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NERVOUS AFFECTIONS OF THE HAND

AND

OTHER CLINICAL STUDIES

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OF

THE HAND

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OTHER CLINICAL STUDIES

BY

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1897

TO

SAMUEL WILKS, ESQ.

M.D., LL.D., F.R.S.

PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS

THIS WORK IS DEDICATED

WITH GREAT RESPECT
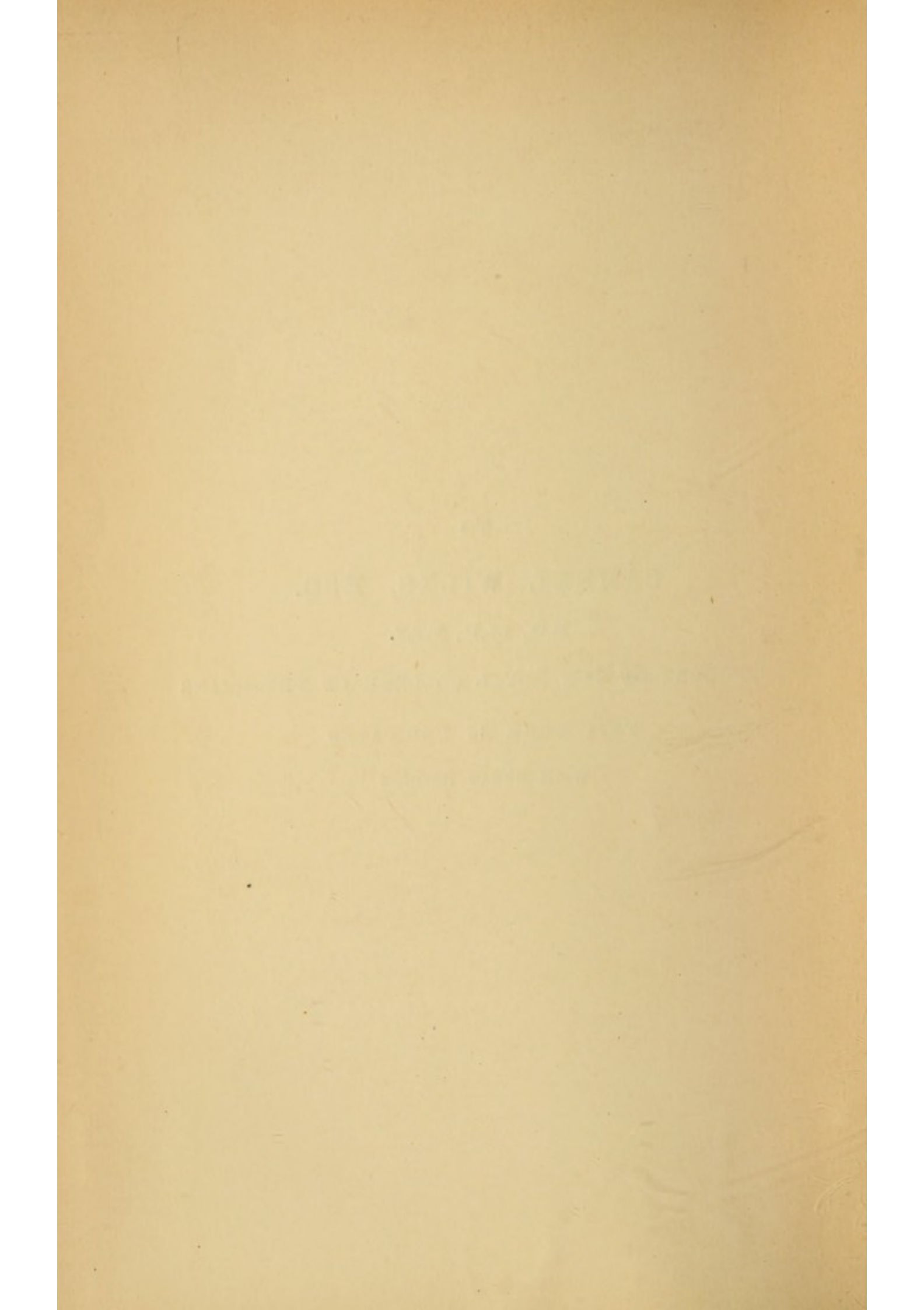
PREFACE.

