious.

The only drug which is probably of any use is hexamine (urotropine). It is a powerful urinary antiseptic and is probably a biliary antiseptic as well. As the chief source of the organism in carriers is the gall-bladder, and the second most important source is the urinary tract, the number of convalescent carriers would be very greatly reduced if it were possible to keep these parts sterile by the systematic use of hexamine. The drug should be given in doses of gr. x three times a day until the patient leaves hospital. It has the additional advantage of diminishing the liability to cholecystitis and pyelitis. If diarrhœa is troublesome, the colon should be washed out with normal saline solution. In severe cases some preparation of opium may be required in addition. Captain H. F. Marris has shown that the intravenous injection of 5 to 10 oz. of 0·5 per cent. sodium citrate solution arrests the progress of thrombosis occurring in the enteric fevers and produces almost immediate relief of the pain.

The patient should not be allowed to get up even in the mildest cases until his temperature has been normal for a week. I have seen fatal perforation occur in a case of paratyphoid A just when defervescence appeared to be almost complete, and at the autopsy numerous ulcers, only a few of which showed signs of healing, were still present, so that a fall in temperature does not always indicate that the ulcers have healed. When the heart is dilated, prolonged rest in bed is necessary, and smoking should not be allowed during convalescence.

I am particularly indebted to Captain R. R. Elworthy, Pathologist to the 29th General Hospital at Salonica, for help in the description of the laboratory methods of diagnosis, which proved exceptionally successful in his hands, and to the late Major C. H. Benham and Captain W. H. Fleetwood of the same hospital for some of the clinical data.

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CHAPTER XVI

EPIDEMIC JAUNDICE

Among the 2,218,559 men in the Federal Army during the American Civil War there were 22,509 cases of jaundice with 161 deaths, and 799 cases occurred amongst 33,380 Bavarian troops stationed near Paris between February and May, 1870. In the South African War there were 5648 cases, with a very small but unknown mortality.

Jaundice was very common in the Mediterranean Expeditionary Force. The symptoms were similar to those of the catarrhal jaundice, which occurs sporadically among civilians in peace time and is not uncommon among soldiers in the field. But the disease was clearly a specific one, as shown by its epidemic nature, the frequent presence of symptoms pointing to a general infection or toxæmia, such as enlargement of the spleen and nervous symptoms, and the results of the bacteriological investigations carried out at Gallipoli, which show that it is due to a bacillary infection. The epidemic in the American, Franco-Prussian and South African wars were probably of a similar nature.

Since the spring of 1916 a variety of infective jaundice has occurred in a mildly epidemic form among British and French troops in France and Flanders and also in the German Army. It differs in etiology and symptoms from the epidemic jaundice of Gallipoli, but closely resembles the disease which every year attacks between 3000 and 4000 miners in Japan, and was formerly common in Alexandria and Smyrna, although it has not attacked British troops in the East during the present war. It is caused by a spirochætal infection, and is best described as epidemic spirochætal jaundice. It has been referred to as Weil's disease, but this name should be discarded, as a similar condition was accurately observed amongst French soldiers by

Larrey at Cairo during Napoleon's Egyptian campaign in 1800, and by numerous other French physicians before Weil's paper appeared in 1886. Thus Carville described an epidemic of jaundice which occurred in the garrison at Gaillon in 1859, and Worms described an outbreak at St. Cloud in 1865; albuminuria was present in all cases, and hæmorrhages in several, so that the infection was probably spirochætal.

(i) Bacillary Jaundice

Epidemiology.—The first outbreak of bacillary jaundice in the present war occurred in July, 1915, among the troops stationed in certain camps in Egypt. There was no jaundice at Gallipoli until the middle of August, when it suddenly appeared in a large number of different units in all parts of the Peninsula; it then rapidly spread among the men of each unit. Six weeks later cases began to arise in Lemnos and Imbros. This is most easily explained on the assumption that the infection was conveyed to different parts of the Peninsula by convalescent or contact carriers in drafts from the infected Egyptian camps, and that the subsequent spread to the islands was caused by patients coming from Gallipoli. The epidemic reached its greatest intensity in the second half of October; it then gradually declined and finally disappeared at the end of the year. Official statistics give no adequate idea of the prevalence of the disease, as large numbers of men continued at duty throughout their illness, especially in November and December; several regimental medical officers told me that at times as many as one-tenth of their men actually in the trenches were jaundiced. In one battalion there were a hundred cases during October, but only thirty-six were regarded as of sufficient severity to be sent into a Field Ambulance.

Epidemic jaundice was as frequent among the French as the British at Gallipoli, but intelligent prisoners stated that no cases occurred among the Turks, although their first line was situated only a few yards from that of the Allies, and their sanitary arrangements were infinitely less good.

Epidemic jaundice occurred, though much less frequently, among the British and French troops, who went from Gallipoli to Serbia in October, 1915, and a few cases continued to develop

among them after their return to Salonica until the beginning of 1916, when the epidemic ceased. Only a few sporadic cases of catarrhal jaundice occurred among the British troops at Salonica who had come from France or England, and there were no cases among the French who came direct from France. It did not reappear in Egypt in 1916. An outbreak affecting 550 men occurred in a single division in Mesopotamia during the very hot weather in June and July, 1916.

Pathology.—No organisms were isolated from the blood in cases of epidemic jaundice occurring among British troops, except occasionally when it was complicated by the presence of ordinary paratyphoid fever. This is probably due to the fact that the cases were only examined after the jaundice had actually appeared; at this stage the general symptoms and fever are less marked than in the pre-icteric period, and any bacillæmia which may have been present has probably disappeared. Two French observers, Sarrailhé and Clunet, who worked in an underground laboratory in a Turkish village on the Peninsula itself, obtained blood from large numbers of patients with fever, anorexia and abdominal discomfort, which often proved to be the symptoms preceding the onset of jaundice. In 112 cases—about half of the number examined—they succeeded in cultivating an atypical paratyphoid organism, which differed slightly from that of paratyphoid A and B in its cultural characters and did not agglutinate with the specific immune sera of A or B, but agglutinated with the serum obtained from the same patient or from other patients suffering from the disease. They suggested calling the organism *B. paratyphosus Dardanellensis*. In a later publication, however, they state that the cultural properties and agglutination reactions of the 94 cultures they brought home from the Dardanelles had altered when examined six months later; 51 were now typical and 33 atypical paratyphoid A, 5 typical and 1 atypical paratyphoid B, and 4 atypical typhoid. The conditions present at Gallipoli had thus resulted in a change in the properties of the paratyphoid and typhoid bacilli, associated with a change in the pathological condition they produced. But the variation was too transitory to justify the classification of the Dardanelles organism as a distinct species, and the name *B. paratyphosus Dardanellensis* should therefore be given up.

The normal duodenal contents are almost sterile, strongly alkaline, bile-stained and clear when first removed, though they become cloudy on standing. I aspirated the contents of the duodenum by means of Einhorn's evacuator * in nine cases of epidemic jaundice at Lemnos. The fluid was neutral, very slightly alkaline, or even slightly acid; it was generally turbid and contained excess of mucus and large numbers of bacteria, but no paratyphoid-like bacilli; its colour was often though not invariably paler than normal, but bile was always present in it. Lieut.-Col. C. J. Martin and Sister F. S. Williams found a non-motile member of the *B. fæcalis alcaligenes* group of organisms, which are common in the upper part of the alimentary canal, in the fluid, but it was also found in three out of four normal controls. Various other bacilli and cocci were found in one or more of the cases of jaundice and in the controls. Frugoni, Gardinghi, and Ancona have, however, succeeded in isolating the *B. paratyphosus* in the duodenal contents of 18 out of 68 cases among Italian soldiers. The gastric contents were normal in appearance, and contained few or no bacteria. In two cases in which death occurred from heart failure a few weeks after the onset of the illness Martin found evidence of hepatitis with necrosis of liver cells. In these cases the catarrh of the duodenum, bile-ducts, and gall-bladder, which is found in cases of jaundice complicating paratyphoid fever, and which is probably at first present in these cases, had disappeared. In an autopsy performed by Willcox on an uncomplicated case intense catarrh of the duodenum and larger bile ducts was present.

It is still uncertain whether the primary infection occurs in the duodenum, from which it spreads up the bile ducts and causes catarrhal cholangitis and cholecystitis, which always subside without suppuration, or whether a primary infection of the blood is followed by excretion of the organism in the bile, by means of which the ducts and the duodenum are secondarily infected. The onset of the disease with digestive symptoms before jaundice appears and before the liver and gall-bladder become enlarged and tender points to the former view as the more probable, the

* This is a small perforated metal bulb attached to a Southey's tube. The bulb is swallowed with some milk; after about 2½ hours it passes into the duodenum, the contents of which are aspirated. The tube is then partially withdrawn in order to obtain the gastric contents.

disease being due to a specific infection with a paratyphoid-like organism, which differs from the *B. paratyphosus A* and *B* in attacking the duodenum instead of the ileum and colon. The swelling of the mucous membrane of the duodenum at the mouth of the common bile duct gives rise to obstructive jaundice, but the infection may exist without any jaundice, the degree of which depends upon the anatomical relations of the ducts in each individual.

In the only autopsy seen by Rolleston on a case of jaundice from Gallipoli there was severe gastro-enteritis with a plug of tenacious mucus in the orifice of the biliary papilla.

Etiology.—The only channel by which the organism can leave an infected individual is the fæces. Infection of other individuals must be due to food and water, which have become contaminated by the fæces of patients suffering from the disease, but dust and flies are less important in this connection than in dysentery and paratyphoid fever, as the disease was most prevalent in the autumn and early winter when there were no flies and often no dust.

There is no doubt that exposure to cold and fatigue predispose to epidemic jaundice by lowering the individual's resistance, and this is probably one reason why the disease became so prevalent at Gallipoli in the last months of 1915. But these factors are not always present, as the epidemic in the Egyptian camps occurred in very hot weather, and only five out of fifty consecutive cases at Lemnos, in which special inquiry was made on this point at the end of November and beginning of December, ascribed their illness to anything of the kind.

In view of the extreme frequency of diarrhœa at Gallipoli, it is noteworthy that epidemic jaundice developed most commonly in men whose bowels had been regular for some time before the onset of symptoms, although two-thirds had suffered from diarrhœa, often on several occasions, since their arrival on the Peninsula. Indeed constipation was noted twice as often as diarrhœa as the immediate precursor of the disease.

Jaundice only became common when the incidence of dysentery was on the decline, and the occasional association was no greater than could be explained by the great frequency of both diseases. Moreover, in the majority of cases an interval, which was often

considerable, occurred between the disappearance of all dysenteric symptoms and the development of the jaundice ; in the remaining cases the onset was more or less simultaneous, or the jaundice developed with its typical premonitory symptoms at any stage during the patient's residence in hospital. The association was therefore of the same accidental nature as that with wounds, several cases of jaundice having developed in patients who were in a Lemnos hospital, in a Hospital Ship on the way to England, or even in English hospitals on account of wounds received on the Peninsula.

The association of paratyphoid fever was undoubtedly accidental when a considerable interval elapsed between the onset of the two diseases, but the discovery of the *B. paratyphosus A* or *B* in the blood during life and in the gall-bladder after death, when jaundice developed in the course of an attack of paratyphoid fever, led some observers to believe that the jaundice was then due to the paratyphoid infection, and even that the cases, in which no symptoms of paratyphoid fever were found and the organism was not isolated from the blood, were also due to a mild infection with the *B. paratyphosus A* or *B*. But the *B. paratyphosus* is constantly found in the gall-bladder of fatal cases of paratyphoid fever whether jaundice is present or not, so that its discovery in fatal cases complicated by jaundice is no proof, as has been too readily assumed, that the jaundice is due to the paratyphoid fever and is not an accidental complication. The extreme rarity of jaundice in typhoid fever, in spite of the constant infection of the bile with the *B. typhosus*, and its absence as a complication of paratyphoid fever in France and Salonica, render it very probable that the association is almost always accidental ; moreover the frequency of jaundice in paratyphoid fever at Lemnos was no greater than its frequency among patients admitted for other conditions.

Chronic indigestion sometimes preceded the onset of jaundice, but this again was no more common than would be expected from the frequency of indigestion due to indigestible food and bad teeth.

Epidemic jaundice has thus no direct causal relationship with diarrhœa, dysentery, ordinary paratyphoid fever, or chronic indigestion, though these diseases may predispose to infection by lowering the general resistance of the individual.

Symptoms.—In all but about five per cent. of cases the jaundice was preceded by other symptoms, the most frequent interval between the onset and the development of jaundice of sufficient intensity to be noticed by the patient or his companions being three days, two days being the next most common interval; in several cases it was between four and seven days, but it was rarely less than two or more than seven. When a longer interval than seven days was recorded, the onset was generally obscured by chronic indigestion which had already been present for a fortnight or more.

Pre-icteric Symptoms.—The most common early symptom is anorexia, which may develop gradually in the course of a couple of days, but is often noticed quite suddenly at one meal, generally breakfast, the previous meal having been enjoyed as usual. The anorexia quickly becomes extreme for all solid food, but the patient is generally ready to drink milk, though occasionally he may for a short time be unable to take even fluids. The mere sight of food makes him feel sick, and if he attempts to eat he is likely to vomit. The anorexia is soon followed and in a few cases is immediately preceded by a feeling of epigastric discomfort, which may develop into actual pain, which is constantly present, but is increased directly any solid food is taken. In only two cases out of a consecutive series of fifty was anorexia absent either at the onset or during the course of the illness. Contrary to what might be expected, the tongue remains clean.

Headache of moderate severity is often present, and may be accompanied by a feeling of swimming in the head and giddiness in the erect position, which is sometimes so great that the patient is hardly able to stand or walk. In many cases he feels very weak and disinclined to exert himself. These symptoms occasionally precede or develop simultaneously with the gastric symptoms, but more commonly they follow a day or two later.

In the comparatively rare instances, in which the temperature was taken before the onset of jaundice, it was often found to be raised to 100°, and occasionally even to 103° or 104°, when the headache was unusually severe with considerable constitutional disturbance, the onset simulating that of influenza or paratyphoid fever.

Symptoms after the onset of Jaundice.—The patient occasionally

notices that his urine is becoming dark, but more often the jaundice is first noticed by his friends, or by the medical officer when he reports sick on account of the premonitory symptoms. The jaundice is never very intense and may be so slight that it is not recognisable except in the conjunctivæ. Its intensity is no indication of the severity of the infection, as pronounced jaundice may occur with no other symptoms, so that one man may be able to continue at duty throughout his illness, whilst another may appear and feel very ill although the jaundice is quite slight. The infection of the bile ducts and duodenum does not necessarily produce sufficient obstruction to cause jaundice. Thus Captain Jolly saw several cases at Anzac, in which the symptoms made him expect that jaundice was about to develop; though bile appeared in the urine, no staining of the conjunctivæ or skin occurred. The jaundice reaches its greatest intensity in a few days and then slowly diminishes, a yellow tinge being often visible a fortnight after all other symptoms have disappeared.

The appetite begins to return a few days after the onset of jaundice. The epigastric pain is almost always accompanied by a moderate degree of tenderness; somewhat less severe pain is felt across the lower part of the back. Occasionally there is a vague pain in the region of the gall-bladder and liver, but never in the right shoulder. After a few days the pain disappears, slight epigastric discomfort remaining for a short time longer. The bowels tend to be constipated, and considerable relief is experienced when they have been sufficiently but not excessively opened. In exceptional cases slight diarrhœa is one of the premonitory symptoms, but it generally disappears soon after the patient becomes jaundiced. The fæces retain their normal colour in more than half of the cases; in the remainder they become pale and sometimes definitely clay-coloured a day or two after the first appearance of jaundice.

Tenderness is almost invariably most marked over the gall-bladder; deep pressure may cause little or no pain with shallow respiration, but on deep inspiration acute pain, which may radiate to the epigastrium, is felt the moment the gall-bladder comes into contact with the fingers. The gall-bladder was definitely palpable in about one-tenth of the cases. If every patient were examined regularly from the onset of the symptoms, it would

probably be felt in a considerably larger proportion of cases, which would be still greater were it not for the tendency of the rectus muscle to contract when palpation is attempted. In a few cases the gall-bladder remains enlarged when the tenderness has almost disappeared ; it can then be easily palpated.

The liver is often slightly enlarged : owing to the rigidity of the abdominal muscles this may only be recognisable by percussion, but in about a third of the cases the lower border is palpable, generally about half an inch below the costal margin in the right nipple line. The liver is firm and at first moderately tender, but the enlargement may persist after all tenderness has disappeared, occasionally for as long as fourteen or twenty days. In a small proportion of cases the spleen is enlarged and slightly tender. This must be due to a general infection secondary to that of the alimentary canal, and it is consequently most marked in severe and febrile cases, especially in the pre-icteric stage.

Bile is always present in the urine. A small quantity of albumin is sometimes found, and occasionally a few casts.

As soon as the jaundice appears the pulse becomes slow ; it is often only 60, but with the exception of one case in which it fell to 38 the day after the jaundice appeared, rising to 72 four days later, I have not seen a case in which it was below 52. The rate begins to increase again about the fifth day, and it is generally normal in ten to fourteen days. In about a quarter of the cases both sides of the heart are slightly dilated. The dilatation is occasionally considerable, especially to the right of the sternum ; the pulse is then rapid and dicrotic and the blood-pressure is low.

Even if the temperature is high in the pre-icteric period, it falls with the onset of the jaundice, and either becomes normal at once or remains between 99° and 100° for three or four days. A higher or more prolonged pyrexia indicates that some other infection is also present.

The headache and vertigo observed in the pre-icteric stage generally disappear very soon after the onset of the jaundice. The patient becomes extremely weak in severe cases. Some medical officers were impressed by the frequency during convalescence of tremor of the hands, which was generally associated with tachycardia. In all cases I saw of this kind the patient had been unfit for some time before the onset of the symptoms which

immediately preceded the jaundice; the absorption of bile salts and the rest in bed generally steadied the pulse, which became more rapid when he got up. The picture of this post-icteric tremor and tachycardia was identical with the picture seen in many exhausted soldiers from the Peninsula, who had had no jaundice, and I regard the association as purely accidental.

The severe form of peripheral neuritis, clinically indistinguishable from beri-beri, which was an occasional sequel of epidemic jaundice, is described in the next chapter. Pruritus was never observed, probably owing to the comparatively short duration of the jaundice. Rolleston saw one case in which glycosuria followed the jaundice.

In the Egyptian epidemic and in the earlier Gallipoli cases the dilatation of the heart was more constant and more considerable than in the cases which occurred at Gallipoli and Lemnos in November and December. The initial pyrexia was higher, and the enlargement of the liver was more common and more considerable. In Egypt the spleen was always considerably enlarged; it was often very tender and gave rise to spontaneous pain. This symptom was rarely so marked in Gallipoli, even in the earlier cases.

Prognosis.—Even among the severe cases in the Egyptian epidemic the prognosis was good with regard to life, no deaths having been recorded. I only heard of two Gallipoli cases, in which death occurred in the absence of some intercurrent disease, such as paratyphoid fever. In one the patient died from heart failure when convalescent from the jaundice, extreme dilatation of the heart being found post-mortem; in the other, recorded by Colonel W. H. Willcox, death occurred from toxæmia, which gave rise to delirium, vomiting, and finally coma.

In cases of moderate severity the patient is generally fit for duty between three and six weeks after the onset of symptoms, if he was previously in good health. If the patient's health was already impaired at the time of infection, as was often the case at Gallipoli, the added toxæmia is likely to weaken him to such an extent that he will require two or three months to recuperate.

The chief causes of prolonged convalescence are general asthenia, and dilatation of the heart with a tendency to dyspnœa, vertigo, and tachycardia on exertion. A man should not be sent back to

duty whilst his heart is still dilated, as otherwise he is very likely to develop a "soldier's heart" (*vide* p. 282).

Prophylaxis.—Although bacillary jaundice is not in itself a serious disease, as it is hardly ever fatal and does not necessarily prevent a man continuing at duty, it is liable to become so widely prevalent that it may form a very serious source of invaliding.

Owing to the similarity of their etiology the precautions which are necessary for the prevention of typhoid and paratyphoid fever are also applicable to epidemic jaundice. All cases should be promptly notified and sent to hospital immediately the diagnosis is made. Sisters and orderlies should be instructed to regard cases of epidemic bacillary jaundice as infective, and should take the same precautions to prevent the spread of infection and prevent contracting the disease themselves as in typhoid and paratyphoid fever.

Diagnosis.—French and Italian soldiers have taken about ¼ grm. of picric acid every ten or fifteen days in order that the yellow colour of their skin might lead to their invaliding as cases of jaundice. But the skin becomes orange-yellow with a copper reflexion instead of yellow with a greenish reflexion, and picric acid, but neither bile nor albumen, is present in the urine. The blood serum becomes deep orange owing to the presence of picramic acid, instead of golden yellow, and it contains no bile. Cases have been recorded in which the yellow colour was maintained for as long as eleven and thirteen months. The general health and digestion are unaffected. Castaigne and Desmontières found it necessary to test the blood of all jaundiced soldiers sent to Vichy: 25 c.c. of blood are shaken with 25 per cent. trichloracetic acid and filtered; if the filtrate retains its yellow colour, the latter is due to picric acid and not to bile.

The diagnosis from spirochætal jaundice is discussed after the description of the latter disease.

Treatment.—The patient should be kept warm in bed on a fluid diet until his appetite comes back and the abdominal tenderness disappears. He should then rapidly return to a full diet. A dose of calomel or castor oil should be given at the onset, but this should not be repeated unless constipation is present. It would be reasonable to give sodium salicylate (gr. 20 three or four times

a day) in order to disinfect the biliary passages, and perhaps in this way to prevent the patient becoming a convalescent carrier of the infection, but the drug does not seem to influence the course of the disease.

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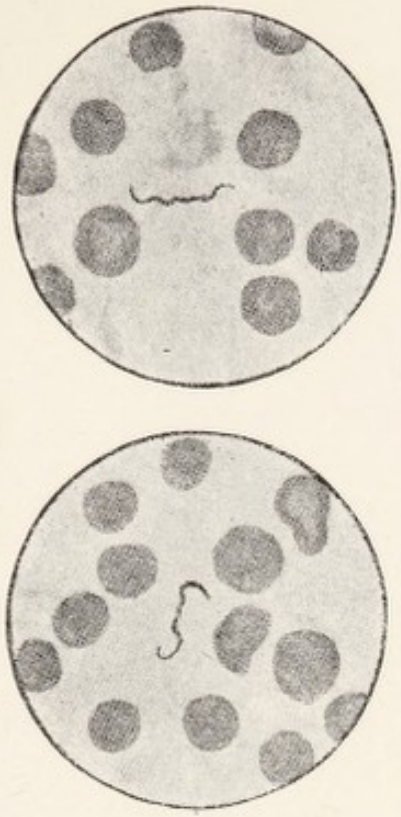
(ii) Spirochætal Jaundice

Pathology.—In November, 1914, Inada, Ido, and their fellow-workers showed that the epidemic jaundice which occurred in Japan was due to infection with a spirochæte, the *Spirochæta ictero-hæmorrhagica*, which is present in the blood during the first week and the urine during the first four weeks. Animals develop jaundice on inoculation with the blood of a patient, and the organism can be recovered from their blood and viscera. Very soon after the results of the Japanese observations became known in France, a number of investigators succeeded in isolating the organism from the blood and urine of soldiers suffering from infective jaundice, and confirmed the animal experiments (Fig. V.). This also occurred in Germany, but no acknowledgment was made of the Japanese work, the first account of which was published in January, 1915, ten months before the appearance of the first German paper.

Noguchi has since found that the strains *S. ictero-hæmorrhagica* isolated from patients in Japan and Belgium and from rats in America are identical in their morphological and serological properties. Much more difficulty has been experienced in cultivating the organism and in isolating it from the urine in cases occurring in France than in Japan, probably because the infection is much milder.

From observations made at two operations and three autopsies Dawson and Hume concluded that the jaundice is due to obstruction of the mouth of the common bile duct by œdema and



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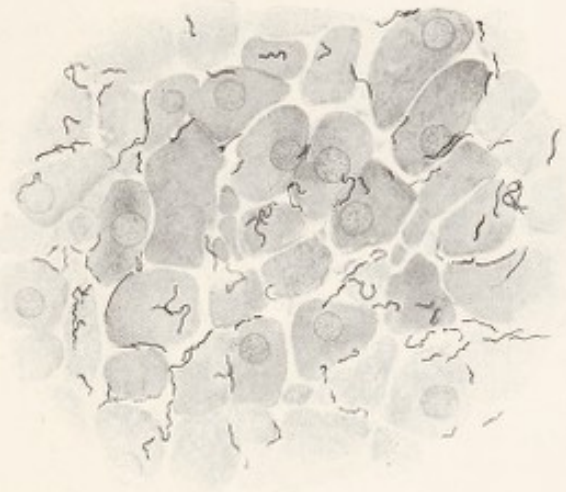

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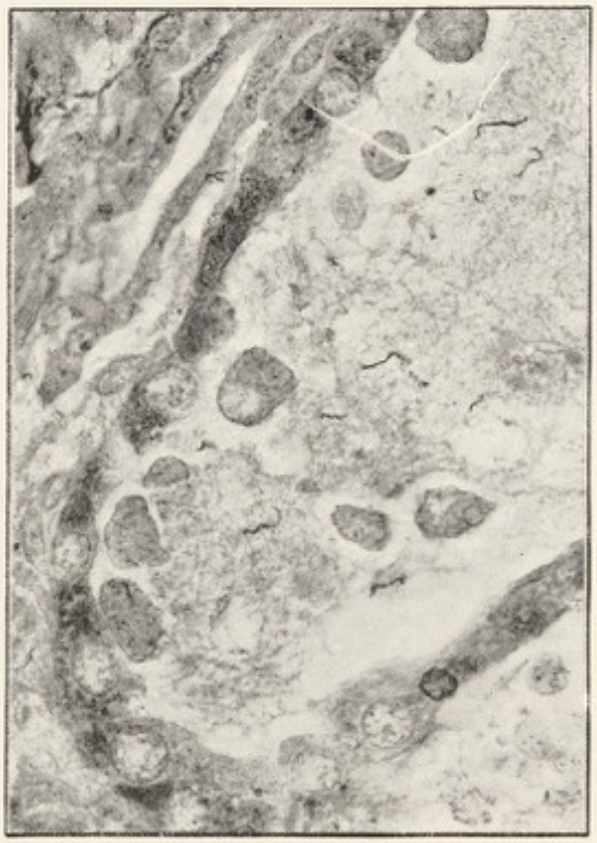

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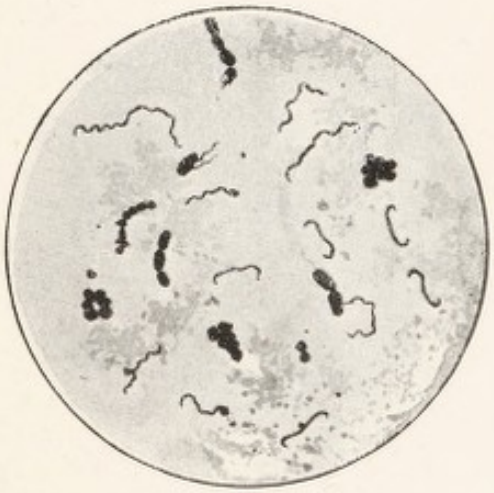

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FIG. V. SPIROCHÆTAL JAUNDICE.

(1) Spirochætes in the blood of an infected Guinea-Pig (Dawson, Hume and Bedson). (2) Drawing of liver of infected Guinea-Pig showing spirochætes. Stained by Levaditi's method. (Dawson and Hume). (3) Microphotographs of spirochætes in tubules of Guinea-Pig's kidney. Stained by Levaditi's method. (Laidlaw). (4) Field showing spirochætes and cocci in urine of patient with spirochætal jaundice. Stained by Fontana's method. (Bedson.)

congestion of the duodenal mucous membrane; the liver, gall-bladder, bile ducts, and pancreas appeared to be normal, but the bile was unusually thick. In one case a probe passed along the common duct into the duodenum dislodged a plug of mucus, which was impacted in the ampulla. Stokes, Ryle, and Tytler, however, believe that the jaundice is due to inflammatory changes in the liver causing obstruction of the smallest bile-ducts, as the interstitial tissue surrounding them was infiltrated with polymorphonuclear leucocytes, whereas the larger ducts and the duodenal mucous membrane were normal in the four autopsies they performed. The kidneys were large and showed evidence of hæmorrhagic inflammation.

Etiology.—The spirochæte was found in the kidneys and urine of wild rats in the infected areas of Japan, and Stokes discovered it in rats caught in trenches in which the disease had occurred. It has also been found in rats in districts in France where the disease is unknown and in America where it is very rare. The infection in rats is chronic, the spirochæte being excreted in the urine for long periods. The infected urine of rats is probably the source of the *S. ictero-hæmorrhagica* in man, and the urine of patients is doubtless a secondary source. In very rare cases in Japan the disease has occurred about six days after a rat-bite.

Infection can be produced experimentally through the alimentary canal and through abrasions on the surface of the body, and even through apparently healthy skin. As the organism can live for a time in water, the infection probably occurs under natural conditions through both of these channels. When several cases occur within two or three days among men living together, in whom infection by the skin can be excluded, the infection must have occurred by mouth. Cases have been observed in Japan, in which the disease began with tenderness and enlargement of the femoral glands, about a week after the corresponding foot had been wounded whilst the man was working barefoot or in sandals in stagnant water or wet earth. The incidence of the disease has greatly diminished in Japan since the mines have been thoroughly drained, and it has only occurred in France among troops who occupied certain badly drained trenches, especially during wet weather. When a unit was moved to a different part of the line

no further cases occurred, but the disease quickly appeared in the unit which replaced it in the infected trenches.

There is no evidence that mosquitoes, fleas, or lice are in any way concerned in the conveyance of the disease, and no cases have been observed in which the infection has appeared to pass direct from man to man.

Symptoms.—The incubation period is short. A colleague of Martin and Pettit, who was engaged in transmitting the infection from one guinea-pig to another on September 2nd and 4th, was seized with fever on the 10th and became jaundiced on the 16th, the spirochæte being isolated from his urine on the 17th. Carville in 1859 stated that the incubation period was six days, and in Japan it is between five and seven days.

The onset is most frequently gradual with a feeling of general unfitness, headache, pains in the back and limbs, unsteadiness or inability to stand or walk, anorexia, nausea, and often vomiting. Sometimes, however, it is sudden with shivering, faintness or giddiness, and great prostration. The temperature rises to between 102° and 105°; it varies irregularly between 100° and 103° for a week, and then descends by lysis, becoming normal between the tenth and fourteenth day. A slight return of fever often occurs a few days later; it may last for ten to fifteen days, and is generally unaccompanied by any exacerbation of symptoms.

Jaundice appears between the second and seventh day and increases up to the ninth or tenth day, when it generally fades rapidly, though occasionally it is more persistent. The degree of jaundice does not vary with the intensity of the toxæmia. Pruritus is absent or slight. In several cases symptoms exactly similar to those of spirochætal jaundice, except for the complete absence of jaundice, have been observed in patients whose blood and urine contained the spirochæte and whose blood produced spirochætal jaundice when injected into guinea-pigs. Many cases hitherto diagnosed as P.U.O. were probably of this nature.

The tongue is dry and coated with a brown fur, and sordes are present on the lips and teeth. Anorexia persists until the jaundice begins to disappear. The initial vomiting sometimes continues for several days. In severe cases there may be persistent hiccough. The patient generally complains of vague discomfort or pain over the whole abdomen, which is diffusely tender, especially

over the upper half. The liver may be slightly enlarged and tender, but the gall-bladder is not palpable and no special tenderness is present in its neighbourhood. The spleen is generally normal in size, but the lymphatic glands in the axillæ and groins are often slightly enlarged.

The patient complains of lassitude, and pains in the head, eyes, back and limbs. The pain is accompanied by tenderness, especially of the calves and back, but every muscle of the body may be involved. In severe cases the myalgia, which appears to be the most constant symptom of the infection, is extreme; it prevents sleep, and the patient feels as if he had been beaten all over and dislikes moving his limbs or turning in bed. The neck may be stiff and Kernig's sign may be present. Costa and Troisier found that the cerebro-spinal fluid in cases of this sort was under pressure, and contained excess of polymorphonuclear cells and lymphocytes with excess of albumin as well as bile. In three cases the fluid produced jaundice on injection into animals and the spirochæte was isolated from their organs; the jaundice was only just perceptible in one of these patients, and herpes was present as well as the other symptoms suggestive of cerebro-spinal meningitis. In severe cases the patient becomes drowsy and delirious.

The conjunctivæ are always injected, and the eyeballs are often tender. Herpes is common on the lips and may spread over the chin and upper part of the neck; it is generally hæmorrhagic.

Bile is present in the urine for four or five weeks in the jaundiced cases, and it may also be found in the absence of obvious jaundice. The urine almost invariably contains from a trace to a considerable quantity of albumin, often with granular and hyaline casts and a few red corpuscles. Some of the symptoms, such as the twitching and convulsions which precede the coma in fatal cases, may perhaps be uræmic in origin. When jaundice is absent, the case may be mistaken for trench nephritis, but œdema and the characteristic dyspnœa are not present. Extreme constipation is common, and the fæces are sometimes clay-coloured, but more frequently contain sufficient bile to produce a light-brown colour.

The heart is not enlarged, and the pulse is generally slow in proportion to the temperature, even when there is no jaundice. The blood-pressure never falls as it does in typhoid and paratyphoid fevers. Slight bronchitis is sometimes present, especially in

severe cases, when the sputum is generally either rusty or streaked with bright-red blood, but broncho-pneumonia and lobar pneumonia have not been observed. In addition to hæmoptysis, a little blood may be present in the vomit and rarely in the fæces. Epistaxis is an occasional symptom, and purpura may occur in severe cases. The number of red corpuscles in the blood is often reduced to 4 or 4½ millions per c.mm. and the percentage of hæmoglobin to 80 or 90. Slight polymorphonuclear leucocytosis is constantly present.

Pathological Diagnosis.—Between the fourth and ninth day the spirochæte may be found in the blood, but it is present in such small numbers that this method of diagnosis generally fails. The same drawback is present with regard to the urine, in which the organism may sometimes be found after the second week if searched for at intervals of a few days (Fig. V., 4). It is important to remember, however, that non-pathogenic spirochætes are not uncommon in the urine : thus Stoddard isolated spirochætes from the urethra in 56 per cent. of 50 soldiers suffering from various medical and surgical diseases and 22 per cent. of 50 healthy soldiers. As the incubation period in guinea-pigs is six or more days, the diagnosis cannot be confirmed by animal experiments in less than eight days. The peritoneal cavity of a guinea-pig should be inoculated with 3 to 5 c.c. of the patient's blood in the early stages of the disease. After the seventh day the centrifugalised deposit from 50 to 250 c.c. of recently passed urine suspended in 5 c.c. of normal saline may be used instead, but it gives a positive result in a smaller proportion of cases. The animal develops fever, jaundice, and hæmorrhages after six to twelve days. Death always follows. The spirochæte is easily found in the blood during life, and in the liver, kidneys, and suprarenal glands after death.

Differential Diagnosis.—Spirochætal jaundice differs from the bacillary jaundice, which occurred in Egypt and Gallipoli, in the more prolonged pyrexia, the dry furred tongue, the conjunctival congestion, the severity of the muscular pains, the character of the urine, the frequency of herpes and the greater tendency to hæmorrhages, and the absence of dilatation of the heart and of tenderness and enlargement of the gall-bladder. A definite diagnosis of spirochætal jaundice can, however, only be made by discovering

the spirochæte in the blood or urine or in the tissues of an inoculated animal.

In catarrhal jaundice the temperature is lower, the general symptoms are much less marked, and there is no albuminuria or conjunctival injection. When jaundice is due to gall-stones the local symptoms are much more prominent, and none of the characteristic general symptoms of spirochætal jaundice are present.

In the absence of jaundice the case may at first be mistaken for trench fever, but in the latter the temperature rapidly falls, the urine is normal, and the pains and tenderness are most marked over the tibiæ instead of in the muscles. In typhoid and paratyphoid fevers the spleen is generally enlarged, the urine normal, muscular pains are not a common symptom, and the conjunctivæ are not congested; in the rare cases, in which jaundice occurs as a complication of these diseases, it does not appear until after the tenth day, and the gall-bladder is then generally tender. The atropine test introduced by Marris is of great value in such cases.

Prognosis.—The mortality appears to be between 4 and 5 per cent. in France and Flanders. The disease is thus milder than the form occurring in Japan and Alexandria, in which the mortality is about 30 per cent. The difference in mortality may be due to the Japanese strain of spirochæte having acquired an increased virulence for man as a result of more frequent passage from man to man, as successive passages of the organism through guinea-pigs increases its virulence for these animals. Dawson and Hume classified 18 of their cases as severe, and 58 as mild; in the former the illness lasted about three weeks and in the latter about one, but few patients were fit for duty in less than three months.

Prophylaxis.—Wherever cases of spirochætal jaundice have occurred the ground should be disinfected or drained as thoroughly as possible, and a special effort should be made to destroy all rats. The urine of patients should be disinfected for nine weeks from the date of onset of the disease.

Treatment.—The general treatment does not differ from that of bacillary jaundice. Saline aperients and enemata are required for the constipation. Japanese observers conclude from their animal experiments that salvarsan is of little value, and Dawson

and his colleagues found it useless in their patients. Recent reports from Japan indicate that the intravenous injection of 60 c.c. of the serum of immunised horses exerts a favourable influence on the disease in man as well as animals, and promising results have been reported by Fiessinger and Leroy after intravenous and subcutaneous injection of the serum prepared by Martin and Pettit.

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CHAPTER XVII

A FORM OF BERI-BERI OCCURRING AMONG BRITISH TROOPS

In October, November, and December, 1915, a number of cases of peripheral neuritis associated with circulatory symptoms occurred at Lemnos among men invalided from Gallipoli, and at least one case occurred in a medical officer who had never been to the Peninsula. A few similar cases were subsequently observed by Major Shellshear in Egypt. Owing to the striking resemblance of the symptoms in well-marked cases to those of beri-beri, the condition was at first regarded as ordinary beri-beri and due to the diet being deficient in certain essential constituents. It was soon noticed, however, that a considerable number of cases were associated with epidemic jaundice, and an examination of seventeen cases, which included all I was able to discover in Lemnos during December, led me to the conclusion that the latter infection was as important a factor as food deficiency.

At a meeting of medical officers held on April 1st, 1916, in Kut during the siege, Captain E. G. S. Cane showed 26 cases of beri-beri, and reported on 60 others which had recovered. These cases appear to have been of the ordinary type, and were not associated with jaundice. Early in the siege the white bread of the ration was replaced by brown bread, and much improvement in the beri-beri cases followed the change.

Etiology.—A diet of pure proteins, carbohydrates, and fats with salts and water does not maintain health, unless a minute quantity of certain substances, known as vitamines, is added. The absence of one of these, a soluble non-protein nitrogenous substance, which contains no phosphorus and is destroyed at 130° C. but not at 100° C., is the chief cause of ordinary beri-beri.

The absence of another, which is destroyed already at 70° C., causes scurvy. Beri-beri is most common among races whose staple diet is machine-polished rice, the husk and aleurone layer of which have been removed. Rice prepared in this way contains none of the vitamine required to prevent beri-beri, but this is present in sufficient quantity in the aleurone layer. The addition of an extract of rice-polishing or the substitution of rice, from which the husk has been removed by steam or hot water which leaves some of the aleurone layer, prevents the development of beri-beri or cures it if it has developed. But beri-beri may also develop in individuals who eat no polished rice, if their diet contains an insufficient quantity of the necessary vitamine. As the heat used in the sterilisation of tinned food destroys the vitamine, a diet consisting mainly of tinned food with no fresh eggs, vegetables, or meat may cause beri-beri.

The facts which will presently be recorded show that it would be impossible to explain the outbreak of the disease among the British troops at Gallipoli solely as a result of a deficiency in the diet ; it is necessary, therefore, to recall the observations on beri-beri made over ten years ago by Hamilton Wright, although they have recently been to a great extent ignored owing to the general acceptance of the food-deficiency theory. Hamilton Wright came to the conclusion that the disease was due to a specific infection of the duodenum, the toxins produced having a specific action on the nervous system. He was led to this view by the fact that acute and subacute cases begin with anorexia, especially for solid food, a dull heavy or burning sensation in the epigastrium, which is prominent and tender, diarrhœa and occasionally vomiting. The nervous symptoms appear between 5 and 72 hours later, but in a few cases simultaneously with the digestive symptoms. In cases which die in the early stages, the mucous membrane of the pyloric end of the stomach and duodenum is congested and shows minute hæmorrhages ; microscopically the mucous membrane and submucous tissue are infiltrated with round cells and bacilli, and well-marked necrosis of the epithelium is present, leading to superficial sloughing of the mucous membrane. The duodenitis disappears in about three weeks, and is therefore not found in patients dying after the acute stage has passed, when nothing but residual paralysis and cardiac weakness are present. Similar

pathological changes have been recorded by other observers, including Malcolmson, Anderson, Simmons, and Miura.

Association with Jaundice.—In fourteen out of my seventeen cases (82·4 per cent.) the symptoms followed an attack of epidemic bacillary jaundice at intervals varying between three and thirty days from the onset of the jaundice, the average interval being eighteen days. In one case seen by Willcox the jaundice only began three weeks after the onset of neuritis. Of the three cases in which there was no jaundice one patient had an attack of vomiting with epigastric pain and anorexia fifteen days before the onset of the neuritis; in both of the other cases the neuritis was immediately preceded by diarrhœa, the patient having had dysentery some time before. It has been suggested that the association was accidental and simply due to the fact that jaundice was very common on the Peninsula, but neuritis occurred at least eight times as frequently among patients who had had jaundice as it would have done if the association were simply accidental. In eleven cases of beri-beri in British troops from Mesopotamia Willcox only obtained a history of recent jaundice in one and of chronic diarrhœa in another, but in most of his cases both from Gallipoli and Mesopotamia epigastric discomfort and flatulence, generally associated with tenderness over the duodenum, were early symptoms.

The association of these cases of neuritis with epidemic jaundice is strikingly similar to the association of diphtheritic neuritis with diphtheria; the patient is generally fit in every way till the onset of the symptoms which immediately precede the development of jaundice. His convalescence from jaundice appears to be normal, but within a few days of getting up after the attack is over the symptoms of neuritis appear.

It must be remembered that jaundice is merely a symptom and not necessarily an essential part of the disease, which is known for the sake of convenience as epidemic bacillary jaundice. In some cases severe jaundice occurs with very trivial symptoms, whereas in others the digestive and general disturbance is considerable, but the jaundice is very slight. Cases probably occur in which the duodenitis, which I believe was the primary condition in the Gallipoli epidemic jaundice, does not give rise to sufficient obstruction either at the mouth of the common bile-duct or by spread of the

infection to the smaller ducts for jaundice to occur. This may well explain the occasional absence of preceding jaundice in these cases of peripheral neuritis. In a case already referred to symptoms occurred a fortnight before the onset of the neuritis, which were in every way similar to the prodromal symptoms of bacillary jaundice, and the constant anorexia and abdominal discomfort caused by the chronic diarrhœa in the two remaining cases might have prevented these prodromal symptoms from being recognised. Such cases of neuritis following the infection but without jaundice would be strictly comparable to the cases of diphtheritic paralysis, in which the preceding diphtheritic pharyngitis escapes recognition.