THE Lectures and Papers which have been collected in this volume have all been previously published. For such previous publication the author is indebted to the Editors of the 'Lancet,' 'British Medical Journal,' 'Clinical Journal,' 'Practitioner,' 'International Clinics,' 'Brain,' and 'Medical Chronicle.'

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    April 1897.

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NERVOUS AFFECTIONS OF THE HAND

CHAPTER I

THE BRADSHAWE LECTURE, DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS, 1881

[BEFORE beginning to discuss the subject which I have chosen for the first Bradshawe Lecture I feel that a few words about him in whose memory this lecture was founded will not be unacceptable.

William Woode Bradshawe studied medicine at the Westminster and Middlesex Hospitals, and became M.D. of Erlangen and M.R.C.S. in 1833, and an Extra-Licentiate of this College in 1841. About this time he was practising at Andover, and married there a widow lady, whose means were sufficient to render him practically independent of his profession. He subsequently moved from Andover to Reading, where he continued to reside till his death on August 18, 1866. He appears to have made use of his ease and leisure for the cultivation of his mind. He entered at New Inn Hall, Oxford, and ultimately became M.A. and D.C.L. He was elected a Fellow of the Royal College of Surgeons in 1854, and became a Member of this College in 1859. Dr. Bradshawe's professional writings comprised papers on the use of cod-liver oil, on narcotics, and on abdominal abscess. He also wrote upon matters of general interest, and was an occasional contributor to the magazines. Of his domestic character the best evidence is the reverence shown for his memory by his widow. This lady, who survived him some fourteen years, bequeathed to this College, and also to the College of Surgeons,

£1,000, for the endowment of a lecture upon some subject connected with physic or surgery, to be called the 'Bradshawe' Lecture, and to be delivered annually upon August 18, the anniversary of the death of William Woode Bradshawe. The already existing endowed lectures of this College have been of great service to scientific medicine, and it will be a satisfaction to Mrs. Bradshawe's relatives to know that by founding this lecture, which I have the honour to inaugurate, our munificent benefactress has conferred what cannot but be of permanent benefit to the profession of medicine, and indirectly to the public. It is unfortunate that by the terms of the will the date for the delivery of the lecture has been rigidly fixed for what is nearly the middle of the medical vacation. I feel deeply the honour and responsibility of being called upon to deliver the first 'Bradshawe' Lecture, and although I dare not question the judgment of our President, I am very conscious that, in spite of every effort, I can barely hope to justify the choice which he has made.]

I have chosen for my subject the 'Nervous Affections of the Hand,' because I have enjoyed somewhat unusual opportunities of studying them, and the reflections and suggestions which I shall have the honour of making have been prompted by the study of 160 cases which I have seen during the past ten years. I have thought it best to proceed methodically, but in the course of an hour I can do little more than open the subject and lay down some fundamental principles, and I trust to be able to find some future opportunity of completing what I have to say.

For a thorough understanding of the nervous affections of the hand a knowledge of the nervous relations of the hand is necessary. That form of the manifestation of nerve-force which we call the will seems, in its passage to the upper limb, to be in some way intimately connected with certain parts of the brain-cortex on either side of the fissure of Rolando in the so-called ascending frontal and ascending parietal convolutions. This area of grey matter lies below the area which influences the lower limb, above that connected with the face, and behind that which seems to influence movements of the

head and eye. Of the upper limb area itself the fore part seems to control extension movements; the middle part, mainly in front of the fissure of Rolando, seems to control adduction, abduction, flexion, pronation, and supination; while the hinder part, entirely behind the fissure of Rolando, is said to control the special movements of the hand. Hitzig, Ferrier, and their followers have shown that stimulation of these convolutions produces movement in the opposite upper limb, and clinical and pathological observations are daily confirming the teaching of these physiologists. From these convolutions, the will, on its way to the upper limb, seems to pass through the internal capsule of the same side; thence along the lower layers of the crus cerebri, through the pons, to the anterior pyramid of the medulla oblongata. At this point the fibres forming the will-path divide. The greater part cross over to reach the opposite side of the spinal cord, while the smaller portion proceed direct to the same side of the spinal cord. The crossed fibres occupy in the cord the hinder central portion of the lateral column, while the direct fibres lie close to the edge of the anterior fissure. Between the fourth cervical and first dorsal nerves is the cervical enlargement of the spinal cord, and here those fibres which seem to originate in the grey matter of the brain presumably form connections with the grey matter of the enlargement, and, issuing from the cord with the anterior efferent roots, they join the afferent posterior roots, and leave the spinal canal to form the brachial plexus. The brachial plexus is a complicated interlacement of nerves, and I have made a hypothetical model of it in different coloured wools in order to show the greatest conceivable complexity which can exist in it. The plexus is formed by the fifth, sixth, seventh, and eighth cervical and the first dorsal nerves, and I have assumed that wherever these nerves effect a junction there is a complete interchange of fibres. The nerves are represented respectively by black, yellow, white, red, and green colours. The fifth and sixth cervical unite, forming a black and yellow cord, and, being immediately joined by the seventh cervical, there results a cord containing black, yellow, and white fibres.