Whilst in some cases the preceding infective process may be latent owing to the absence of jaundice, in others I believe the neuritis may be latent. Major Shelshear and I examined twenty consecutive convalescent patients, who had become jaundiced between three and twelve weeks before and were now in a convalescent depôt in Mudros West. In no less than eleven cases we found definite signs pointing to the presence of peripheral neuritis, generally associated with cardiac dilatation and tachycardia and in two cases with slight œdema of the ankles, and in three others we found some slight paræsthesia of the feet, tenderness of the calves or difficulty in rising from a squatting attitude, together with slight cardiac dilatation. Several of the patients showed some tremor of the hands or general unsteadiness, and others had a dilated heart with a rapid pulse, but we did not include these cases among the abnormal ones unless signs of neuritis were also present, as similar symptoms were not uncommonly found among patients convalescent from various other conditions and even among apparently healthy soldiers from Gallipoli. The patients had either not noticed anything abnormal themselves or had not thought the symptoms sufficiently important to mention. It is difficult to draw a line between these cases and the slighter ones of definite "beri-beri." If we are correct in regarding them as examples of latent neuritis following epidemic jaundice, it becomes all the more probable that the severe cases are directly due to the same infection as that which causes the jaundice. A similar conclusion may be drawn from the condition of the heart. It was recognised by all observers during the epidemic of jaundice at Gallipoli that the heart was very

frequently dilated and the pulse sometimes very rapid after the initial bradycardia had disappeared. It is not surprising, therefore, that a similar dilatation should be observed in the cases of peripheral neuritis.

It has also been suggested that the association of beri-beri with jaundice might be due to the strict dieting given for the latter together with the associated toxæmia being sufficient to act as a final stimulus to the development of the disease, the occurrence of which was already imminent owing to certain deficiencies in the diet. But if this were the case, peripheral neuritis of the same character should have been still more common after dysentery and paratyphoid fever, as the former at any rate caused more invaliding than epidemic jaundice, and in both diseases the strict dieting was likely to be much more prolonged and the toxæmia much more serious. But I know of only two cases which followed dysentery and none which followed paratyphoid fever.

The earliest cases of peripheral neuritis were recognised about the middle of October, 1915, and they continued to occur in small numbers until the evacuation of Gallipoli; allowing for the interval which elapses between the onset of the jaundice and that of the neuritis, the incidence of the latter is what would be expected if it were due to the same infection, as the epidemic of jaundice assumed serious proportions about the middle of September. If the neuritis were solely due to deficiency of certain essential constituents of the diet, in agreement with the generally accepted etiology of true beri-beri, it should have made its first appearance before October among those who landed on the Peninsula in April, and it would be difficult to explain why it should have developed equally frequently among those of my cases who had been on the Peninsula for 6 to 16 weeks as among those who had been there for 20 to 24 weeks; I saw no cases among men who had been on the Peninsula for over six months. The supply of fresh food was more regular at Helles than at Anzac, and it was thought at first that the disease was least common at Helles, but this was not the case, as 11 out of my 17 patients (65 per cent.) came from Helles. Moreover many of the patients had had a fair supply of fresh bread and meat and occasionally even of eggs —articles of diet which would have prevented the development

of beri-beri. An army baker developed the disease in spite of having had a very much more satisfactory diet than that of the majority of the troops. It is also noteworthy that the symptoms of neuritis generally did not begin until the patient had returned to a full diet for some days.

Lieut. C. R. N. Pattison has told me of a somewhat similar epidemic of peripheral neuritis following jaundice, which attacked about 80 out of the 300 lepers in the colony at Mokagai, Fiji, of which he was acting superintendent, between June and August, 1914. The jaundice was followed in almost every case by pains in the calves and thighs, and difficulty or inability to walk with diminished or lost knee-jerks ; the heart was greatly dilated, but there was no œdema, and three patients died of heart failure. He did not observe similar symptoms among any of the lepers who had not had jaundice.

It seems highly probable that the beri-beri, which occurred among British troops, was due at any rate in Gallipoli to a similar duodenal infection to that observed by Hamilton Wright, though deficiency in the anti-beri-beri vitamine was probably a contributory factor. The organism which infected the duodenum produced greater local swelling than in his cases, and it consequently caused jaundice in most cases with comparatively little damage to the nervous system, but the toxæmia occasionally caused symptoms indistinguishable from those of beri-beri. In most cases the interval between the onset of the duodenitis and the onset of the neuritic and circulatory symptoms was longer than in ordinary beri-beri.

In two fatal cases from Gallipoli the post-mortem appearances described by Willcox were very similar to those found by Hamilton Wright. The mucous membrane of the stomach was very red, especially in the pyloric half, where the colour was deep crimson. The duodenum showed intense crimson congestion of the mucosa, most marked in the upper part. The rest of the small intestine and the colon were also congested.

Symptoms.—The onset was generally rapid. In well-marked cases peripheral neuritis, very similar in character to that caused by alcohol, was associated with general œdema and dilatation of the heart, with a rapid pulse and dyspnœa on the least exertion. The earliest symptoms were generally weakness of the legs and

shortness of breath, but these were occasionally preceded by œdema of the ankles.

The muscles of the legs and to a less extent of the arms were weak ; the extensors were more affected than the flexors, so that foot and wrist drop developed and a " steppage gait " like that of alcoholic neuritis was often present. In severe cases walking was impossible. The muscles showed considerable atrophy and were very tender on pressure, but the arms were less affected in this way than the legs. The deep reflexes were lost ; in some of the earlier cases seen by Lieut.-Col. de Crespigny the knee-jerks disappeared before the ankle-jerks, but this was never observed among my cases, both jerks being absent in eight, both present in five, and the knee-jerks were present but the ankle-jerks lost in four.

Paræsthesia was common, the patient complaining of tingling or a sensation of pins and needles in his toes and fingers. More or less cutaneous anæsthesia and analgesia were always present and extended a varying distance up the legs ; the arms were less frequently affected.

Œdema was most marked in the legs, but was also present over the lower part of the back, in the scrotum, over the abdomen and sternum and in the face in the severer cases. As a rule it disappeared much more rapidly than the other symptoms. In a few cases the beri-beri was of the " dry " form, with no œdema at all or only a little affecting the ankles.

The heart was dilated, and occasionally a systolic murmur was heard at the apex, but this disappeared as the condition improved. The pulse was rapid and sometimes irregular ; the least exertion greatly increased its frequency and at the same time caused marked dyspnœa.

Pyrexia was generally absent, but in some cases slight fever continued for a few weeks.

Anorexia, slight epigastric discomfort, and in severe cases vomiting were often present, especially at the onset.

Case of Beri-beri following Jaundice.—Driver W. landed at Helles on May 6, 1915. He remained well in every way, complaining of no weakness, shortness of breath, or other symptoms until October 16, when he suddenly lost his appetite. He felt sick when he saw food and he vomited when he tried

to eat. On October 19 he became jaundiced, and on the 21st he was sent to the Lowlands Convalescent Depôt at Lemnos. He had had fresh bread daily and fresh meat five days a week whilst on the Peninsula until his illness began. After that he only had bread and condensed milk both at Gallipoli and at the Convalescent Depôt, with the addition of fresh meat on three occasions, and about two eggs a week. On November 15, when the digestive symptoms were much better, the jaundice had almost disappeared, and he had been up for a few days, his legs felt stiff and weak and he became breathless and suffered from violent palpitation on the slightest exertion. On November 25 he was transferred to the 3rd Australian General Hospital. His legs, back, and face were œdematous, but there was no albuminuria; his pulse was 102, his heart was dilated and its action was thumping. The legs, and to a less extent the arms, were very weak; foot and wrist drop were well marked. The knee- and ankle-jerks could not be obtained. The calves were very tender. There was some loss of tactile and painful sensation on the feet and round the mouth. He became progressively weaker for about ten days, but began to improve on December 10, soon after I first saw him with Lieut.-Col. Stawell. Between December 5 and 22 his temperature varied between 99° and 100°.

Arrangements were made for the patient to be transferred on his arrival in England to the care of Major Judson Bury, who reported on January 13, 1916, that the patient was much better, the cardiac symptoms having disappeared, and in May he was able to leave hospital almost completely cured. He wrote to me in April, 1917, that he was now working full time at motor driving, and was quite well, except that he occasionally felt a little dizzy, if he stood up suddenly after lying down, and after unusual exertion. Major Bury saw him on May 2 and found slight weakness of the dorsi-flexors of the left ankle. The knee-jerks were normal, the left being slightly brisker than the right. The right ankle-jerk was normal, but the left one was absent. The heart was normal.

Case of Beri-beri without preceding Jaundice.—Driver B. landed at Helles on May 9. He had dysentery for a fortnight in August; this was followed by diarrhœa, which continued on and off until he was sent to No. 2 Australian Stationary Hospital on

November 5. He had never been jaundiced; he had had no appetite and had complained a good deal of abdominal pain with the diarrhœa, especially in the middle of October. He had had sixteen injections of emetine in August and September. On November 3 his legs began to get weak and he felt short of breath; on November 8 his legs became swollen. His heart was very dilated and his pulse rapid. His arms and legs became progressively weaker, till he had well-marked wrist and foot drop; his knee- and ankle-jerks were lost. His calves were very tender and there was definite anæsthesia of his feet and legs. His temperature rose almost every night whilst he was in hospital to between 99° and 100°. Slight improvement began to occur in the middle of December. Major Judson Bury reported on January 13, 1916, that there was not much change; the patient had slight movement at the knees and hips, none of the feet; he could feebly extend his wrists. Anæsthesia was present up to the knees and there was well-marked hyperæsthesia of the muscles of the arms and legs, but no cutaneous hyperæsthesia. There was still some diarrhœa and abdominal pain. Soon afterwards he began to improve and left the hospital in May.

On August 22, 1916, the patient wrote to me that he had been discharged from the army; his legs were still weak, but he could now dorsi-flex his feet. There was no anæsthesia, except over the soles of his feet. The arms, though thin, were quite strong. The ankles became swollen in the evening, but there was no palpitation or other cardiac symptom. He returned to clerical work in February, 1917. I saw him on April 17, seventeen and a half months after the onset of the illness. His heart was still dilated, the impulse being an inch outside the nipple line, and a systolic murmur was audible in the mitral area. But he had walked seven miles in one afternoon the previous week and only felt short of breath on violent exertion. The œdema had completely disappeared. The extensors of the hands and feet were still rather weaker than normal; the knee-jerks were normal, and the ankle-jerks were present, but not easy to obtain. There was no anæsthesia or paræsthesia.

The first of the above cases was selected as typical of the "beri-beri" following jaundice and the second as an example of the exceptional cases with no jaundice. It was afterwards discovered

that the two men, who were in different hospitals in Lemnos, belonged to the same Field Ambulance at Gallipoli. The neuritis developed about the same time in both, and when one had jaundice, the other had noticed an aggravation of his abdominal pains, though no jaundice was present. It seems not unlikely that this aggravation represented an attack of duodenitis, the local results of which were insufficient to cause jaundice.

Prognosis.—Out of a total of approximately thirty well-marked cases from the Dardanelles three died from heart failure. In the others improvement began to take place after two or three weeks, but in the severer cases recovery was very slow.

Prophylaxis.—The occurrence of beri-beri in British soldiers shows the importance of providing special vitamine-containing food, which can be added to the ordinary ration, when troops are engaged in an expedition in which there may be difficulty in providing regular supplies of fresh food. This is particularly important if an epidemic of jaundice should break out, and all jaundiced soldiers should under these conditions be given a special supply of anti-beri-beri vitamine, both whilst they are actually ill and during convalescence. Haricot beans, pea soup made from pea powder, and porridge are valuable foods and are generally obtainable, but dried yeast or yeast cakes, which contain a specially large proportion of the necessary vitamine, should be carried with the medical equipment, and should be widely used if an outbreak of beri-beri occurs and fresh food is unobtainable. Fresh vegetables and fruit should also be provided whenever possible in order to supply the vitamine necessary to prevent scurvy. The following list of articles of diet which contain the anti-beri-beri vitamine was prepared by Colonel W. H. Willcox and Major C. J. Martin; the articles are arranged in order, beginning with the one which contains the largest quantity: yeast, eggs (raw or lightly cooked), brain, liver, sweetbread, kidneys, heart muscle, peas, haricot beans, lentils, porridge, brown bread, fresh milk, fish and meat, ordinary bread and biscuits.

A preparation of yeast extract called marmite has been added to the ration of British troops in Mesopotamia since the autumn of 1916. Colonel Willcox describes it as being like meat extract in taste and appearance. It can be taken alone or in stew and soup. It is extremely rich in anti-beri-beri vitamines, and the

very small number of cases of beri-beri which have occurred since its issue have been mild, and there have been no deaths. At the same time a very palatable bread has been made from a mixture of British flour and "atta," the India flour, which contains the aleurone layer and germ of the wheat grain, and oatmeal has been added to the ration since July, 1916.

Treatment.—A generous mixed diet should be given with as large a proportion as possible of the articles already mentioned as containing the essential vitamine. In severe cases the indigestion and anorexia may make it impossible for a few days to take such a diet. Two ounces of "export yeast" or six "royal yeast cakes" should be given daily in such cases; the yeast is mixed with a little boiling milk into a cream, and then more warm milk and sugar are added (Willcox). If neither of these preparations is available, half a pint of yeast brew should be obtained from the army bakery and drunk after sweetening with sugar. Pea soup and three or four raw eggs beaten in milk should also be given.

The patient should rest in bed until his heart is no longer dilated and the pulse rate is normal. It is doubtful whether any drugs assist the heart, but digitalis may be tried in full doses. Exercises should be begun at an early date for the arms and subsequently for the legs, as apart from their value in hastening recovery from the paralysis, they are very useful in restoring tone to the cardiac muscle.

The treatment of the paralysis does not differ from that caused by other forms of neuritis. It is most important to prevent the paralysed muscles becoming over-stretched whilst the patient is still in bed; the hands should be kept extended by splints if wrist-drop is present, and the feet dorsi-flexed if there is foot-drop. Massage and passive movements should be begun as soon as the muscular tenderness has sufficiently subsided.

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CHAPTER XVIII

TETANUS

The danger of tetanus as a complication of wounds has been recognised since the days of Hippocrates, and little can be added to the classical descriptions of the acute disease. The main facts concerning the pathology of tetanus were discovered between 1885, when Nicolaier produced the disease in mice by inoculating them with garden soil and isolated a characteristic bacillus from the local lesion, and 1890, when Kitasato, having prepared in a pure form the toxin, which Faber had discovered the previous year, laid the foundation of serum prophylaxis and treatment, by showing that animals could be rendered immune by the repeated injection of non-lethal doses of the toxin and that their serum had the property of neutralising it in vitro. But tetanus deserves a special chapter in a book on medical diseases of the war, as the war has been the means of at last proving beyond doubt the value of antitetanic serum in prophylaxis ; it has shown how preventive inoculation diminishes the severity of the disease and modifies it in such a way that the majority of cases show premonitory symptoms, which were formerly scarcely recognised, but which now enable treatment to be instituted so early that the chances of success are greatly increased, and that in other cases the symptoms remain localised, giving rise to a remarkable form of tetanus, which had never before been described; lastly it has conclusively proved the value of treatment with anti-tetanic serum, and it has made it possible to form a judgment on the relative merits of different methods of administering serum.

Etiology.—Tetanus bacilli and their spores do not inhabit uncontaminated soil, but are frequently present in the intestinal contents of horses and cattle. As the spores are exceedingly resistant, soil once contaminated with horse or cattle dung continues to be a source of danger for an indefinite period. As

cattle and horses are much more numerous in some districts than in others, and the frequency with which tetanus bacilli are present in their fæces varies greatly in different localities, the danger of contamination of wounds with soil is very inconstant. Thus in spite of the absence of prophylactic inoculation, the disease occurred in only 505 or 0·2 per cent. out of 246,712 wounded men in the American Civil War, and it was so rare in the South African War that Makins only heard of a single case. On the other hand, there is little doubt that it would have been extremely common in France and Flanders had not universal prophylactic inoculations been introduced at an early date. The greater frequency of tetanus after wounds received at the battles of the Marne and Aisne than in the neighbourhood of Ypres was doubtless due to a corresponding difference in the number of tetanus spores in the soil. The rich soil in the valley of the Aisne has indeed such an evil reputation for tetanus that the farm-horses have for several years been periodically inoculated with prophylactic doses of antitoxin.

The tetanus bacillus may enter a wound the moment it is inflicted, or the infection may occur owing to the wound becoming soiled by earth when the soldier falls or crawls to a place of safety. A bullet, being sterile, is not likely to cause tetanus except after a ricochet. Tetanus spores are much more frequently introduced by shrapnel, as the shell bursts on the ground so that its fragments are likely to carry earth with them. Moreover, fragments of shell are likely to carry dirty pieces of clothing with them, whereas a pointed bullet often makes a clean hole through the uniform. Tetanus is most frequent when heavy fighting occurs in wet weather, when the mud leads to severe infection of wounds.

In all cases the wounds have been septic at some period, but they have often almost or completely healed by the time tetanus develops. They may be severe or trivial, but the risk appears to be greater the larger and more septic the wound. Dead tissue and foreign matter provide a favourable site for the multiplication of the tetanus bacillus, which is a saprophyte with little capacity for growth in living tissue. Hence the liability to tetanus is greatest in lacerated and contused wounds, in cases of compound fracture, and in wounds in which a foreign body is present.

In the American Civil War it was observed that 26 per cent. of the cases of tetanus followed immediately after a secondary

operation. The present war has afforded numerous instances of this, the appearance of tetanus being delayed for weeks or even months after a wound has been inflicted, when a secondary operation, often of a quite trivial nature, at the site of the original wound, even if it has completely healed, is followed a few days later by the appearance of tetanus.

A considerable number of cases of tetanus developed in France as a complication of trench foot, and I saw several cases of this sort at Lemnos after the great storm at Gallipoli at the end of November, 1915. The œdema fluid infiltrating the connective tissue of the foot affords a perfect culture medium for the growth of the tetanus bacillus, and the chilling of the tissues inhibits the leucocytes from taking up the spores. Tetanus has also followed septic burns; in one case the man had rolled in the mud in order to extinguish the flames.

Incubation Period.—The incubation period of tetanus in the present war has varied between 2 and 365 days. The average of cases occurring in France was twelve days, the largest number developing on the eighth day, three days earlier than in the cases occurring in England (Leishman and Smallman). The incubation period has become longer since the vast majority of cases have received prophylactic injections. In the first year of war 47 per cent. of cases, but between December, 1916, and March, 1917, only 10 per cent. had an incubation period of less than ten days (Bruce). An incubation period of more than twenty-two days occurred in 69 per cent. of cases in a recent series, but in only 6·4 per cent. in the first year and 5·7 per cent. in the Franco-Prussian War.

When symptoms do not appear until three weeks or longer after the wound, the true incubation period is probably much shorter than it appears to be. The *B. tetani* is generally, if not always, introduced into wounds in the form of spores, which do not themselves produce toxin. The spores may never germinate, or they may only produce the bacillus after an interval of days, weeks, or even months, during which the wound may have completely healed. In many cases of this sort the true incubation period dates from a secondary operation, which has had the effect of stimulating the spores to develop into bacilli, which at once begin to produce toxin.

The Cause of the Symptoms in Tetanus.—The toxin

produced by tetanus bacilli in a wound passes into the blood and lymph, in which it can actually be found both in animals and man, the amount reaching a maximum at the time the first symptoms appear. It has been conclusively proved that part of the toxin is carried to the central nervous system by the motor nerves; the nerve endings in the neighbourhood of the wound take it up from the surrounding lymph. The first part of the central nervous system to be involved is consequently the segment connected with the motor nerves in the neighbourhood of the wound. From this situation the toxin spreads along the spinal cord to the brain, except in the case of wounds of the face, when the toxin passes direct to the pons and medulla by the facial nerve. At the same time some of the toxin circulating in the general blood-stream reaches the central nervous system without passing up the motor nerves.

The toxin, having entered the spinal cord or brain, combines with the nerve-cells. It renders them abnormally sensitive to all stimuli, so that the afferent impulses which are constantly reaching them produce a condition of tonic contraction in the muscles they supply, and every additional afferent impulse causes additional spasms. In unprotected men and in large animals, such as horses, the toxin which reaches the medulla and pons from the blood produces trismus, which is followed by other symptoms as other centres become affected.

In the cat, which is relatively insusceptible to the tetanus toxin, a non-lethal dose leads to very persistent local spasms of the limb into which the toxin was injected, but no generalised contractions occur, as the only toxin to take effect is that which reaches the spinal cord by the motor nerves. If in the more susceptible guinea-pig a sufficient quantity of antitoxin is injected with the toxin or shortly afterwards, the result is the same. Local tetanus hardly ever occurs in uninoculated men, as the susceptible centres in the pons and medulla are attacked before the spinal centres. But antitoxin has the same effect in man as in the guinea-pig. Any toxin circulating in the blood is neutralised by the antitoxin introduced by prophylactic inoculation. The centres in the pons and medulla are thus protected, as they are not affected by the toxin ascending the motor nerves, except the facial nerve in the case of wounds of the face. But the spinal

centres may be attacked, as they are reached directly by the toxin travelling up the nerves from the wound; the spasms therefore generally begin in the neighbouring muscles. If no more serum is given, the protection afforded by the antitoxin gradually disappears and the pontine and medullary centres may finally be attacked. When, however, sufficient serum has been injected, no toxin reaches the pons and medulla, and the symptoms remain localised. The spasms may spread to the muscles of the rest of the limb or even to those of the opposite side and the neighbouring part of the trunk, as they receive their nerve supply from adjacent parts of the spinal cord into which the toxin can readily spread.

The muscles at first relax under the influence of a general or intraspinal anæsthetic, which depress the abnormal excitability of the nerve-centres. But after about thirty hours the muscles themselves are altered in such a way that the spasm persists under these conditions, and in animals it is not completely abolished by dividing the motor nerves or even excising pieces of the affected muscles.

Generalised spasms only appear in men who have received protective inoculation under two conditions: such an overwhelming quantity of toxin may be absorbed that the antitoxin cannot neutralise it, and generalised tetanus occurs after a short latent period; or toxin may be formed by bacilli, which only develop from spores after all the antitoxin has disappeared from the circulation, and generalised tetanus occurs, but the latent period is then prolonged.

Premonitory Symptoms. — In his description of the tetanus which occurred in the Armée d'Orient in Egypt and Syria in 1803, Larrey wrote: "Cette maladie commence par un malaise générale et une sorte d'inquiétude qui s'empare du blessé. . . . Ce phenomène est accompagné de douleurs aïgues, qui augmente par le contact de l'air et des plus légers corps extérieurs; la totalité du membre devient douloureuse. L'irritation musculaire s'étend rapidement des muscles voisins de la plaie, aux plus éloignés qui se contractent; ou bien elle se transporte tout-à-coup aux muscles de la gorge et des mâchoires où elle se concentre."

Although similar cases were collected by Poland from the Guy's records between 1820 and 1857, very little attention was

paid by subsequent writers to these premonitory signs, the great importance of which has only become recognised since the beginning of the present war.

The occurrence of pain in the neighbourhood of a wound is so common that it cannot by itself be regarded as of any importance. But if a patient, who has been quite comfortable for some days, suddenly develops a sharp recurring pain in the neighbourhood of the wound without obvious reason and without any change in the appearance of the wound, the possibility of tetanus should be considered. In several instances this premonitory pain has been regarded as rheumatic. The surrounding muscles, which were previously soft and relaxed, may appear to be abnormally rigid, and exaggerated irritability of the muscles is shown by their spasmodic contraction on the slightest stimulation.

Scattered through the literature of tetanus are references to cases which began with spasms in the wounded limb. Key in 1836 and Fagge in 1886 described cases they had seen at Guy's, in which the local spasms were throughout the most prominent feature of the illness. Now that all wounded men receive prophylactic injections, the first symptom is local in about half of the cases in which the disease finally becomes generalised. Some of the muscles around the wound twitch or show irregular clonic or tonic contractions, or tonic contractions may occur in a group of muscles near by, especially in the flexors. These symptoms may persist for a few hours up to twenty-five days before the jaws become involved. Andrewes records the case of an officer, who received an injection of antitoxin six hours after being wounded in the thigh. Nine weeks later, when the wound was healed, though a small fragment of shrapnel was still present, the leg felt rather stiff when he walked. In spite of this he returned to duty in France and remained at his work for a fortnight, when the stiffness was so great that he could no longer walk. Trismus, stiff neck, and opisthotonos now developed, but recovery followed the daily intrathecal injection of large doses of antitoxin for eighteen days with occasional intramuscular injections in addition.

Other symptoms occasionally precede the development of tetanus, probably as a result of the general toxic action of the tetanus poison. Outbursts of temper with unusual restlessness

and insomnia occur, and the patient, who was previously feeling very well, complains of severe headache and giddiness. Excessive yawning and slight difficulty in micturition have also been observed.

In a case recorded in the *British Medical Journal* of November 17th, 1917, a man complained of urgency of micturition, followed by difficulty and finally inability to pass urine, six weeks after receiving a gunshot wound of the buttock. Severe spasm of the sphincter vesicæ was found to be present, and the violent contraction of the bladder almost expelled the catheter. Symptoms of generalised tetanus developed four days later, and severe spasm of the bladder continued until the patient died.

Symptoms.—Apart from the muscles in the neighbourhood of the wound, the earliest to be involved are the masseters and posterior muscles of the neck. The patient generally notices the stiffness of his jaws for the first time when he wakes in the morning. The spasm increases until it is impossible to force the jaw open. At a later stage the face becomes affected: the lips are stretched over the clenched teeth so as to produce a fixed and mirthless smile—the *risus sardonicus;* the naso-labial folds are exaggerated, the forehead is wrinkled and the eyelids half closed, but the muscles of the eyes and tongue are not involved.

I saw an officer with Sir Alfred Fripp, who had had his jaw shattered by a piece of shrapnel so severely that the dental surgeon in charge found it impossible to fix the fragments. On the sixth day he developed tetanus, and the contracted masseters acted as a splint so efficiently that when he recovered from the tetanus the fragments of the jaw had united firmly in excellent position, and there was no obvious deformity except a depressed scar where the fragment of shrapnel had entered.

Before any definite retraction is present it is often found that the head cannot be bent sufficiently forward for the chin to touch the chest. The recti abdominis muscles are next involved, and then the other muscles of the abdomen and back. In the fully developed disease the head is retracted and the back is arched. Aretæus described a form of tetanus under the name of emprosthotonos, in which the body is bent forward instead of backwards, as in the familiar opisthotonos, but this, like pleurosthotonos, in which the body is bent to one side owing to the asymmetrical distribution of the spasm, is extremely rare. The muscles of

the legs, especially the flexors of the hips and knees, together with those of the upper arm, take part in the general spasms, but the forearms and hands are spared.

In addition to continuous tonic contractions of the affected muscles, tonic and clonic spasms occur. They may appear to be spontaneous, but more frequently they are obviously reflex in origin and follow a touch, exposure to a draught, a bright light, or a sudden noise.

Sherrington has shown in animals that the tetanus toxin disturbs "reciprocal innervation." Thus stimulation of the cerebral cortex in the area, which should cause the mouth to open by contraction of the depressors and relaxation of the elevators of the jaw, produces trismus, as the normal relaxation of the elevators, which are stronger than the depressors, is converted into contraction. Similarly in man, an effort to open the mouth, instead of causing relaxation of the masseters at the same time as the depressors contract, leads to their contraction, the trismus being thereby increased. In the same way voluntary efforts to move other affected parts increase the spasm. Thus apart from the difficulty in taking food caused by the trismus, an attempt to swallow often causes spasm of the pharyngeal muscles.

The constant tonic contractions make the muscles ache, and the spasms cause a varying amount of pain, which often prevents sleep. More or less complete relaxation may be produced by drugs, which in this way make sleep possible, but the spasms quickly return a few minutes after waking.

The increased excitability of the nervous system is shown by the exaggerated tendon and bone reflexes. Thus the wrist- and knee-jerks are increased, ankle-clonus may be present, and the jaw-jerk, according to Major A. G. Gibson, is obtained with unusual ease, even in local tetanus involving a leg.

When the spasms are severe, the pulse becomes rapid, and sweating is profuse, but the temperature may remain normal, especially if the wound has already healed. A pulse of 100 to 130 with a normal temperature is characteristic. In fatal cases, however, hyperpyrexia may develop shortly before death.

Even in the severest cases the mind remains clear until the end, but in rare instances delusions and even hallucinations have occurred. Complications are rare: a few cases of thrombosis

in the femoral or other veins, and one case of multiple neuritis have been recorded.

Recurrence occurred after apparent recovery in several of the cases in the series collected by Leishman and Smallman. Westwater records a case which began on the sixteenth day after the wound was received. The patient had apparently recovered by the twenty-fifth day; but on the forty-second day the symptoms returned and he died on the seventy-fourth day.

When recovery occurs, some stiffness of the masseters may persist for a considerable time. In one case it lasted for as long as five months. The nerve-centres remain abnormal even after recovery appears to be complete. In two patients, four and five weeks respectively after the last symptoms had disappeared, Monier-Vinard found that the tendon reflexes, especially in the neighbourhood of the wound, became exaggerated under chloroform, and when the corneal reflex was lost, true tetanic spasms occurred; finally, with the abolition of the pupil reaction to light, the spasms ceased, but the exaggeration of the reflexes remained.

The cerebro-spinal fluid is normal, but in severe cases its pressure is raised. It may contain the tetanus toxin, but Professor C. S. Sherrington found that injection of the fluid in three of my cases of localised tetanus produced no symptoms in cats.

Diagnosis.—Early diagnosis is essential for successful treatment. The possibility of tetanus should be constantly borne in mind, and the occurrence of unexplained spasms of pain, "rheumatism," or twitching in muscles in the neighbourhood of the wound, should immediately arouse suspicion. Any hardening of the muscles, either spontaneously or on pressure, and exaggeration of the tendon and bone reflexes, especially of the wounded limb, would render tetanus still more probable. The slightest stiffness or aching of the jaws or the back of the neck should also at once suggest the possibility of tetanus.

The diagnosis must always depend upon the clinical features of the case. It would be fatal to wait for the discovery of the characteristic drumstick bacilli in the discharge from the wound before injecting serum, as other organisms exist which are morphologically almost identical, and the non-spored *B. tetani* is not characteristic in appearance; moreover in many cases repeated

examination fails to reveal the organism in undoubted cases of tetanus. Conclusive evidence is afforded by the production of tetanus in animals by the subcutaneous injection of the discharge from the wound, or, if this fails, of the filtrate of anaerobic fluid cultures; but such tests are valueless as a guide to treatment, as the incubation period of experimental tetanus is between three and four days.

Even in the earliest stages there should never be any real difficulty in differentiating trismus due to tetanus from that produced reflexly by a dental abscess or an impacted wisdom tooth, or directly by inflammation of the temporo-maxillary joint or parotitis. A careful examination of the masseters and the neighbouring structures, the history of the onset, and the very early presence of slight spasm of the posterior cervical muscles in tetanus should prevent the possibility of a mistake.

I have once seen a case of hysterical trismus, which was at first diagnosed as tetanus. A man had trodden with his bare foot on the point of a rusty nail, and two days later he developed well-marked trismus. I was asked to see him in order to advise how much serum should be injected. The very short latent period, however, aroused my suspicions, and I inquired from the man whether he had ever known or heard of anybody having lockjaw. He told me that he had read in the paper on the previous Sunday how a man had died of lockjaw after an accident precisely similar to his own. I told him that his lockjaw was the kind that always got well in a few hours: he was already much better an hour later, and by the next morning the spasms had completely disappeared.

Prognosis.—In the American Civil War the mortality of tetanus was 89·3 per cent., and in the Franco-Prussian War it was 90 per cent. among the German troops. In civil practice the average mortality before the introduction of serum for prophylaxis and treatment was about 85 per cent. In the present war the mortality has become steadily smaller as prophylactic injections of serum have become more universally adopted. It is not yet possible to say to what extent treatment with serum has helped to reduce the mortality, but there can be no doubt that improved methods of treating septic wounds and earlier diagnosis have been important factors. In the 231 cases occurring

in England during the first year of war the mortality was 57·7 per cent., and that of the next 195, which occurred up to the end of July, 1916, was 49·2 per cent.; between August and October, 1916, there were 200 cases with a mortality of 36·5 per cent., and between December, 1916, and March, 1917, 100 cases with a mortality of only 19·0 per cent., which would, however, be 28·3 per cent., if the 19 cases of localised tetanus, in which there was no mortality, were excluded (Bruce). Owing to the shorter incubation period and the greater severity of the wounds in most of the cases of tetanus arising in British troops before they leave France, the death-rate is much heavier among them than in England. In 160 cases occurring between July 1st and October 31st, 1916, the mortality was 73·7 per cent. in contrast with a mortality of 78·2 per cent. in 179 cases occurring in the spring of 1915 (Leishman and Smallman).

The earlier the prophylactic dose of serum is given, the greater is the chance of recovery.

The mortality is greater with a short than with a long incubation period. The average incubation period in 115 fatal cases occurring in France was 10·7 days; that in the 42 cases of the same series which recovered was 14 days (Leishman and Smallman). In Sir David Bruce's last series of 100 cases occurring in England the mortality was 40 per cent. when the incubation period was 10 days or less, 25 per cent. when it was between 11 and 24 days, and only 13·6 per cent. when it exceeded 25 days. Tetanus, however, is not without danger whatever the incubation period: a patient of Miller's died, although the symptoms only appeared 75 days after he was wounded. But every day the patient survives after the sixth the more hopeful is the outlook.

The prognosis is worst with extensive, severe, multiple, or very septic wounds. When tetanus is complicated by gas gangrene, as is frequently the case in France, the mortality is very high, this being the chief cause of the much greater death-rate among British troops in France than in England. The mortality in England can be much more fairly regarded as the true mortality of tetanus with modern methods of prophylaxis and treatment than that in France, in which death is frequently due in part, if not entirely, to the severe nature of the wound itself or to septic complications.

The prognosis is worst when successive groups of muscles are affected in rapid succession, as happens especially when the incubation period is short. Stridor, indicating spasm of the glottis, a temperature over 104° F. before the fifth day, and hyperpyrexia at any time are bad signs.

Cause of Death.—Death may result from asphyxia caused by spasm of the respiratory muscles, especially the glottis. Sudden or gradual heart failure may occur, generally after the pulse has been very rapid for a time. When death takes place in the later stages, when the trismus is diminishing and the spasms have become rare or have even ceased, it is generally due to the exhaustion caused by toxæmia and malnutrition, corresponding with the experimental *tetanus sine tetano*, in which the spasms are prevented from developing, but death occurs from the general toxic action of the tetanus poison, which is normally concealed by the more obvious motor symptoms. Occasionally extreme hyperpyrexia has heralded death. Œdema of the lungs, and septic pneumonia, which may follow aspiration of food, are very fatal complications.

Localised Tetanus

Until the present war the only form of local tetanus which had been recognised was the so-called cephalic or head tetanus. A few cases had been recorded in which the spasms began in the injured limb and remained most severe in it after generalisation occurred; but the first case of tetanus, in which the symptoms were strictly localised to one limb throughout the illness, was described in May, 1915, by Courtellement. Numerous observations on the subject have been published in France and England since that date, but even now many cases of localised tetanus probably escape recognition through want of familiarity with the condition. In October, 1914, I saw an officer, who I am now convinced was suffering from local tetanus, but at that time I regarded the spasms as reflex in origin. Although there is no doubt that the extensive use of prophylactic injections of antitetanic serum is the cause of the relative frequency of localised tetanus since 1915, I believe that the disease is not an entirely new one, but existed before the introduction of serum prophylaxis, as at least one typical case

has been observed which occurred in spite of the fact that no serum had previously been injected (Claude and Lhermitte). The tetanic nature of the spasms in these cases was not, however, recognised before 1915, the condition being regarded as hysterical or reflex in origin. Although in charge of a neurological section during the past year and a half, I have seen nine cases of local tetanus, so that there can be little doubt that the condition is by no means uncommon.

The large majority of cases of localised tetanus occur as a result of the protection afforded by prophylactic injections of serum being sufficient to prevent generalisation, but insufficient to prevent the production of symptoms by the action of the toxin, which is absorbed by motor nerve endings near the wound and acts upon the corresponding segment of the spinal cord. In the few cases in which no serum has previously been injected, the absence of generalisation is probably due to the number of tetanus bacilli in the wound being small or the toxin they produce being unusually feeble.

(*a*) *Cephalic or Head Tetanus.*—In 1867 Rose described a form of tetanus, in which the spasms were confined to the masseters and facial muscles, with the addition in some cases of the pharynx. This condition, which has been called cephalic or head tetanus, is generally complicated by facial paralysis, and is always due to a wound of the face or head.

The facial paralysis is generally confined to the injured side, but it is occasionally bilateral. It may begin before any spasms occur, but more frequently it develops at the same time as the trismus or shortly afterwards. It is never sufficiently severe to prevent spasms occurring at intervals in the paretic muscles, the cells in the medullary centre of the facial nerve being abnormally excitable owing to the action of the tetanus toxin. In most cases the paralysing effect of the toxin on the facial nerve disappears by the time the spasms have completely relaxed.

Poland described a case at Guy's in 1871, in which fracture of the left orbital plate of the frontal bone by the point of an umbrella was followed by tetanus, associated with left-sided facial paralysis and fixation of the left eye in an upward and outward position, the right eye being directed forwards. Since then a very small number of other cases have been recorded, in which cephalic

tetanus was associated with oculomotor or hypoglossal paralysis, either alone or with facial paralysis.

The spasm of the pharynx may lead to dysphagia, which is occasionally so severe on attempting to drink that the condition has been called hydrophobic tetanus.

Cephalic tetanus has been very rare in the present war, the following case described by Captain H. Burrows and two others being the only cases I have seen reported.

Cephalic Tetanus with Facial Paralysis.—The patient was wounded on September 25th by a piece of shell, which entered just above the left tempero-maxillary joint, penetrated the temporal bone and passed upwards into the tempero-sphenoidal lobe of the brain. He received 750 units of antitetanic serum shortly afterwards. When he reached hospital he was aphasic, and the right arm was weak. The fragment of shell was removed on September 30th. On October 6th, eleven days after he had been wounded, trismus and clonic spasms of the left facial muscles appeared. The next day incessant masticating movements of the lower jaw and lips were added to the facial spasms, and the tongue, which took part in the movements, was severely bitten. On October 8th left-sided facial paralysis developed, but the spasms continued, though they were less severe than on the right side of the face, which was now also involved. By October 22nd the spasms, which had finally spread to the left sterno-mastoid muscle, ceased, but the left facial paralysis remained.

(*b*) *Tetanus localised to a Limb.*—The incubation period in some cases is as short as in ordinary tetanus, periods of 5, 8, 9, 10, 11 (twice), 13 (three times), 14 and 15 (twice) days having been recorded. Just as frequently the symptoms appear between the twentieth and thirtieth days, and occasionally still later—after 5, 6, 7, 8, 9 or even 12 weeks. But, as I have already pointed out, the true incubation period in most of the latter cases is really shorter, as the symptoms have generally followed a few days after an operation, which must have disturbed the wound in such a way that a quantity of tetanus toxin has suddenly been absorbed.

Symptoms of Localised Tetanus.—The premonitory symptoms, which I have already described as often occurring in protected individuals before the onset of generalised tetanus, may also precede the appearance of localised tetanus. The first obvious symptom is either twitching or stiffness in the muscles near the wound. The spasms consist either of painful clonic movements or

of momentary and painless twitches. They become gradually less frequent as the stiffness of the limb becomes more pronounced, and in many cases they disappear entirely owing to the permanent tonic contraction of the affected muscles being the maximum possible contraction, so that additional twitching is impossible.

The contractions may remain localised to a single segment of the limb, but more commonly the whole arm or leg is involved. The tonic contraction produces a characteristic attitude. The leg is generally in a position of extension with the foot dorsi-flexed, the limb being as rigid as a poker; less frequently the knee is flexed. The arm is generally flexed at the elbow; the upper arm is adducted and the shoulder shrugged.

In true localised tetanus the contractions remain strictly localised throughout. The nature of such cases might be regarded as doubtful, were it not for the fact that every intermediate type has been observed between the entirely local form and the severe generalised form beginning with slight local symptoms. In the intermediate cases the indications of generalisation may be very slight and only last 24 hours. There may be slight difficulty in swallowing or some pain and stiffness is felt in the masseters or neck, especially at the height of spasms affecting the limb; but neither definite trismus nor retraction of the head is present, and the chest can still be touched with the chin. The muscles of the face and trunk are not affected. These slight indications of generalisation must be distinguished from the result of direct spread, in which the muscles of the trunk in the neighbourhood of the affected limb are involved. Thus spasm of the lower abdominal muscles of the same side may be present in local tetanus of the leg, and the scapular and pectoral muscles were contracted in two of my cases of tetanus of the upper arm. Moreover the spasms occasionally spread to the corresponding limb of the opposite side, though they are never so powerful as on the wounded side; this is very rare in the case of the arm, but several cases of so-called paraplegic tetanus, in which both legs are affected, have been described.

Prognosis of Localised Tetanus.—The prognosis is very much better in local than in generalised tetanus, as there is no danger of asphyxia or respiratory complications, and the general toxæmia is comparatively slight. One of my cases ended fatally,

and Desplas records a case of sudden death. In both instances large quantities of serum were injected intraspinally and subcutaneously. In my case the patient apparently died from some form of toxæmia and not from the effects of the localised spasms; but it was not clear whether the toxæmia was due to some independent infection or to a product of the tetanus bacilli, or whether it was a result of the injections of serum. There were no deaths among the nineteen cases of localised tetanus which occurred in England between December, 1916, and March, 1917 (Bruce).

Diagnosis of Localised Tetanus.—Most cases of localised tetanus which occurred before the middle of 1915 must have been diagnosed as traumatic hysterical contracture, and five out of the nine cases I have seen during the last year and a half were at first regarded as hysterical in origin. The observations published by Babinski and Froment on reflex neuroses led to renewed interest being given to the possible reflex origin of contractures following wounds; but I believe that many cases diagnosed as reflex contractures are really examples of localised tetanus. On the other hand, the contractures in at least two cases which have been published as examples of local tetanus must really have been hysterical or reflex in origin, as they developed immediately after the wound was received, although it is impossible for tetanus to develop until the bacilli introduced at the time of the wound have had time to produce toxin, which has then to travel to the central nervous system before it can give rise to symptoms.

The diagnosis between local tetanus and reflex spasms and hysteria may be extremely difficult. Thus I have seen cases in which tetanic or reflex contractures have been maintained by auto-suggestion after the primary condition had more or less completely disappeared; the removal of the hysterical element by psycho-therapy left behind a small organic residue due to the primary condition.

The onset of spasms immediately after the wound is inflicted excludes tetanus: they are generally reflex and protective in nature, but are often maintained after the first few minutes or hours by auto-suggestion. A later onset is compatible with all three conditions, and in each the extent of the contracture is often out of proportion with the size of the wound. If the contracture persists in sleep, hysteria can be excluded, but it may be due to

a reflex or to tetanus. A general anæsthetic causes hysterical contractures to disappear as soon as consciousness is lost, but both reflex and tetanic contractures persist, although under deep anæsthesia reflex spasms finally disappear, whereas those due to tetanus may be still present in a minor degree. The tendon and bone reflexes are never altered in hysteria; they are often exaggerated both in reflex conditions and in tetanus, and the exaggeration is often most obvious when partial relaxation has been produced under an anæsthetic. If the muscles are of a wooden and unvarying hardness, tetanus is almost certainly present. Vasomotor disturbances are most common and best developed in reflex neuroses, but they may occur in a mild degree in hysteria and tetanus. An increase in the size of the muscle, possibly due to obstruction of its lymphatics, without tenderness or subcutaneous œdema, is conclusive evidence in favour of local tetanus. The continued tonic contraction in tetanus is generally accompanied by spasmodic and more or less painful contractions, which are often brought on by external stimuli.

When there is any doubt as to the diagnosis the condition should be assumed to be local tetanus, as in local tetanus it is never possible to say whether generalisation may not develop later. Early injections of antitetanic serum should prevent this, and they will do no harm even if the spasms are not really due to tetanus. Whenever it is possible that the spasms are wholly or in part hysterical in origin, the effect of psychotherapy should be tried.