The last cervical and first dorsal unite, forming a red and green cord. From the black and yellow nerves come branches which supply muscles used for respiration—viz. the diaphragm by the communicating phrenic branch, the rhomboids which fix the scapula during extraordinary respiratory efforts, and the scaleni, serratus magnus, and subclavius, which elevate the ribs. The black-yellow-white and the red-green cords each give off a branch to form the posterior cord of the plexus. This cord possibly contains all the five colours, and from it arise the three subscapular, circumflex, and musculo-spiral nerves. These nerves, therefore, possibly correspond, as it were, to the whole of the cervical enlargement of the cord. The median nerve in like manner is also formed by a branch from both the inner and outer cords, contains all the five colours, and presumably corresponds to the whole of the enlargement. It is interesting to note that the musculo-spiral and median nerves, which so largely antagonise each other, both presumably are connected with equal lengths of the cervical enlargement of the cord. The musculo-cutaneous nerve which arises from the outer cord, and contains only black, yellow, and white fibres, is connected with not more than the upper three-fifths of the enlargement ; while the ulnar nerve, which contains only red and green fibres, is connected with not more than the lower two-fifths of the cervical enlargement. The practical fact to be remembered is this, that the motor and sensory branches of the median and musculo-spiral have relations possibly with the whole of the cervical enlargement, whereas the motor and sensory branches of the musculo-cutaneous represent, as it were, not more than the upper three-fifths, and the motor and sensory branches of the ulnar not more than the lower two-fifths of the cervical enlargement. These speculations, founded upon coarse anatomical facts, receive a large amount of confirmation from the recent experiments of Professors Ferrier and Gerald Yeo. These gentlemen noted the muscles which were made to contract when each of the anterior roots of the nerves form-ing the brachial plexus of the monkey was stimulated. The result of their experiments was shortly this. No ulnar

movement occurred when any of the roots above the eighth cervical were stimulated ; no musculo-cutaneous movement followed stimulation of any roots below the fifth cervical, but both median and musculo-spiral movement followed stimulation of the fifth, sixth, seventh, and eighth cervical nerves. These experiments, therefore, bear out the speculation already made that the median and musculo-spiral have a wide origin from the cervical enlargement, while the musculo-cutaneous is limited to the upper and the ulnar to the lower end. One of the effects of such an interlacement of fibres as is found in the brachial plexus is necessarily to scatter, as it were, the influence of the motor cells of the spinal cord, so that the cells of a particular level have a widely diffused action on the muscles of the limb. We may conclude—1. That as many muscles receive their innervation through the cells corresponding with more than one nerve root, the function of these muscles will remain, in some degree, as long as any of the cells are intact from which any nervous influence is derived. 2. That when the motor cells in a limited area are destroyed the abolition of function is extremely limited, while impairment of function is far more extended.[1]

We are thus enabled to explain what takes place when, at a certain level in the cord, the motor cells in the front horn are destroyed, as in the disease which it is now the fashion to call 'anterior poliomyelitis,' but which has long been known as infantile or spinal paralysis. In this disease we constantly find, after the acute stage has subsided, a few paralysed muscles surrounded by others which are merely paretic. The paralysed muscles probably derived their entire motor power through the cells which have been destroyed, while the paretic muscles were probably dependent upon the destroyed cells for only part of their motor power. Let us suppose that the motor cells at the level of the first dorsal nerve have been destroyed. We should expect to find in such a case a complete paralysis of some muscles in the hand, while certain muscles supplied by nerves into which fibres of the first dorsal

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[1] See p. 132 for a description of Herringham's conclusions on this subject.

penetrate would be to a greater or less extent weakened. The improvement which always takes place in the paretic muscles in anterior poliomyelitis is probably due to the fact that the power lost is in time compensated by an increased conduction of force through fibres of another colour. The co-existence in infantile paralysis of paralysed and merely weakened muscles is a fact of much practical importance.