Localised Tetanus following a Wound of the Pectoral Muscles.—Pte. M., aged 31, was wounded on July 25th, 1916, by a small fragment of shrapnel, which lodged in the outer part of the left pectoralis major without injuring the ribs. Nineteen hours later he received a prophylactic injection of antitetanic serum. Two small pieces of metal were removed from the wound on August 17th. The next day he had a "jumping" sensation over the wound, which was now about the size of a shilling. On August 20th he complained of spasmodic pain in the left arm, which soon became so severe that it could only be relieved by injections of gr. 1/4 of morphia. When he came under my care on the 30th, the pain had much diminished; the outer part of the pectoralis major, though not the clavicular portion, together with the biceps, brachialis anticus, deltoid, trapezius, and supra- and infraspinatus were in a condition of constant spasm (Fig. VI.). Some days later the triceps and flexor carpi radialis also became affected.

The patient's head was slightly inclined towards his left shoulder, which was considerably raised; his arm was at first slightly abducted, but later it was held firmly to his side and the forearm was fully flexed; the hand was semi-pronated, the palm being in contact with the epigastrium. The muscles of the forearm

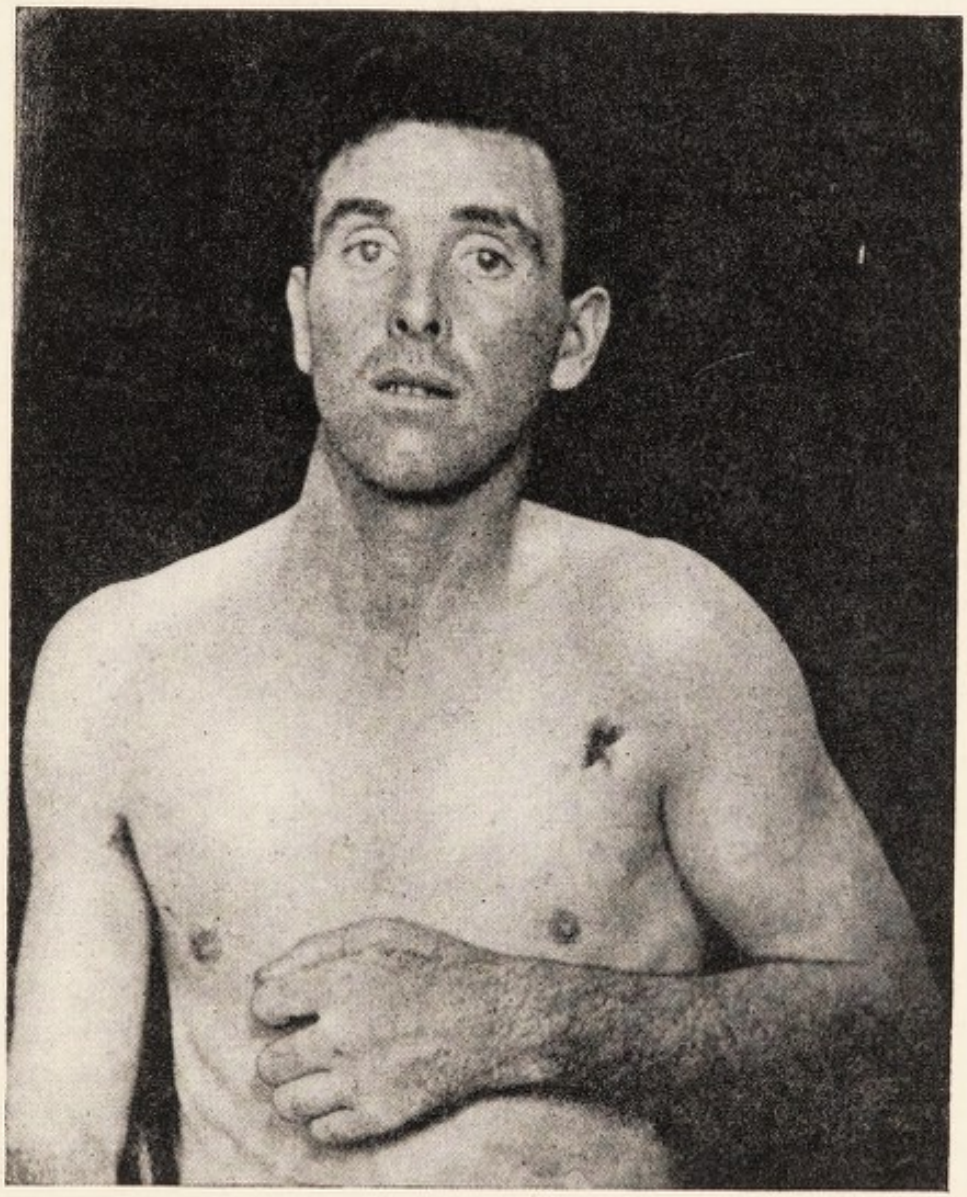

FIG. VI.—Case of tetanus, localised to left arm, left side of neck and pectoral muscles, following wound in outer part of pectoralis major.

and hand were not affected. The spasm was so extreme that it could not be overcome even sufficiently for a towel to be inserted in the flexures, and a mild degree of intertrigo consequently developed in the axilla and in the angle of the elbow. The affected muscles were of a wooden hardness. The circumference of the left arm was 13 inches; that of the right, when the biceps was

contracted as strongly as possible, was only 11½ inches, although the patient was right-handed. The increased size of the muscles was so great that the skin could hardly be raised from them, but there was no œdema, and the forearms were equal in circumference. The contracted muscles were not tender on pressure. No sound could be heard on auscultation of the muscles; there was no variation in the degree of contraction, and no twitching occurred on touching the skin or with excitement. There was no rigidity of the muscles of the jaw, neck, abdomen, or right arm. The constant spasm produced an aching pain which interfered with sleep. Major W. J. Turrell applied diathermy to the muscles, which relaxed slightly, so that they could be tested electrically; the reactions to both faradism and galvanism were normal and equal on both sides. A hot bath also produced slight temporary relaxation. Relaxation was very incomplete even under deep general anæsthesia. The skin over the contracted muscles felt warmer than on the other side, and the patient himself noticed the difference, but otherwise sensation was unaffected.

On August 23rd and September 7th and 11th, 1000 units of antitetanic serum were injected subcutaneously. The same quantity was given intrathecally on September 9th. The cerebrospinal fluid was under normal pressure; it contained no cells and had no effect when injected by Professor C. S. Sherrington into the muscles and nerves of cats.

By September 11th the muscles, except the pectorals, had considerably relaxed. The arm was now only painful at night. Its circumference was 11¾ inches, and by September 16th it was only 11¼. On the 22nd the deltoid and the outer part of the pectoralis were the only contracted muscles.

From this time the patient's condition steadily improved, but when I last saw him on November 30th the deformity caused by the contraction of the muscles had not completely disappeared, and he was unable to raise his arm above his shoulders in spite of exercises and passive movements. The increased size of the muscles had given place to definite atrophy, the circumference of the left arm being now only 10 inches. He felt very well, and was able to do light work in the garden.

Localised Tetanus Involving a Leg.—Pte. R., aged 21, received wounds of both legs on July 14th, 1916, from a canister grenade. A few hours later he received a prophylactic injection of antitetanic serum. Fragments of metal were found with the X-rays in various parts of both legs. All the wounds healed rapidly except one in the region of the left tendo Achillis, in which a sinus persisted.

On August 1st the patient noticed some twitching in the toes of his left foot, and soon afterwards cramp in the calf. On August 3rd he was given 1000 units of tetanus antitoxin intrathecally, on August 5th 8000 intramuscularly, and on the 8th the same dose subcutaneously. By this time the whole leg had become rigidly fixed with the knee flexed to a right angle and the foot and toes plantar-flexed. All the muscles of the leg and thigh were involved; they never relaxed completely, and in addition to their tonic contraction very painful spasms occurred whenever the limb was touched or the patient was in the least excited. The sound of footsteps near his bed was enough to cause a spasm. The muscles of the jaw, right leg, abdomen, neck and back were never affected. The spasms began to diminish in frequency about August 20th, but the tonic contraction did not abate until the beginning of September. By September 9th spasms only rarely occurred, but attempts to straighten the knee still caused very painful contractions. The tonic contractions had, however, by now diminished sufficiently for slight voluntary movements to be possible. In spite of the constant contraction of the muscles of the left leg, a considerable amount of wasting had taken place. On September 29th the left thigh and calf measured 16 and 11 inches respectively in circumference, the measurement on the right side being 17 and $11\frac{3}{4}$ inches. All spasmodic contractions had now ceased, but the muscles of the thigh and leg were still hypertonic, and the patient was unable to extend his leg completely or dorsi-flex his foot to a right angle. The knee-jerks on both sides were slightly but equally exaggerated; all other deep and superficial reflexes were normal, and there was no increase in the muscular irritability of the affected muscles, which only partially relaxed under deep anæsthesia.

As the sinus in the left foot had not yet healed, Captain G. R. Girlestone operated upon it on October 24th, and removed some fragments of cloth. The external saphenous nerve passed across the sinus and was doubtless the channel by which the tetanus toxin had ascended from the wound to the spinal cord. An injection of 500 units of tetanus antitoxin was given at the same time. As the patient was still unable to extend his knee voluntarily, it was forcibly straightened under the anæsthetic; as the flexor muscles offered considerable resistance and the leg bent again immediately the forcible extension was released, extension was maintained by a splint.

On November 20th the leg remained extended even without the splint, and all movements were possible. At first the patient would not put his foot to the ground, and would only move with

crutches, but with encouragement he was soon persuaded to walk with a stick.

Fatal Case of Localised Tetanus.—Lance-Corpl. T. L., aged 28, received several small superficial wounds in the left arm and right thigh on August 19th, 1916. His general condition was good and the wounds healed satisfactorily, except for one on the front of the right thigh. Some swelling was observed round this on September 3rd, and on the following afternoon, whilst he was walking in the ward, secondary hæmorrhage began from it. The vessel was ligatured and the patient appeared to be improving satisfactorily, when on September 9th painful spasmodic movements of the muscles in the front of the thigh occurred. The spasm extended, and when I first saw him on the 12th, the limb was rigidly fixed with the thigh and leg extended and the foot dorsi-flexed; clonic contractions occurred every two or three seconds, resulting in a slight degree of flexion at the hip and knee. The pain was now very acute and was only partially relieved by the injection of gr. ½ of morphia: the clonic contractions ceased, but the tonic contraction was unaffected. The patient's general condition was good, and there was no spasm elsewhere. On the 15th he was given an anæsthetic; the spasms soon ceased, but the rigidity of the left leg never entirely disappeared, and squeezing the thigh and calf always brought on spasms, even under deepest anæsthesia. After several cubic centimetres of clear cerebro-spinal fluid had slowly run out 6000 units of antitetanic serum were injected intraspinally; 3000 units were injected at the same time into the muscles above the wound, which was not touched, and 6000 units were injected intravenously. The wound showed signs of healing, but there was still some purulent discharge. The spasms returned directly after the anæsthetic, and the patient required large doses of chloretone to control the pain in the left leg. Four hours later the temperature rose to 105°, and the pulse became faster and weaker.

As the patient was very much worse the next day (16th), and the spasms and pain remained extremely severe, but still localised to the left leg, he was given an anæsthetic, and after draining off 15 c.c. of cerebro-spinal fluid, 10 c.c. of 25 per cent. magnesium sulphate were injected; no pain and no spasm occurred during the rest of the day, and no morphia nor chloretone was required. On the 17th the effect of the magnesium sulphate had passed off; the pulse was still weaker, and the patient appeared to be in a profoundly toxic condition. Three pints of normal saline solution were injected intravenously; the general condition and pulse greatly improved, and the patient regained consciousness for some

hours. On the 17th the general improvement was maintained, but the pain became more severe, although now the tonic contration was present without any spasms. On the 18th the pain was worse and the spasms had returned, so 10 c.c. of 25 per cent. magnesium sulphate were again injected into the spinal canal. Although the leg and foot remained rigidly extended, there was no pain, and squeezing the calf caused no spasm or pain. On the 19th the pain was worse again, and 3000 units of antitoxin were given intravenously, the dose being repeated on the 21st, 22nd, and 23rd. By the 23rd the spasms were rare and there was no pain, no more chloretone nor morphia being required; but the patient had again become mentally dull and confused and slept most of the day. The temperature, which had been raised since the 15th, fell nearly to normal. The typhoid condition became more and more profound; the tonic contraction in the left leg persisted, but there was no pain and no spasms occurred in other muscles. The patient died in coma on the 26th.

The cerebro-spinal fluid removed on the 15th was injected by Professor C. S. Sherrington into the nerves, spinal theca, and subcutaneous tissue of a number of cats. No symptoms of tetanus developed, but all were obviously ill; they refused food and their coat was ruffled; three recovered after three days, but the fourth, which had been inoculated into the peroneal nerve sheath, died in forty-eight hours with vomiting, but no local reaction.

Prophylaxis.—I have already described how the mortality of tetanus has fallen, the incubation period has become longer, and the relative frequency of localised tetanus has greatly increased as a result of prophylactic injections of antitetanic serum. The symptoms also tend to develop much more slowly. The following statistics show how the incidence of the disease has at the same time been enormously reduced. The proportion of cases of tetanus to the total number of wounded British troops arriving in England reached the high figures of 15·9 per 1000 in September, 1914, and 31·8 in October. The incidence at once fell when prophylactic injections of serum began to be used on a large scale in the middle of October: only 600 doses had been sent to France in August, 1914, and 12,000 in September, but 44,000 were sent in October, 112,000 in November, and 120,000 in December. Only 1·7 cases per 1000 wounded occurred in November, and 0·9 per 1000 in December, 1914; the incidence then remained constant, but during the last year it appears to have fallen still

further, owing to the more universal use of serum for even trivial wounds, and to the greater frequency with which repeated injections are given in septic wounds and additional injections are given before secondary operations.

Similar reports have been published by French and German observers. Bazy gives a striking example of the value of prophylactic inoculations. Owing to an accident only 100 out of a batch of 200 wounded men were given serum. Among the 100 who had been inoculated the only one who developed tetanus did so on the day of inoculation, before the serum had had a chance of exerting any prophylactic action, whereas eighteen cases occurred among the uninoculated 100, although their wounds were no more severe or septic.

Tetanus toxin does not appear to lead to any appreciable degree of active immunity in man. Consequently when the prophylactic dose of 500 U.S.A. units of antitoxin, which is generally given directly a soldier arrives at a dressing station or field ambulance, has disappeared from the circulation, any further toxin produced by tetanus bacilli still in the wound is free to act, unless the formation of a granulation-tissue barrier prevents its absorption. A second subcutaneous injection of 500 units should therefore be given in all cases of septic wounds seven days after the first injection, in order to anticipate the disappearance of the antitoxin from the body about the tenth day and the consequent loss of immunity. This should be done, however trivial the wound may appear to be, as in at least five out of twenty-five cases of tetanus seen in England by Dean the wounds were so slight that the most conscientious medical officer would hardly have thought of giving a second prophylactic injection.

In chronic septic wounds, especially those caused by shells or bombs, a third, fourth, or even more numerous injections should be given at seven-day intervals. The danger of anaphylaxis with prophylactic doses of 500 units given subcutaneously is quite negligible, whatever the interval may be between the injections.

If more than a week has elapsed since the last dose of antitoxin was given, an additional 1500 units should be injected whenever an operation is to be performed in the neighbourhood of the wound, even if it has completely healed. This should be done two days before the operation if time allows, as forty-eight

hours elapse before serum injected subcutaneously reaches its maximum concentration in the blood. If this delay is undesirable, the serum should be injected into the muscles, as only twelve hours then elapse before protection is secured, and when the operation is urgent the serum should be given intravenously. In each case additional serum should be injected at several points and at various depths into the muscles just on the proximal side of the wound.

Prophylactic injections of antitetanic serum should also be given in all cases of severe burns, and in trench foot whenever the skin is broken. The danger of tetanus as a complication is almost completely abolished when this is done.

Treatment.—*Antitetanic Serum.*—When symptoms of tetanus appear, some toxin must be present in the nerve-cells; some is still circulating in the blood, and a further quantity is probably still being formed by the tetanus bacilli in the wound, from which it continues to be absorbed. The object of treatment by antitoxin is to neutralise and destroy at once the toxin present in the circulation at the time of inoculation, after which a sufficient quantity should be maintained in the blood to neutralise further toxin directly it is absorbed from the wound. It is theoretically possible that some of the toxin actually combined in the nerve-cells may be dissociated, if the concentration of antitoxin in the surrounding fluid is sufficiently great, but it is doubtful if this object can ever be attained in actual practice.

The complicating factors, such as the varying severity of the wound, the presence and intensity of mixed infections, the inconstant dosage and times of administering the serum, render it impossible to draw any definite conclusions as to the relative value of subcutaneous, intramuscular, intravenous and intrathecal injections from statistics collected from records made by many different observers. The unexpected results of Leishman and Smallman's analysis of their statistics of cases treated in France, which seemed to indicate that the intramuscular and subcutaneous routes are preferable to the intravenous and intrathecal, thus lose most of their value, particularly in view of the fact that larger doses of serum are generally injected by the former methods. Moreover, Greenwood has shown that the variations of case mortality in the different groups in their statistics are such as

could arise by chance in a uniform population, so that no conclusions can be legitimately drawn from them as to the relative value of the different methods of giving antitoxin.

More reliance can be put on the conclusions formed by individual medical officers, who have had special opportunities of themselves treating a considerable number of cases. Thus the thirteen recoveries in Major H. R. Dean's fourteen cases of generalised tetanus treated by a single large intravenous injection cannot be due to any accidental conditions; they afford strong support of the value of the intravenous route. The excellent results obtained by Major A. G. Gibson and others with daily intrathecal injections also cannot be ignored.

Much has been written on the theoretical advantages of one or other method. There is no doubt that the slow absorption of subcutaneous injections is a grave disadvantage; though serum given by the intramuscular method is absorbed more rapidly, the delay in reaching the central nervous system is much greater than with intravenous injections. The intrathecal method has been recommended and also disparaged on theoretical grounds; on the one hand, it has been said that it is obviously the shortest and most direct route to the central nervous system; on the other hand, it is pointed out that the existence of channels of communication between the subarachnoid space and the perivascular and pericellular spaces in the central nervous system has not been anatomically demonstrated, and that the chief, if not the only, path of absorption from the spinal canal must be into the large intracranial venous sinuses and meningeal veins, through which the serum is carried away from the spinal cord and brain into the general circulation.

Such theoretical considerations are, however, of very small value when compared with the results of experiments on animals, from which it is possible to eliminate the disturbing factors, which bring confusion into the conclusions drawn from the results of treating the disease in man. Captain F. Golla has recently shown that the intrathecal and intravenous routes saved life much more readily than the subcutaneous route in cats and rabbits. Still more conclusive are the experiments of Professor C. S. Sherrington on monkeys. All but 3 of 25 monkeys which were inoculated with eight times the minimal lethal dose of tetanus toxin and

treated between 47 and 78 hours later, after symptoms of tetanus had begun to develop, by intramuscular injections of 2000 units of serum per kilo. of body weight, and all but 2 of 25 treated by subcutaneous injections, died ; whereas 14 out of 25 treated by intrathecal and 7 out of 25 by intravenous injections of the same dose recovered. In 5 of the 14 survivors of the intrathecal group the dose of antitoxin was given later than in any of the surviving cases of the intramuscular group. All of 10 monkeys treated by injection of serum under the cerebral dura-mater died. Permin had already found that the tetanus, which could be produced in dogs and rabbits by a certain dose of toxin, was prevented by giving antitoxin intrathecally at the same time, whereas the same dose given intravenously had no effect. Park and Nicoll obtained similar results with guinea-pigs. They gave antitoxin by various routes directly local spasms began as a result of the injection of double the minimal lethal dose of toxin. In one series of experiments, which is typical of the four which were carried out, two control animals which received no antitoxin and ten which received 100 or 200 units intravenously or intraneurally died ; but five out of six animals which received serum intrathecally recovered, although three received only 10 and three only 50 units.

Dean has shown that tetanus antitoxin injected by lumbar puncture in men spreads upwards within the subarachnoid space. The experiments on animals just described can leave no doubt that at any rate a part of the serum injected by lumbar puncture passes up the subarachnoid space and filters through to the cells of the medulla more rapidly and in larger quantity than when injected into a vein.

(i) *Subcutaneous and Intramuscular Injections.*—Absorption is slow from the subcutaneous tissues ; the quantity in the circulation does not reach its maximum until forty-eight hours have passed. It is more rapid from the muscles, but at least twelve hours elapse before absorption is complete. Both these methods, and especially the former, have the disadvantage when compared with intravenous injections of causing pain at the time and leaving behind a more or less tender area. Whatever method is adopted for administering the serum, an additional quantity may be injected at various depths into the muscles on the path from the

wound to the spinal cord, in the hope that some may be absorbed by the motor nerve-endings and then pass up the nerves to the spinal cord. For the same reason it has been recommended that serum should be injected direct into the chief motor nerve in the neighbourhood. But it is doubtful whether these measures have any value, as Embleton has pointed out that the serum does not pass up the axons like the toxin, but is conveyed in the perineural lymphatics, which leave the nerve when they reach the intervertebral foramina.

(ii) *Intravenous Injections.*—A very large dose should be given at once in order that all the free toxin in the body may be neutralised, and in the hope that some which has already reached the nerve-cells may be dissociated. It is, however, useless to expect any immediate improvement; the best that can be hoped for is that no more nerve-cells will be damaged. The recovery of those already affected leads to slow improvement, which may manifest itself by a fall in the pulse-rate in forty-eight hours, although diminution in the spasms is rarely obvious before the fourth and sometimes not until the seventh day. Dean recommends the immediate injection of 30,000 units into a vein. He found that the blood still contained antitoxin twenty, thirty and even thirty-nine days later, although no further serum had been injected. It is, therefore, generally unnecessary to repeat the injection, but in serious cases a second injection can be given after four or five days. In fourteen cases of generalised tetanus with well-marked symptoms, thirteen recovered and only one died; as the death occurred under the anæsthetic, though not as a result of anaphylaxis, all cases in which the serum had an opportunity of acting recovered. Six, who received one or more additional doses by intramuscular and subcutaneous injection, improved no more rapidly than the seven who each received a single injection. The technique of intravenous injections has already been described in connection with the use of anti-dysenteric serum (p. 162). The danger of anaphylaxis, which is almost negligible with subcutaneous and intramuscular injections and very slight with intrathecal injections, is undoubtedly present when the intravenous method is used, but with proper precautions it can be minimised. Undoubted cases of anaphylaxis have been extremely rare, in spite of the fact that the vast majority of men

who develop tetanus are hyper-sensitive owing to having already received a prophylactic dose of serum a short time before. The precautions already described in connection with antidysenteric serum (p. 161) should be used in all cases in which intravenous injections are given. Experimentally general anæsthesia greatly diminishes the danger. It is therefore best to act upon Dean's suggestion and always give the first large intravenous dose under a general anæsthetic. This has the additional advantage that an intrathecal injection can be given at the same time.

(iii) *Intrathecal Injections.*—As the antitoxin rapidly passes out of the cerebro-spinal fluid into the blood, frequent intrathecal injections are required in order to maintain a sufficient concentration in the central nervous system. The injection should, therefore, be repeated daily for three to six days, according to the severity of the case. Gibson has found that the diminution in the activity of the tendon reflexes forms a good basis for deciding when the injections can be safely discontinued.

The quantity which can be injected intrathecally is strictly limited. As the pressure of the cerebro-spinal fluid in tetanus is generally raised, between 20 and 40 c.c. can often be removed without danger; 15 to 20 c.c. of serum should then be injected very slowly under low pressure. As 20 c.c. of ordinary serum only contain about 3000 units, specially strong serum, prepared by redissolving the ammonium sulphate precipitate of globulin, which carries the antitoxin down with it, should be used; this allows 16,000 units to be introduced. When more than 20 c.c. of fluid escape, the relief of tension followed by the secretion of fresh fluid may account for some of the benefit ascribed to the intrathecal injections. The spasms may already begin to abate twenty-four hours after the injection.

The meningeal irritation sometimes produced by the injection of a large dose of a foreign protein may cause slight pain and stiffness, and the cerebro-spinal fluid becomes turbid with polymorphonuclear cells, but no harm ever results. In one case in Leishman and Smallman's series definite spinal meningitis was found post-mortem, but with proper attention to asepsis such an accident should be impossible. Leishman and Smallman also mention a case in which death rapidly followed an intrathecal

injection, when the patient was progressing satisfactorily, and I have also seen this occur. These deaths may perhaps have been due to anaphylaxis.

(iv) *Combined Method.*—From a consideration of the theoretical, experimental and clinical observations of others and my own clinical observations, I am convinced that the correct treatment of tetanus is to inject 30,000 units into a vein and 20 c.c. of the strongest available serum into the spinal canal under a general anæsthetic at the earliest possible moment. In severe cases 10,000 or more units may be injected at the same time into the muscles on the proximal side of the wound. The intraspinal injection should be repeated daily as long as may be necessary, and if the patient is not out of danger five days after the first intravenous injection, this should also be repeated.

General Treatment.—The patient should be kept in a quiet, darkened room by himself. As much fluid food as possible should be given, and the opportunity should always be taken of giving a pint of peptonised milk by a nasal tube whenever a general anæsthetic is administered for any purpose.

Drug Treatment.—Injections of carbolic acid, as first recommended in 1888 by Bacelli, have been much used in France, but the theoretical grounds for their employment are unsound, and the clinical evidence in their favour is quite unconvincing.

The use of drugs should be confined to the relief of violent spasm and insomnia. In my experience chloretone has proved most satisfactory. The spasms are relieved for several hours after an injection of 40 grains in 2 ounces of olive oil by rectum; the injection can be repeated three or four times a day. When the injections are not retained, chloral and bromide should be given by mouth, or morphia may be injected subcutaneously. Chloroform inhalations are useful when the spasms are exceptionally severe, and if feeding is impossible when awake, it can be carried out through a tube three times a day under light general anæsthesia. At the same time any serum which is to be given can be injected, and the wound can, if necessary, be dressed.

Magnesium sulphate has been injected intravenously, intrathecally, and subcutaneously in order to diminish the exaggerated reflex excitability of the central nervous system. It has no effect on the tetanus toxin, and although it has been extensively

used in America since Meltzer first employed it, it does not appear to have any advantages over such drugs as chloretone, which has none of the dangers which undoubtedly accompany the injection of magnesium sulphate.

Surgical Treatment.—The wound should be treated in the ordinary way, but experience has shown that all operations should be postponed unless they are very urgent, as interference is likely to disturb the granulation tissue which acts as a barrier to the absorption of tetanus toxin. Extensive operations, such as excision of the wound or amputation, which were formerly often carried out with the object of removing the source of the toxin, are now recognised as being more likely to aggravate the condition.

Treatment of Localised Tetanus.—Localised tetanus, if recognised early, should be treated like ordinary tetanus, as it is impossible to foretell whether generalisation will occur. If only recognised at a later stage, when the danger of generalisation is almost negligible, it is sufficient to inject 10,000 units intramuscularly; this may be followed by further injections of 5000 units at five-day intervals. The stiffness, which often persists for weeks or months after the acute stage has passed, requires regular passive and active exercises, preferably after hot-air baths or diathermy, which produce a certain amount of relaxation; but violent stretching under an anæsthetic only does harm. I have found that some improvement often results from suggestion, as the persistence of spasm may be in part hysterical in origin.

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CHAPTER XIX

SOLDIER'S HEART

Soldiers not infrequently suffer from symptoms due to functional circulatory disturbances during their period of training and still more often whilst on active service. In neither case do the symptoms differ from those which may occur among civilians, but their relative frequency has led to the adoption of the term "soldier's heart" to describe the various functional cardiac disorders specially common among soldiers. The effect of active service on the heart was first investigated during the American Civil War, and the present war has given an opportunity for a renewed study of the subject.

The importance of these conditions can be gathered from a return published under the War Pensions Act of 1915, which shows that 2503 out of 33,919 or 7·4 per cent. of the soldiers and sailors invalided from the services from the beginning of the war up to May 31st, 1916, were discharged on account of "heart disease."

Some confusion has arisen from the fact that different writers have described different conditions under the same designation, and that there is a tendency for each observer to regard the particular form of cardiac disorder in which he is specially interested as the only one of sufficient importance to be called the "soldier's heart." Although much new light has been thrown on the subject, the different varieties of soldier's heart cannot yet be defined with sufficient accuracy to make it possible to give a separate description of their etiology, symptoms, and treatment.

Etiology.—Soldier's heart is most commonly due to the effect of over-exertion, often associated with prolonged mental strain and insufficient sleep, on a heart and nervous system, which were already weak before the war or which have become weakened

on active service as a result of some form of toxæmia. Although physical strain, mental strain, and toxæmia generally act together, I have seen many cases in which one or other of these factors could be definitely excluded.

(*a*) *Feeble Circulation, Congenital or Acquired before the War.* (i) *Congenital Cardiac Insufficiency.*—A man may be born with a circulation which is sufficient for ordinary purposes, but which has a deficient amount of reserve power for increased strain. In such cases the peripheral circulation has generally been feeble from early childhood, as shown by the cold, damp, blue extremities, small pulse and distended veins. The chest is often narrow and badly developed, and the heart is small. The circulation is unable to cope with strenuous efforts, and the pulse becomes rapid on slight exertion. Individuals of this kind, having learnt their limitations, generally adopt a sedentary occupation in civil life and do not indulge in out-of-door sports. They are very liable to break down during military training, but if sufficient care is taken, they may develop into efficient soldiers, though they remain abnormally liable to develop cardiac symptoms under the conditions of active service.

(ii) *Feeble Development resulting from Sedentary Occupation.*—Out of 543 patients suffering from soldier's heart Lewis found that 306 or 57 per cent. were recruited from sedentary or light occupations. Some of them had given up heavier work on account of similar, but less severe, symptoms, which disappeared when they lived a less strenuous life and took comparatively little exercise. But apart from these individuals, who were apparently affected with the condition before they joined the Army, men who have followed a sedentary occupation are much more likely to break down during training than those who have previously done heavy work, unless special care is taken in the early days of training, as their hearts are less developed and have less reserve power.

(iii) *Congenital or Acquired Organic Disease.*—The majority of the men with definite evidence of congenital or acquired organic heart disease, who escape the vigilance of the recruiting medical officer, break down during training and are invalided from the service. But if the myocardium is unaffected, valvular disease may be so far compensated that the strenuous life of a soldier on active service is possible. Several cases have been observed

in which soldiers have carried out their duties satisfactorily at the front for many months, and a valvular lesion has only been discovered accidentally on admission to hospital for a wound or other cause. There is, however, no doubt that a man with valvular disease of the heart has less reserve power than a man with a normal heart, and any infection which he contracts is particularly likely to affect his heart, so that he develops symptoms when he tries to resume his former duties. It is probably wise, therefore, whenever old valvular disease is accidentally discovered in a sick or wounded man to reclassify him for home service, though there is no reason why he should be invalided from the army if he has had no symptoms of cardiac insufficiency. A history of rheumatism or chorea in childhood is often obtained in cases of soldier's heart in which there is no evidence of valvular disease; it is clear that the myocardium must have been weakened, and this naturally predisposes to the development of cardiac symptoms under the strain of active service.

(iii) *Old Heart Strain.*—In many cases of soldier's heart the patient admits that he has had similar but generally less severe symptoms in the past after taking unwonted exercise. Officers not infrequently say that they suffered from somewhat similar symptoms when fifteen or sixteen years old after too strenuous indulgence in long-distance or cross-country running. This has often been regarded as "heart strain," but careful inquiry generally shows that the heart was probably already in a weakened condition owing to some slight infection which immediately preceded the supposed strain or which was actually present at the time. Allbutt long ago pointed out that so-called heart strain is almost always due to the effect of stress upon hearts already enfeebled by infections, and he quotes Dr. Lambert of Harrow, who wrote in 1904 that he regarded "the specific fevers as the determinant of heart strain in schoolboys."

(*b*) *Intoxication.*—(i) *Toxæmia from Infections.*—The toxæmia is most frequently bacterial in origin, the symptoms dating from some preceding infective disease. This is sometimes a definitely recognised infection. Some cases have followed rheumatic fever, apart from those in which a history of rheumatism or chorea in childhood is obtained, but these should probably be regarded as examples of organic myocardial disease, although the

symptoms may be indistinguishable from those of soldier's heart. Dilatation of the heart and tachycardia may develop in the course of typhoid and paratyphoid fever; epidemic bacillary jaundice is often accompanied by signs of cardiac poisoning and was a common cause of soldier's heart at Gallipoli, though this complication is not observed after spirochætal jaundice; trench fever appeared to have little effect on the heart until the hot weather began in Salonica, when it was very frequently followed by cardiac symptoms associated with a moderate degree of dilatation, and more recently it has had similar results in France. Bacillary and amœbic dysentery may be followed by cardiac weakness, but less frequently than the non-specific intestinal disorders associated with diarrhœa and little or no rise of temperature, in which the nature of the infection is obscure. A history of malaria, recurrent tonsillitis, pneumonia, pleurisy or bronchitis, and occasionally of measles, diphtheria, or scarlet fever may be obtained. The improvement which has followed the removal of septic teeth and even of a chronically inflamed appendix has sometimes been so marked that it is probable that the heart was being poisoned by the toxins absorbed from the septic focus. It is, however, often impossible to ascertain the nature of the infection, which may have been described as "influenza," though true influenza has been rare among troops on active service, or simply as "pyrexia of unknown origin." In some cases the infection is not sufficiently severe to cause the soldier to report sick, and keen men often continue at duty in spite of having a febrile illness, which ought to be treated by complete rest. According to Weil, latent tuberculosis, whether of recent origin or of long standing, is a not uncommon cause of cardiac symptoms in French soldiers. A history of venereal disease is unexpectedly rare. When the condition is once present, any intercurrent infection is very likely to aggravate the symptoms.

(ii) *Excessive Smoking.*—I am convinced that the toxæmia which results from excessive smoking is often a very important factor, but it is not an essential one, as more than twenty cases of soldier's heart have been observed at the Hampstead Heart Hospital in non-smokers, and Macgregor states that the condition is as common among Sikhs, who do not smoke, as among other Indian troops. Many soldiers, especially those under twenty years old, smoke much more than they did before they joined the

army, and the custom of giving cigarettes to convalescent soldiers, whilst they are in hospital recovering from various infections, is responsible for much subsequent trouble, as the heart and nervous system, already poisoned by the toxins produced by the infection, are particularly liable to be further damaged by the toxins inhaled whilst smoking. Some patients discover for themselves that their symptoms are aggravated by smoking, and spontaneously reduce their allowance of cigarettes.

Parkinson and Koefod have investigated the immediate effect of smoking five cigarettes in forty minutes on the circulation of forty smokers, of whom thirty were suffering from soldier's heart and ten were healthy soldiers. The pulse rate was raised an average of nine beats during the smoking in the former class and six in the latter, and the blood-pressure was raised by 5 to 10 mm. Hg in both; no irregularity was produced, and respiration was unaffected. Two patients complained of precordial pain. The pulse-rate was more affected by exercise after smoking than before in the patients, but not in the controls, and half of the former, but only two of the latter were more breathless. Parkinson and Koefod conclude that excessive smoking often increases the breathlessness and precordial pain in men suffering from soldier's heart.

(iii) *Consumption of Alcohol.*—The statistics collected by Lewis at the Hampstead Heart Hospital reveal the unexpected fact that 53 per cent. of 454 patients suffering from soldier's heart were abstainers, and that those who are abstainers or drink very little do considerably worse than those who drink more heavily. Lewis suggests that this is due in part to the greater frequency of abstinence among men following sedentary occupations than among the stronger men who live a more active life. Although his statistics prove that alcohol does not in any way help in the development of soldier's heart, there is no doubt that it not infrequently gives rise to much more serious cardiac symptoms, but such men are so obviously suffering from something quite different from soldier's heart that they would rarely be sent to Hampstead, especially if other symptoms of alcoholic poisoning, such as delirium tremens, were simultaneously present.

(iv) *Cordite poisoning.*—In the South African War many soldiers purposely produced cardiac symptoms by eating cordite.

With the exception of a few cases which were observed in 1914, this has, I believe, been a very rare occurrence in the present war.

(v) *Gas poisoning.*—Cardiac symptoms occasionally date from gas poisoning. The toxæmia and the strain thrown upon the heart by the respiratory complications may cause it to be abnormally easily upset by exertion after apparent recovery and return to duty.

(vi) *Hormonic Type.*—In a small proportion of cases the intoxication is due to the excessive activity of the ductless glands, which results from the nerve strain of active service. This condition is described in detail in another chapter (p. 35). Cases have been reported of men taking thyroid pills in order to escape service, but this form of doping does not appear to have been practised after enlisting.

(*c*) *Over-exertion.*—Over-exertion is a relative term. A well-trained man can do work which would be impossible in the early stages of his training, and which would again be impossible if his heart and nervous system become damaged by the toxins produced by an infection, excessive smoking, or excessive activity of the thyroid and other ductless glands. The irritable heart of the recruit, which was at one time ascribed to restriction of the thoracic movement by the accoutrements and at a later date to a badly devised form of drill, but continued to occur with undiminished frequency after the accoutrements and drill had been altered, is to a large extent a result of attempting to train too rapidly. It is as common in the big guardsman who has outgrown his strength as in the under-developed and under-fed recruit from the slums. Skilful and graduated training would prevent the development of an irritable heart in peace time as in war.

The trained soldier very rarely develops cardiac symptoms as a result of over-exertion whilst on active service, unless the toxic factor is also present. But with the nervous system and heart enfeebled by the toxæmia, the physical exertion and mental strain of life in the trenches, which hitherto produced no ill-effects, are sufficient to cause nervous exhaustion and cardiac weakness.

(*d*) *The Nervous Factor.*—The nerve strain of active service affects the circulation, in part through the intermediation of the ductless glands, the activity of which may be so profoundly modified by prolonged strain to the nervous system that the condition

already referred to as the hormonic type of soldier's heart with its circulatory and other symptoms may result. But the nervous system may affect the circulation more directly. One of the worst cases of "soldier's heart" I have seen developed in a previously healthy soldier immediately after being blown up by a shell in November, 1914. He did not lose consciousness, but his heart at once began to palpitate and his breath became rapid and laboured. He had never before suffered from similar symptoms. His pulse continued to be rapid and the cardiac impulse was very forcible. The least excitement or exertion caused unpleasant palpitation, tremor of the eyelids and to a less extent of the hands, and flushing of the face. No improvement occurred even after he was invalided from the Army in May, 1916. I first saw him in August, 1916. Under hypnosis his heart became rapidly more quiet, until after five minutes it appeared to be perfectly normal. Repeated hypnosis and graduated exercises resulted in rapid recovery. I saw a soldier in Salonica who was suffering from severe paroxysmal attacks of tachycardia, especially at night. On cross-examination he admitted that they were invariably caused by a dream or the sudden recollection when awake of the horror he had experienced a year and a half before, when he had found the dead body of a man in the bedroom of his billet.

The soldier's heart is often only one manifestation of the nervous exhaustion or neurasthenia, which results from the combined effects of physical fatigue, mental strain, and toxæmia. The neurasthenia gives rise to an excessive irritability of the central nervous system, which causes slight circulatory disturbances to produce precordial discomfort, pain, and palpitation, although the actual condition of the heart is such that no subjective symptoms would occur in a man with a normal nervous system.

Symptoms.—As already mentioned, the onset of symptoms often dates from some febrile attack, the patient having returned to duty before he felt completely fit. In many cases actual cardiac symptoms are preceded, often for some weeks, by neurasthenic symptoms, which are due to the effect of toxins on the central nervous system, the resistance of which is frequently already weakened by prolonged physical and mental strain with insufficient rest. Physical and mental exhaustion occur very readily, and headache, lassitude, irritability, insomnia, and tremor

of the hands are common. The patient feels unfit even when at rest, and he is often greatly depressed about his health. Excessive sweating, especially of the palms of the hands and soles of the feet, is a frequent symptom both when at rest and with exercise. Salivation may also occur.

A moderate amount of exercise, which would formerly have produced no ill-effect, now gives rise to shortness of breath and exhaustion, which may be accompanied by giddiness and faintness. Giddiness may also occur on suddenly standing up. In some cases it is associated with a fall in blood-pressure, which is greater than occurs normally under the same conditions. The patient may actually faint after a fright or without obvious cause; the pulse becomes imperceptible, the heart beating at a rate of about 50; when it can be felt again the blood-pressure is found to be as low as 60 mm. Hg, the rate and pressure rising slowly to normal in the next half-hour. As Lewis points out, the slow pulse and low blood-pressure suggest that the attacks are vagal in origin.

More violent exertion causes dyspnœa and palpitation, which may last for some hours. In severer cases the patient gets out of breath after gentle exercise and palpitation may be troublesome even when he is at rest, especially on first getting into bed at night. The breathlessness manifests itself in a greatly accelerated respiratory rate, exertion which would hardly produce any effect in normal individuals increasing the rate to 40, 50, 60 or even 70 a minute, and ten minutes or more may elapse before the rate returns to normal. All the accessory muscles of respiration come into action, and the reality of the distress is shown by the anxious expression of the patient. Lewis and his fellow-workers have shown that the dyspnœa is generally due to deficiency of the sodium bicarbonate, sodium phosphates and protein of the blood, which normally combine with the CO_2 and other acids produced in metabolism and thereby prevent sudden changes in the reaction of the blood. They found that the blood takes up less CO_2 in loose chemical combination than normal blood; consequently the CO_2 produced by exercise makes the blood abnormally acid, and this acidity acts upon the respiratory centres and causes the rapid respiration. This view is confirmed by the fact that the patients could not hold their breath for more than about ten seconds and that inhalation of air containing 4·8 per cent. CO_2 produced

intolerable hyperpnœa, whereas 5·6 per cent. was required in normal individuals. The dyspnœa is thus an expression of disordered metabolism, which is possibly the result of abnormal thyroid or suprarenal secretion. The vasomotor centre is frequently affected, so that the hands and feet are always cold: at one time the fingers are shrivelled, white or blue, and numb; at another they are swollen, red and tingling. The white mark caused by pressure on the skin of the hand takes an abnormally long time to recover its colour.

Occasionally slight swelling of the ankles is present, but œdema is never a prominent symptom. Discomfort, which may amount to severe pain, is often felt in the neighbourhood of the heart, not only on exertion, but also when at rest. The pain is occasionally accompanied by referred hyperæsthesia and hyperalgesia in an area which varies between a single rib space and the greater part of the left side of the chest; it may even extend over the left arm. The hyperæsthesia is most common in men who have had rheumatic fever or who have recently recovered from some acute infection. The pain is likely to make the patient worry about himself, as he begins to think that his heart must be seriously diseased.

In some cases, especially when the condition is in part due to excessive smoking, there is persistent tachycardia. In primarily nervous cases there may be paroxysmal or persistent tachycardia, which varied in a case of Déjérine's between 160 and 180. More frequently the pulse is normal or only slightly increased in rate whilst resting, but the least exertion or excitement unduly accelerates it, and several minutes elapse before it returns to normal; sitting up in bed or standing may change a pulse of 70 to one of 100. Lewis has pointed out that if a patient and a normal individual do enough work to produce the same degree of dyspnœa, the former of course doing much less than the latter, the pulse rate and blood-pressure rise on an average to the same height in both. The blood-pressure is normal or only slightly raised when at rest, except in the hormonic type, which is constantly accompanied by hypertension. The pressure, however, is frequently high if the patient is examined when up, owing to the exaggerated response to exercise and excitement. The influence of emotion was well seen in a case I examined with Captain G. H. Hunt.

The blood-pressure for some weeks had constantly been about 160 mm. Hg and the pulse rate about 80. On each of two occasions on which he was hypnotised the pressure rapidly fell to 115 mm. and the rate to 68; both gradually rose to their original level after he woke up, though he was still lying quietly in bed. He was of a nervous temperament, but was not frightened or excited on the numerous occasions when his blood-pressure had been measured. In another apparently similar case there was no fall of blood-pressure, though the patient was as deeply hypnotised.

In many cases the size of the heart is normal, and orthodiagraphic observations at Hampstead have even shown that it is occasionally subnormal, doubtless owing to the patients having followed a sedentary occupation. But there may be definite dilatation of both sides of the heart, which is occasionally extreme. This is due to atony of the poisoned heart muscle. Garrod has pointed out that in the variety of soldier's heart which follows acute infections the cardiac dulness often extends $1\frac{1}{2}$ or 2 inches to the right of the middle line, though no conspicuous extension is present towards the left, except in extreme cases.

The simultaneous dilatation of the auriculo-ventricular ring may result in mitral imcompetence, shown by the presence of an apical systolic murmur, and the dilatation of the conus arteriosus gives rise to a systolic murmur in the pulmonary area, which is sometimes very loud and widely propagated. The presence of these murmurs often leads to the diagnosis of valvular disease of the heart, which is not only incorrect, but has also a very unfortunate effect on the mind of the patient. The murmurs are always systolic, and are generally much louder in the recumbent than in the erect position, in which the heart becomes elongated and the dilated chambers narrowed; in some cases they disappear completely on standing. They vary more with deep respiration, are diminished to a greater extent by pressure of the stethoscope, and are more exaggerated by emotions and after exertion than murmurs due to valvular disease. The researches of Lewis and his colleagues at Hampstead have shown that the importance of murmurs has been very much exaggerated in the past. Among 462 men suffering from soldier's heart, 196 or 42 per cent. had systolic murmurs. Of these 49 per cent. improved sufficiently to be discharged from hospital to some sort of duty, the proportion

being only 42 per cent. among those with no murmurs. Lewis concludes that "the presence or absence of systolic murmurs is of no value in estimating the soldier's capacity for work, irrespective of the character, conduction, or point of maximum audibility of the murmur." However true this may be for men who have been admitted to hospital suffering from symptoms of soldier's heart, it would be a mistake to extend it to soldiers in general or as a guide in the examination of recruits, as it is doubtful whether 1 per cent. of healthy soldiers have a murmur, whereas one is present in over 40 per cent. of men suffering from soldier's heart. There can be little doubt that a man with a "functional murmur," even if he is at the moment suffering from no symptoms of any kind, is much more likely to develop a soldier's heart whilst training or under the stress and strain of active service than a man without any murmur.

Grace Briscoe found that there is often a considerable increase in the number of leucocytes in the blood with relative as well as absolute lymphocytosis. In normal individuals the leucocyte count rose by an average of 15·4 per cent. after a certain amount of exercise, but the average rise in patients with soldier's heart was 33·6 per cent., that of the lymphocytes being double the rise in polymorphonuclear cells. The highest counts were obtained in severe and chronic cases, and the number of cells fell as improvement occurred. Levy found an average of 5,837,000 red corpuscles per c.mm. in 15 cases of soldier's heart; in 5 of these the number exceeded 6,000,000. The excessive number of corpuscles was not accompanied by any excess of hæmoglobin, the average percentage of which compared with the normal was 93·4, the average colour index being 0·8.

Diagnosis.—The functional disorders grouped together as soldier's heart must be differentiated from the three organic conditions which may cause cardiac symptoms in young men—infective endocarditis, chronic valvular disease and angina pectoris; the first and last of these are, however, very rare in soldiers. In functional disorders the temperature is generally normal, although there is often a history of an earlier pyrexial attack, and in some cases the infection is so chronic that a slight degree of pyrexia continues for many weeks, or recurrent short attacks of a slight degree of fever occur. In infective endocarditis, on the

other hand, the temperature is either constantly or intermittently raised. Whereas any murmurs which may be present in the soldier's heart are always systolic, and tend to become less marked as time goes on and the patient's condition improves, diastolic as well as systolic murmurs are often present in infective endocarditis, and new ones may develop owing to organic changes occurring in the valves; they tend to vary in character from week to week and to become more and more marked. Symptoms of infarction in various organs may occur, and the general condition of the patient is more serious. In chronic valvular disease there is almost always a history of true rheumatic fever, chorea, or occasionally of scarlet fever. Murmurs may be diastolic as well as systolic; they do not become fainter but may actually become louder as the patient's condition improves. More or less hypertrophy is present, which varies with the exact lesion, and the murmurs are as loud in the erect as in the recumbent position: the systolic apical murmur is traceable further into the axilla and often to the back, and pulmonary systolic murmurs are rare except in congenital heart disease. In the poisoned heart of soldiers a feeling of exhaustion often precedes the cardiac symptoms, whereas in chronic valvular disease dyspnœa is generally the first symptom and œdema is much more common. Angina pectoris is rare in young men, in whom it is almost always due to syphilis; pain which may be the only symptom, is usually substernal and not over or below the heart, as in soldier's heart, in which, moreover, the characteristic radiation is rarely observed.

Prognosis.—Men of feeble physique, who even in civil life have been unable to take much exercise without becoming dyspnœic and experiencing pain in the cardiac region, and have never been really fit from the time they joined the Army, are unlikely to improve sufficiently to make useful soldiers. With this exception the prognosis is good, even in apparently severe cases, and very few men should be invalided out of the Army for soldier's heart. Complete recovery requires treatment for a period varying between a fortnight in the mildest cases and three to nine months in severe cases.

The response to graduated exercise is the best guide to progress. The presence or absence of a systolic murmur is of no

value as a guide, but the occurrence and long continuance of tachycardia and dyspnœa after very slight exertion, the presence of persistent extensive hyperalgesia, and leucocytosis over 14,000 indicate that the case is severe and the immediate prognosis so bad that the man is unlikely to be of any further use in the Army, although ultimate recovery is almost certain to occur.

Prophylaxis.—Feebly developed recruits, who have hitherto followed a sedentary occupation and have taken comparatively little exercise, should not be trained too rapidly. Skilful handling of recruits results in many men, who would otherwise soon complain of cardiac symptoms, becoming strong and capable of considerable endurance. Soldiers should not be allowed to smoke excessively, and when suffering from any febrile disorder or from the slightest cardiac disturbance, they should not be allowed to smoke at all until they have completely recovered—and then only in strict moderation. Care should be taken not to allow convalescents from infective disorders to get up too soon. The heart should be frequently examined, and if any dilatation is observed the patient should be kept in bed until it has almost, if not completely, disappeared. The tonsils should be enucleated if recurrent attacks of acute tonsillitis occur, as the latter often lead to poisoning of the heart muscle.

Treatment.—The first essential in the treatment of soldier's heart is to encourage the patient and to convince him that his heart is not really diseased and that he will completely recover his health. In the numerous cases which have already been erroneously diagnosed as V.D.H.—valvular disease of the heart—this may be exceedingly difficult. It is therefore of the utmost importance that this diagnosis should only be made when there is no possibility of mistake about it. Even the commonly accepted official diagnosis of soldier's heart—D.A.H. or "disordered action of the heart"—is best avoided, as when the word "heart" has once been employed it becomes extremely difficult to eradicate the idea of heart disease from the soldier's mind, and it is comparatively rare for him to become an efficient fighting man again, although the actual cardiac condition is generally quite curable. For this reason I believe that some such diagnosis as "debility" is preferable; it is a perfectly correct one, as there is no doubt that the functional capacity of other parts of the

body as well as the heart is impaired. In many cases the cardiac condition is simply part of general neurasthenia, and a diagnosis of neurasthenia would be quite satisfactory, were it not for the unfortunate tendency of some medical officers to regard neurasthenia as synonymous with malingering.

In the acute stage rest in bed is necessary, but this should never be prolonged; even if the heart is still much dilated and the pulse rapid, the patient should be allowed to get up after a short initial period of rest, so long as the temperature is normal, except when the dilatation is a direct sequel of an infection, in which case more rest is required. He should be given graduated exercises, which should always be just insufficient to cause exhaustion, dyspnœa, or pain. Walking exercises first on the level and then on hills of increasing steepness are of great value. Major C. H. Benham organised graduated exercises of this sort for soldiers with functional cardiac disorders at a General Hospital in Salonica, and the results were very satisfactory. Whenever possible the exercise should be of a congenial nature and taken in the open air with a cheerful companion. Officers should be encouraged to golf, shoot or ride, whichever they prefer, and they should always rest for a time if they feel tired, out of breath or otherwise unwell. In convalescent hospitals non-commissioned officers and men with soldier's heart should not only be encouraged to play games, but they should be given a regular occupation, such as gardening, farm-work, or carpentering, for a prescribed length of time twice a day, and at a still later stage regular drill of gradually increasing duration is most valuable. A man should not be sent back to his unit until he is able to do an average day's work.

Cardiac stimulants, such as digitalis, do no good and may do harm. The only drug which is often useful is sodium or potassium bromide, which should be given in small doses, such as gr. v, two or three times a day for several weeks in the numerous cases in which the nervous system is irritable. A small dose of some hypnotic, such as medinal, with gr. x of acetyl-salicylic acid, will generally procure sleep if insomnia is present. If this is not effective, and especially if the patient is much worried by palpitation at night, treatment by suggestion is almost always successful.

The teeth should be put into good order; not infrequently

this results in the immediate disappearance of pain, which had been supposed to be of cardiac origin. The removal of infected tonsils or other septic foci, such as a chronically inflamed appendix, may also lead to rapid improvement and prevent the tendency to relapse which would otherwise have existed. Giddiness is often relieved by the support afforded by an abdominal belt, especially if the symptom is aggravated by standing and exercise.

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CHAPTER XX

WAR NEPHRITIS

Acute nephritis is a comparatively rare disease in civil life, an average of 13 male cases, only 5 of which were in men of military age, being admitted each year into St. Bartholomew's Hospital out of a total of 7000 patients. It does not appear to have been common in any previous campaign except the American Civil War, in which over 14,000 soldiers of the northern armies were invalided for nephritis, and to a less extent in the Franco-Prussian War. Up to June, 1915, there had been 1062 cases in the British Army in France and Flanders, a very large proportion of which had occurred since March. The numbers continued to increase until the beginning of 1916, after which there was a slight fall. Very few cases occurred among French troops until July, 1915, by which time the infection appears to have spread from the neighbouring British line. There were similar, but less severe, outbreaks among the troops at Gallipoli, except at Suvla and at Salonica, and in the Italian Army in 1916. The disease has also been very prevalent among German and Austrian soldiers on every front since the spring of 1915, but L. R. Müller says that not a single case has been observed in the Turkish Army. It is very rare among officers of all nationalities, but less so now than in the first year of the war. No true cases of war nephritis have occurred in training camps in England.

Definition.—War nephritis is a form of acute glomerular nephritis, which has occurred among soldiers on active service during the present war ; it differs in its great frequency, its comparatively favourable course, and the constancy of certain clinical characteristics from the acute nephritis observed among young adults in civil life. The condition has been called " trench nephritis," but the name is unsuitable, as there have been many

cases in men of the Army Service Corps, ammunition columns, headquarters troops, and R.A.M.C., who have never been in the trenches.

Etiology.—The majority of cases occur among soldiers who were previously healthy. There is no doubt that a former attack of nephritis renders an individual more liable to develop the disease, which also tends to be unusually severe. In a small number of cases a definite history of a previous attack of nephritis has been obtained, and in a few others the clinical symptoms or the autopsy points to the existence of chronic nephritis.

Exposure to cold or wet aggravates war nephritis when it has once developed, and may produce an acute attack in men whose kidneys are already damaged, but it is certainly not an important factor in the large majority of cases. The disease was rare in France during the first winter of the war in spite of the frequency of exposure to cold and wet; it only became common in March and April, during which months more cases occurred than in the preceding seven months, and the high incidence continued through the summer. The majority of cases of nephritis which occurred before March, 1915, were due to acute exacerbations in middle-aged men with renal disease, and were not genuine examples of war nephritis at all. Sudden changes in temperature also appear to be of small importance, as such changes have never been as extreme on the Western Front as in the South African and Russo-Japanese wars, in both of which nephritis was very rare. If cold were an important factor, Indian troops would probably suffer greatly from the disease, but only three cases were reported among them; this, however, is perhaps due in part to the protection against chills afforded by the loin cloth they constantly wear.

The nephritis might conceivably be due to metallic poisoning caused by the consumption of tinned rations, especially those which are cooked in the tins, although MacKenzie Wallis could find no trace of metallic poison in the urine. Powell White, using a different method, found lead in the urine of each of four cases of war nephritis which he examined, and tin in one of them. The latter was the most recent case and more lead was present than in the others; a month later there was less lead and no tin.

Oliver and Clague also sometimes found lead in the urine of soldiers with nephritis, but the importance of this was much reduced by the discovery of traces of the metal in the urine of 9 out of 29 soldiers suffering from other diseases. Powell White found that salt solution boiled in a fruit or meat tin extracts traces of lead and tin, the amount being greatly increased if the slightest trace of acid is present, and Oliver and Clague found a minute quantity of lead in bully beef and the Maconachie ration. Further investigations are required in order to show how much importance can be ascribed to these observations, but some other factor must be present to explain why the disease did not become prevalent before March, 1915, and why the Army at Suvla and the troops in the South African War escaped. Oliver, Clague and Watson have shown that traces of lead are absorbed from shrapnel, but not from fragments of shells or intact bullets, after it has been present in the tissues for three or four weeks. As over 1 mg. of lead may be excreted in the urine in 24 hours, the kidneys are likely to suffer in time, and Oliver believes that the anæmia and albuminuria which occasionally develops in wounded soldiers may be due to this cause, as improvement at once follows removal of the shrapnel. War nephritis is certainly not due to the chlorination of water, as it has been just as common among men who have never had any as among those who have constantly drunk it. In the Austrian Army soldiers have purposely taken cantharides and chromic acid in order to produce nephritis, on account of which they hoped to be discharged. Alcohol is not a factor in its production, as 13 out of Hogarth's 75 cases were total abstainers.

Acute tonsillitis and other infections may be followed by nephritis among soldiers as among civilians, but this only accounts for a very small proportion of the cases. Cultures from the throat in ten soldiers with nephritis did not differ from cultures from ten wounded soldiers, and in both cases the throat was less septic than in ten civilian patients who were in the same hospital for various surgical conditions (R. G. Cantie). Eighteen out of fifty-six cases gave a positive Wassermann reaction (Mackenzie Wallis), but it was negative in all of another series of 13 cases (Oliver); the proportion is very much greater than would be found in the Army generally, so that syphilis, which under normal conditions

gives rise to albuminuria in 4 per cent. of cases during the secondary stage, appears to predispose to war nephritis.

Diarrhœa frequently preceded the onset of nephritis in the troops at Gallipoli, but this was probably accidental and due to its extreme frequency, as no such history was obtained in the majority of cases occurring elsewhere. The urine is generally sterile; in a few cases the *B. coli* has been isolated, but this was probably due to coincident intestinal disturbances and is not likely to be of any pathological significance. The absence of urobilin and of any increase in ethereal sulphates in the urine is also against an intestinal infection. Bronchitis often precedes or accompanies the nephritis, but it was common among Indian troops in spite of their freedom from nephritis.

There is some evidence that the disease is contagious. Thus Davies and Weldon report that a sister and two orderlies, who were on duty in the nephritis wards of a General Hospital, contracted the disease, though they had never been to the front, and the laboratory attendant, who examined all the urines of nephritis cases, also developed nephritis. The contagion is not present in the soil, as a division already providing a moderate number of cases continues to do so when moved to a different part of the line; and another division, which has hitherto been comparatively immune, becomes no more affected than before when it takes over the trenches belonging to troops among whom the disease is well established (Dunn and McNee). There is, however, something about the conditions of life of the soldier as distinct from the civilian, which makes him specially liable to the disease, as it has not occurred among the civilian population or refugees of Belgium or France living in the midst of soldiers and with many soldiers billeted on them. This is in striking contrast to the parallel incidence of most epidemic diseases among civilians and soldiers.

The incidence of the disease is never very great. In the records examined by Dunn and McNee the largest numbers occurring in a battalion were 10 cases in $3\frac{1}{2}$ or 4 months. Other battalions in the same division would have 3 or 4 or perhaps not a single case in the same period. Thus only 1 case occurred in a certain brigade in seven months, though the other brigades of the division had an average number. Sometimes 3, 4 or

5 cases may occur in the course of a few days in a unit which has been free from nephritis for several months, and the outbreak is not necessarily followed by the occurrence of additional cases.

As the majority of cases occurring both in France and in the Eastern Mediterranean Forces resemble each other closely, whilst differing in many respects from the ordinary acute nephritis seen in civil life, it seems probable that the disease is due to a specific infection, which is perhaps identical with that which caused the epidemic in the American Civil War.

Morbid Anatomy.—Dunn and McNee have had unique opportunities of investigating the morbid anatomy of trench nephritis, as they have examined the organs of 35 cases, in which death occurred between the second and fourteenth day and in which there was no evidence, such as overgrowth of connective tissue, pointing to pre-existing renal disease. The pathological features were remarkably uniform. The appearance of the kidneys to the naked eye was almost normal. A few were slightly enlarged, and the cortex was often pale and the pyramids somewhat congested. The capsule was never adherent and the surface was smooth. The only constant change occurred in the capillary blood-vessels of all the glomeruli uniformly through the whole kidney. There was often some hyaline thickening of their walls; the lumen was dilated and more or less completely obstructed by numerous cells, consisting chiefly of swollen endothelial cells with a few polymorphonuclear leucocytes and lymphocytes, but few or no red corpuscles. The vascular obstruction gave rise to secondary fatty degeneration and catarrh of the glomerular epithelium and to a less extent of the epithelium of the tubules. There was no evidence of inflammatory reaction, no cellular or fibrinous infiltration of the connective tissue being visible, even round the glomeruli, but small hæmorrhages were occasionally found. Beyond the slight secondary changes already mentioned, the tubular epithelium was remarkably little affected, so that the epithelial casts must be formed from the agglomeration of cells which have been desquamated separately and not from cells which have separated together from one segment of a tubule. Some long hyaline casts and small collections of blood were found in the tubules.

The appearance of the kidneys suggests that the disease is due

to a poison which exerts a specially irritant action on the smaller blood-vessels. This would also explain the constancy of subcutaneous œdema, and the presence of multiple small hæmorrhages in most of the lungs, and in some of the brains and spleens of fatal cases. The œdema is due to increased permeability of the blood-vessels, which allows excess of fluid to pass into the tissues from the blood, the increased concentration of which is shown by the abnormally large number of red corpuscles per c.mm. in the early stages. Possibly the rise of blood-pressure is due to direct irritation of the blood-vessels by the same toxins, and a similar action on the cerebral blood-vessels may act with the cerebral œdema and increased intracranial pressure in producing uræmic symptoms. Though the morbid anatomy of trench nephritis is very characteristic and differs from that of most varieties of acute nephritis, it is almost identical with a form of acute glomerular nephritis, which has been described as an occasional sequel of pneumonia and scarlet fever.

In all but 2 out of the 22 cases, in which Dunn and McNee examined the lungs, pathological changes were present. In 16 characteristic alterations were found in addition to the œdema, which is constantly present, and the acute bronchitis or broncho-pneumonia which is one of the chief causes of death. The walls of the terminal bronchi with their infundibula and occasionally the mucous membrane of the bronchioles and some of the larger bronchi are swollen with œdema and the lining epithelium is lost, the surface being covered by a layer of material resembling fibrin. Many of the corresponding pulmonary capillaries are thrombosed, but no inflammatory reaction is present. There is no exudation into the alveoli in the absence of broncho-pneumonia, and, when this is present, other parts of the lungs generally show the characteristic changes just described, which have only been observed before as a result of the inhalation of irritant gas.

Symptoms.—The incubation period is said by Langdon Brown to be between six and fifteen days. The first symptoms are generally headache, œdema of the face and ankles, and breathlessness. The headache, more commonly frontal than occipital, may be sufficiently severe to prevent sleep; it is sometimes accompanied by vertigo. Œdema, which was uncommon in the cases which occurred before March, 1915, is almost always present;

but it is rarely universal and never sufficient to require puncture; slight ascites sometimes occurs, but hydrothorax is rare; pulmonary œdema is common. The breathlessness is a very constant and characteristic early symptom; it is at first only present on exertion, but it generally continues after the patient is taken into hospital, when it tends to be most marked at night. There is often some pain in the lower part of the back and the limbs. I have frequently noticed tenderness in the renal region on both sides, and in some cases the kidneys were palpable and appeared to be enlarged as well as tender. The tenderness is probably due to stretching of the inelastic capsule of the congested kidneys. The temperature may be raised to 100° or 101° at the onset, but numerous cases of equal severity occur, which are completely afebrile from the earliest stages. Sweating is much more frequent than in other forms of nephritis, and herpes labialis may occur. Bronchitis is often present and may cause a troublesome cough. The headache and perhaps the dyspnœa may be regarded as uræmic symptoms; nausea, vomiting, slight drowsiness and apathy are not uncommon. Epileptiform convulsions sometimes occur suddenly in apparently slight cases, generally early in the course of the illness, but the blood-pressure is then always high. Sundell and Nankivell have pointed out that severe uræmic convulsions, if not fatal within the first few hours, are generally followed after a few days of semi-coma by rapid recovery. An ammoniacal odour is often noticeable, and in one case of Bradford's the patient complained of a taste of ammonia. Amaurosis may occur, and I have seen one case with transitory acute mania. Cutaneous eruptions are rare, and cramps and hissing dyspnœa do not occur. I have seen one case of hysterical paraplegia which developed during convalescence from war nephritis; rapid recovery followed vigorous persuasion and re-education.

The blood-pressure is always found to be raised if taken in the evening. Those observers who have found the rise less constant have probably taken their records in the morning, as Captain R. G. Abercrombie has shown that there is a considerable diurnal variation in this form of nephritis, the pressure at 6 p.m. being often 20 to 60 mm. Hg higher than at 10 a.m. The increased severity of the headache and dyspnœa at night are probably

connected with the rise in blood-pressure. The increased pressure often only lasts from five to ten days, but in some cases it may continue for a few weeks. The morning systolic pressure is generally between 135 and 180 mm. Hg; it is highest when uræmic symptoms, especially convulsions, are prominent, and it may then be even 200 mm. Hg. According to Atkinson very high blood-pressures occur most frequently in men, who were plumbers or painters in civil life and may thus have had their kidneys previously damaged by chronic lead poisoning. The heart is not dilated, and cardiac hypertrophy and arterial changes rarely develop, owing perhaps to the fact, observed by Langdon Brown, that the pulse-pressure or difference between the systolic and diastolic blood-pressure is much greater than is usually the case in nephritis and in healthy individuals. True albuminuric retinitis only occurs in rare cases with persistent albuminuria and a high blood-pressure, when the disease appears to be becoming chronic. Slight hyperæmia and œdema of the optic disc is, however, not uncommon in the early stages, and Jessop saw several cases of transitory retinœdema, sometimes with plaques and occasionally detachment of the retina, but very rarely hæmorrhages or changes in the blood-vessels. The retinal changes disappear about the same time as the facial œdema. In cold weather severe bronchitis and broncho-pneumonia are not uncommon, pleurisy is rare, and pericarditis has not been recorded.

The urine may be at first scanty, especially when there is much œdema, and uræmic symptoms may be accompanied by suppression for twelve or twenty-four hours, but Dunn and McNee point out that in many cases there is little or no reduction in the quantity, and they record the case of a man dying from uræmia who passed 52 oz. of highly albuminous urine in twelve hours. The quantity of albumin varies greatly in different cases and from day to day in the same case, but it is often considerable at first. The amount often diminishes greatly in the first few days, and it may almost disappear in a week. Casts are generally found in large numbers; the majority are hyaline, many of which contain a few intact cells from the renal tubules; others are partly granular and partly epithelial; but fatty and blood casts are uncommon. A varying and occasionally considerable amount of blood is present; fever is generally well marked and œdema

slight or absent in cases in which the urine is at first smoky or red. There is often a good deal of pus ; this is probably of renal origin, as definite evidence of pyelitis is rarely present. A similar condition of the urine and the kidneys is very unusual in acute nephritis with the exception of that due to scarlet fever.

Improvement occurs rapidly. When fever is present the temperature generally falls in a few days, but may remain slightly raised for some weeks in the more prolonged cases. Sometimes the œdema only lasts two or three days ; it generally disappears within ten days and is rarely present longer than a fortnight ; by this time the patient often feels quite fit. Uræmic symptoms disappear within the same period ; the blood-pressure falls, the oliguria is replaced by polyuria, and the abnormal constituents of the urine diminish in quantity, disappearing completely in three or four weeks in most cases, although a little albumin, a few casts, and occasionally a good deal of blood may remain for a considerable period after all other symptoms have disappeared. Sometimes the albuminuria is intermittent before it finally disappears. In other cases blood may reappear in the urine and the albumin increase in quantity : other symptoms may return, but œdema is rare. A return of albuminuria and sometimes of blood, but without other symptoms, may occur after they have been absent for some weeks, apparently as a result of an indiscretion in diet or over-exertion.

According to Sundell and Nankivell there is generally a relative increase of large mononuclear cells in the blood with a slighter increase in eosinophile cells. Giroux and Quirin found that the blood contained excess of urea during the acute stage ; the percentage then gradually fell to normal, except when there was evidence of preceding chronic nephritis.

Diagnosis.—Before labelling a case of nephritis in a soldier as " war nephritis," it is necessary to exclude the ordinary causes, such as scarlet fever, acute tonsillitis, and in rare cases typhoid and paratyphoid fever, malaria, and dysentery. If jaundice is present, however slight, the nephritis is probably nothing more than one symptom of infection with *S. ictero-hæmorrhagica.* Owing to the frequency of syphilis among soldiers, it is probable that some cases diagnosed as war nephritis are really cases of syphilitic nephritis ; the recognition of such cases is extremely important, as they promptly respond to treatment with mercury

and iodides, whereas mercury generally aggravates the symptoms and increases the albuminuria in nephritis due to other causes.

Young soldiers are occasionally sent from their units to hospitals as cases of nephritis, but on examination of the urine no albumin is found. These are probably examples of " functional albuminuria " and not nephritis at all ; a man may have fallen out during a march on account of faintness, and examination of the urine has shown the presence of albumin. This is simply due to excessive exertion, and is comparable to the temporary albuminuria which frequently occurs after athletic contests. McLeod and Ameuille found that the incidence of albuminuria in apparently healthy British and French troops was between 1·6 and 4·7 per cent. under various conditions in France, but was as high as 10·1 per cent. among recent recruits during strenuous training in England, and Dixon found that 21 per cent. of 4085 patients admitted into a field ambulance in France during December, 1916, and January and February, 1917, had albuminuria, though in only 14·7 per cent. of these was there more than a cloud of albumin in the urine. Such cases can be sent back to their units after a short rest, as the albumin generally disappears in two or three days and almost always within a fortnight, and this form of albuminuria does not in any way predispose to nephritis.

The characteristic features of war nephritis are the transitory and comparatively slight œdema, the frequency of dyspnœa, the rarity of inflammatory complications and of the pale waxy swollen face characteristic of ordinary acute nephritis, and the low mortality in spite of the liability to sudden attacks of severe uræmic convulsions. It is important to distinguish war nephritis from acute exacerbations of ordinary chronic renal disease ; the latter occurs as a rule in older men, the heart is hypertrophied, the blood-vessels thickened, retinal changes are present, and œdema may be slight or absent.

Prognosis.—According to Bradford, death is extremely rare, not more than three or four patients dying per 1000 ; it is almost always due to uræmia, acute bronchitis or bronchopneumonia, and in a few cases chronic nephritis or a congenital abnormality of the kidneys has been found at the autopsy. Davies and Weldon, however, had a mortality of 4 per cent. among 664 cases treated between October, 1916, and March,

1917, in a General Hospital in France. As most of the deaths were due to bronchitis, broncho-pneumonia and lobar-pneumonia, it is possible that the renal condition was sometimes secondary and not really war nephritis at all, as the changes found in the kidneys differed considerably from what previous observers had described. Most patients get apparently well within four weeks, and complete recovery may occur even after six months. No case should be regarded as cured until albuminuria has been absent for three months in spite of a full diet and an average amount of muscular exertion. The ultimate fate of those in whom slight albuminuria persists for many months is still doubtful, but it seems probable that the majority of cases which last over twelve weeks become chronic. Of 166 cases investigated by Langdon Brown only two were fatal in the early stages, but two or three more died in relapses several months later.

Treatment.—Rest in bed, warmth, avoidance of the slightest constipation, and a diet consisting of milk, milk puddings and bread and butter are all that is required in most cases. A larger quantity of nitrogenous food should only be allowed when the headache and dyspnœa have disappeared; an ordinary diet can then be given whatever the condition of the urine. In the rare cases in which the œdema shows a tendency to persist a salt-free diet leads to its rapid disappearance.

The Wassermann reaction should be tested, and mercury and iodide given if it is positive. Any local infection, such as inflamed tonsils, sinus disease or diarrhœa should of course be treated.

Severe headache can be relieved instantaneously by lumbar puncture. Uræmic convulsions can be controlled by chloroform, and their return prevented by morphia, lumbar puncture, and by bleeding; in the exceptional cases in which the blood-pressure is not raised, saline solution should at the same time be injected intravenously. I have never seen any benefit follow the use of hot-air baths, and in three cases mentioned by Abercrombie fits appear to have been brought on by hot packs.

Owing to the danger of relapse on exposure to the hardships inseparable from active service, a man who has once suffered from acute nephritis should never return to more than light duty at home. Possibly, however, an exception could be made for very slight cases, which appear to recover completely within two or

three weeks. If the albuminuria persists for more than four months, the patient should be discharged from the Army, but a longer period may be allowed for young men in whom no cardio-vascular charges have developed.

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CHAPTER XXI

GAS-POISONING

Poisonous gases are produced by all explosives, but owing to their rapid diffusion the majority are harmless unless they collect in closed spaces such as dug-outs or cellars. Under such conditions carbon-monoxide poisoning may occur (*vide* Shell-shock). Shells which are specially constructed to set free asphyxiating and lachrymatory gases have been much used, but very little is known about their composition. The most serious form of gas-poisoning and the only one which will be considered in this chapter is that produced by heavy gases, which are set free in the enemy's trenches to drift with the wind. The employment of gas by the Germans compelled us, in self-defence, to use similar methods, as a result of which the enemy suffered heavy casualties. In Sir Douglas Haig's dispatch on the Somme offensive he states that our experience of the numerous experiments and trials necessary before gas could be used, and of the special training required for the personnel employed shows that the use of gas by the Germans was not the result of a desperate decision, but had been prepared for deliberately. Moreover, Professor Lehmann, a leading German pharmacologist, and his pupils published papers shortly before the war, which show that they were investigating the subject in great detail.

Asphyxiating gas was first used on April 22nd, 1915, in a German attack on Algerian and Zouave troops, who, being taken completely by surprise, broke before it. On the two following days attacks under cover of gas were made on Canadian and English soldiers in the neighbourhood of Ypres. Although no respirators were available and the losses were heavy, the troops held their ground. A few improvised respirators by soaking handkerchiefs in urine, the composition of which fortunately

rendered it a useful antidote. Another attack was made early in May; the men had now been provided with respirators, consisting of pads soaked in a solution of sodium bicarbonate, and these gave some protection. The third attack on May 24th and all subsequent ones have produced less serious results, as respirators of steadily increasing efficiency have been introduced.

The gas used has never been collected for analysis, but it is probably chlorine in most cases; indeed, a deposit of chlorides has been found on the buttons of gassed soldiers. Chlorine was chosen on account of its exceptionally irritating character; it rapidly puts a man out of action when inhaled in a strength of only 1 in 10,000, whereas sulphur dioxide is only effective in a concentration four times as great. Chlorine, being much heavier than air, readily drifts and sinks into trenches, dug-outs, and cellars. It can be rapidly manufactured in enormous quantities and is easily compressed into cylinders, in which it can be conveyed to the front.

The first effect produced by the irritant action of the gas is a profuse exudation of a thin, pale yellow, albuminous fluid by the bronchial mucous membrane, as well as a very active secretion by the lachrymal and salivary glands; these are the results of protective reflexes, the object of which is to dilute the irritant poison and render it innocuous. At the same time spasm of the bronchial muscles occurs in an attempt to obstruct the passage of the gas into the alveoli. In severe cases the bronchial secretion and spasm not only fail to protect the alveoli, but obstruct the entry of air into the lungs, to such an extent that the patient becomes asphyxiated and may die before the fluid is expectorated and the spasm relaxes. An autopsy at this stage shows slight congestion of the larynx and severe congestion and œdema of the trachea and larger bronchi, which are filled with frothy fluid. The lungs are intensely congested and œdematous, but the violent respiration caused by the asphyxia produces small patches of over-distended lung, seen on the surface as light-grey areas in the least damaged parts, into which air can still pass. The distended alveoli may rupture into the interstitial tissue, and air may spread into the mediastinum and even to the neck.

In all but the mildest cases the asphyxial stage is followed by a stage in which acute inflammation with profuse exudation of

lymph occurs as a result of the irritant action of the gas on the bronchial mucous membrane and the alveoli. If the patient dies in this stage the serous fluid in the bronchi is replaced by muco-pus, and more or less extensive broncho-pneumonia is found.

There is no conclusive evidence that the chlorine is absorbed by the blood and conveyed by it to other parts of the body. Nephritis has occasionally been found post-mortem, though there has very rarely been any clinical evidence of its presence; thus albumin and casts are rarely found, œdema never occurs, and only one case of uræmia has been recorded. According to Leonard Hill, the nephritis is not due, as suggested by Bradford and Elliott, to the toxic action of the gas after absorption; he regards it as a result of asphyxiation and analogous to the condition which results from temporary occlusion of the renal artery. At a later stage secondary toxic effects may be caused by absorption of the products of the pathological changes in the lungs. If death occurs in the earlier stages, the right side of the heart is greatly dilated and the brain and all the abdominal organs show marked congestion due to asphyxia. The mucous membrane of the stomach is red and covered with thick yellowish mucus, submucous hæmorrhages are common, and superficial erosions may be present: these changes are partly due to the asphyxia and partly to the irritant action of chlorine, dissolved in swallowed saliva and nasal and bronchial secretion.

Symptoms.—Lewis Freeman, a Canadian soldier, who was taken prisoner after being wounded and gassed in the attack near Ypres on April 23rd, 1915, and subsequently escaped from Germany, gave the following graphic description of his symptoms in the *Cornhill Magazine* for February, 1917. "I shall never forget the horrible agony of surprise in the eyes of the men who got that first dose. It was the look of a dog being suddenly beaten for something it hadn't done. They looked at each other with questioning eyes—I only recall hearing one man start cursing—then they began gulping and coughing, and then fell down with their faces in their hands. . . . My first sensation was of a smarting away up inside of my nose; this quickly extended to my throat, and then as my lungs suddenly seemed filled with red-hot needles, I was seized with a spasm of coughing. Coughing up red-hot needles is not exactly a pleasant operation, and the

pain was intense. Mercifully, it was only a few minutes before a sort of stupor seemed to come on, but even as I passed into half-unconsciousness I was aware of my outraged lungs revolting, in heaves that shook my frame against the poison that had swamped the trench. With some of my comrades the fighting instinct was the last thing that died, and I have a sort of recollection of two or three of them clutching at the parapet and firing from cough-shaken shoulders off into the depths of the rolling yellow gas clouds. . . . I had rolled and writhed, in the agony of the pain of the gas in my lungs, in a pool of slush in the bottom of the trench, and it must have been the lying with my face buried in the shoulder of my wet woollen tunic that saved my life. Most of my comrades were quite unconscious when the Huns, with their heads protected by baggy 'snods,' came pouring into the trench, but I had enough of my senses left unparalysed to be able to watch them in a hazy sort of way."

The sensation of suffocation, which follows the initial burning pain in the nose, throat, and eyes, is accompanied by pain, which is often severe, in the chest, especially behind the sternum. The irritation of the throat leads to coughing, and that of the eyes to profuse lachrymation. Respiration becomes painful, rapid, and difficult. Retching is common and may be followed by vomiting, which gives temporary relief. The lips and mouth are parched and the tongue is covered with a thick dry fur. Severe headache rapidly follows with a feeling of great weakness in the legs; if the patient gives way to this and lies down, he is likely to inhale still more chlorine, as the heavy gas is most concentrated near the ground. In severe poisoning unconsciousness follows; nothing more is known about the cases which prove fatal on the field within the first few hours of the "gassing," except that the face assumes a pale greenish-yellow colour. When a man lives long enough to be admitted into a clearing station, he is conscious, but restless; his face is violet red, and his ears and finger nails blue; his expression is strained and anxious as he gasps for breath. He tries to get relief by sitting up with his head thrown back, or he lies in an exhausted condition, sometimes on his side with his head over the edge of the stretcher in order to help the escape of fluid from the lungs. His skin is cold and his temperature subnormal; the pulse is full and rarely over 100. Respiration is

jerky, shallow and rapid, the rate being often over 40 and sometimes even 80 a minute ; all the auxiliary muscles come into play, the chest being over-distended at the height of inspiration and, as in asthma, only slightly less distended in extreme expiration. Frequent and painful coughing occurs and some frothy sputum is brought up. The lungs are less resonant than normal, but not actually dull, and fine râles with occasional rhonchi and harsh but not bronchial breathing are heard, especially over the back and sides.

Headache is generally severe, and there is also considerable epigastric discomfort, due partly to the strain of coughing and partly to gastric irritation, as it is increased if an attempt is made to eat.

The intense dyspnœa of this asphyxial stage lasts about thirty-six hours, after which it gradually subsides, if death does not occur before. The patient, exhausted from his fight for breath, then falls asleep and wakes up feeling much relieved.

A few hours later acute bronchitis or broncho-pneumonia develops. In severe cases the quiescent interval is short and the bronchitis very severe. The sputum is now viscid, yellow or greenish, and muco-purulent with occasional streaks of blood. Respiration becomes more shallow and rapid, and the rate may finally be even 70 or 80 a minute. The pulse is small and very rapid ; the temperature rises, and is often as high as 104°. The patient may now become delirious. Pleurisy may occur, and in some instances empyema and gangrene of the lung have followed.

After recovery from the bronchitis and pneumonia the patient remains weak and exhausted for a considerable time. He gets tired very rapidly and is unable to walk quickly or uphill without getting short of breath, even after the last signs of bronchitis have disappeared. He may continue to have attacks of dyspnœa and cyanosis for several weeks. The frightful experience he has passed through often affects his nervous system, and some of the attacks are doubtless aggravated by apprehension. Headache, vertigo, and dyspepsia may continue for several weeks.

Colard and Spehl have shown that the number of red corpuscles in the blood is sometimes increased. In severe cases Miller and Rainy found that a relative increase of small lymphocytes occurs without any increase in the total number of white blood-corpuscles.

The change may persist for eighteen months or more if respiratory, digestive, or nervous symptoms are present. The coagulability of the blood is increased; this may result in femoral thrombosis, and Giroux has recorded a case of hemiplegia following gassing, apparently as a result of cerebral thrombosis.

I have seen two cases in which hysterical symptoms followed gassing. A sergeant, 27 years old, was gassed on February 20th. When he recovered from the acute symptoms he complained of pain in the chest and hoarseness. He then became aphonic, and he was sent home as a case of aortic aneurism. He was kept in bed for several months, and was still completely aphonic when I first saw him on September 10th. I told him he would be cured by the treatment I would give him; I applied faradism to the interior of his larynx, and in two minutes he was talking normally. The aphonia resulting from the laryngitis, caused by the inhalation of irritating gas, was perpetuated by auto-suggestion and by the suggestion of the medical officers who diagnosed aneurism, the patient being abnormally suggestible as a result of the strain of active service and the horror of being gassed. The symptom being due to suggestion was readily cured by counter-suggestion.

The second patient was gassed on July 2nd, and was unconscious for several hours. Three weeks later when he tried to get out of bed for the first time, he could not stand or walk without assistance, and his gait was like that of a drunken man. He was sent to my section as a case of cerebellar tumour. Captain J. L. R. Symns, having recognised the hysterical nature of the paralysis, cured him completely in forty-eight hours by vigorous suggestion with the aid of faradic stimulation.

Prognosis.—Nothing is known as to the proportion of men who die from "gassing" on the field. Before efficient respirators were in use about 5 per cent. of those who reached the clearing stations died within forty-eight hours. Of those who reached the base hospitals between 1 and 2 per cent. died in the second or third week from broncho-pneumonia or other pulmonary complications. The mildest cases are often fit for light duty after a short period of rest, but they should not be sent back until all adventitious sounds have disappeared from the lungs. A considerable time elapses before complete restoration of health occurs in the more

severe cases, and it is still doubtful whether more or less permanent incapacity may not sometimes follow.

Prophylaxis.—The introduction of efficient respirators has almost abolished the danger of drift gas. Regular drill in the use of the respirators and inspection to see that they are in good condition are most important, as it takes time to get accustomed to breathing whilst wearing a respirator, and a damaged respirator may be worse than useless.

Treatment.—The patient should be kept warm with extra blankets, hot-water bottles and hot drinks, and his bed should be near an open window or out of doors. Owing to the irritated condition of the stomach a fluid diet should be given at first. Absolute rest is of the greatest importance.

In severe cases an effort should at once be made to expel the fluid, which is drowning the patient, from the lungs, and to allow the air to escape from the over-distended portions of the lungs by forcing it through the obstructed bronchi. This can be done by artificial respiration, repeated whenever the dyspnœa becomes excessive. After squeezing the fluid out of the lungs, it may be necessary to blow air in from mouth to mouth in order to overcome the resistance of the froth in the smaller bronchi. An apparatus was introduced by Leonard Hill for use in collapsed and unconscious cases : a foot-pump feeds a face-mask through a flexible tube ; by each downstroke a measured volume of air or oxygen is pumped into the lungs, and by each upstroke a valve is opened which allows the air to escape by the elastic recoil of the thorax and lungs. From time to time the fluid is evacuated by squeezing the thorax and hanging the head over the side of the stretcher.

Unless the patient is collapsed or unconscious, vomiting gives great relief by expelling large quantities of yellowish frothy fluid from the lungs ; if this does not occur spontaneously, the patient puts his finger down his throat after drinking half a pint of warm salt water. Ipecacuanha and apomorphine should not be used.

The inhalation of oxygen relieves cyanosis and improves the patient's condition. But it is very difficult to get a patient who is fighting for breath to tolerate any form of mask, without which it is impossible to give oxygen really efficiently. Administered in the ordinary way through an open funnel held near

the patient's face the oxygen in the alveolar air is only increased by 1 or 2 per cent., whereas by using a mask the alveolar air should contain as much as 70 per cent. of oxygen. It should be given continuously for hours or even days.

Theoretically atropine should help to diminish bronchial spasm and secretion during the first twenty-four hours; but it has been found useless in severe cases and disappointing in slighter ones. Atropine is certainly valuable in the attacks of dyspnœa which may occur during convalescence, and I have found that stramonium taken regularly diminishes the liability to these attacks; potassium iodide in small doses is also useful.

Inhalations of ammonia are useful in the earliest stages, and after the second day ammonium carbonate in doses of gr. v every three hours produces copious expectoration, which results in improvement of colour and considerable relief. When great restlessness and mental distress are present, morphia should be injected.

Extreme cyanosis with a full pulse is greatly relieved by bleeding: breathing becomes easier, headache is relieved, and the patient falls into a refreshing sleep. Lian and Hebblethwaite found that the effect is most marked if venesection is performed in the first few hours. From 15 to 25 ozs. of blood should be slowly removed. The blood is dark and coagulates with abnormal rapidity. Bleeding is contra-indicated if the patient is pale and collapsed.

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