Clinical observation has led Remak to think that the motor cells in the cervical enlargement are arranged in functional groups. The group for the deltoid, biceps, brachialis anticus, and supinators he places in the upper part, and that for the extensors of the digits and the intrinsic muscles of the hand in the middle part of the cervical enlargement. A study of the brachial plexus seems to render it unlikely that any limited lesion of the cord would cause complete paralysis of any considerable group of muscles ; the structure of the plexus seems designed, among other things, to prevent such a catastrophe. On the other hand, it is likely that a lesion occurring at any of the junctions of the plexus might cause curious combinations of muscular paralysis ; and indeed Hoedemaker, who has recorded two cases of Erb's paralysis (deltoid, biceps, brachialis anticus, and supinators), is inclined to place the lesion, not in the spinal cord, but at the point of junction of the fifth and sixth cervical nerves, and the fact that faradisation of a spot in the neck close to this point causes simultaneous contraction of the muscles concerned in Erb's paralysis is in favour of Hoedemaker's theory.

The junctions of the nerves are of importance in another connexion. It is well known that inflammatory action occurring in a nerve may travel in either direction along the nerve trunk, and thus an ascending neuritis may strike a junction and seriously impair the function of other nerves emanating from that junction ; records of such occurrences are tolerably common. Weir Mitchell gives the history of Stephen Warner, who was shot through the left chest by a bullet. Three days later the movements of the arm below the shoulder were perfect, the pectoral muscles being alone paralysed. Shortly

there ensued a neuralgia which spread to the median region of the arm and hand, and was soon followed by wasting and weakness of the flexors of the forearm and hand. Mitchell thinks that the external anterior thoracic nerve had been wounded, and that neuritis travelling along this nerve reached the external cord of the plexus, and involved the musculo-cutaneous and median trunks, which were distinctly tender. Duchenne, Putzel, Cæsar Boeck, and others have recorded similar cases. Inflammation once set up in a nerve seems to have a potentiality for evil not unlike that of a clot in an artery or vein.

It must not be forgotten that the nerves of the brachial plexus, as they issue from the spinal canal, form connexions with the sympathetic chiefly by branches from the inferior cervical ganglion. The many trophic changes occurring in the upper limb in consequence of nerve lesions give importance to this connexion between the nerves of sensation and muscle-motion and those of vessel-motion.

It is not necessary to dwell upon the origin and distribution of the nerves of the arm and hand. A few words may be said, however, upon the effect of paralysis of these nerves on the attitude and movement of the hand. When the median is paralysed we get an abolition of pronation and grasp (except in the ring and little fingers). The near phalanges can still be flexed by the interossei, and owing to the unopposed action of these same muscles the mid and far phalanges are liable to be over-extended and curved backwards. It is the median nerve which confers what may be called the human functions on the thumb, and when it is paralysed the thumb cannot be abducted or opposed ; the first and second metacarpal bones lie in the same plane, the thumb looks directly forwards, and we get what Duchenne called the ' ape's hand.' In paralysis of the musculo-spiral we get loss of supination and extension, which is total in the wrist and thumb and partial in the fingers—i.e. the near phalanges cannot be extended, but the mid and far phalanges can still be extended by the interossei. The ulnar nerve is the nerve *par excellence* of delicate manipulation. It supplies all the intrinsic muscles of the hand,

except the two outer lumbricales, the opponens, abductor, and outer head of the short flexor of the thumb. All the ulnar muscles acting together give a conical shape to the hand, like that observed in tetany. When the ulnar is paralysed, sewing, writing, and all delicate manipulation become impossible. The patient cannot move the fingers to and fro in the same plane. The mid and far phalanges cannot be extended, nor the near phalanges flexed, and the patient cannot make a billiard bridge. After a time the hand, by the unopposed action of the flexors and extensors of the fingers, becomes 'clawed'— i.e. the near phalanges are extended, while the mid and far phalanges are flexed. I need not specify the well-known areas in the hand which cease to feel when either of these nerves is paralysed. I will merely say that motor function is always more easily abolished than sensory function ; that, in cases of recovery from damage, sensation invariably returns before motion ; and that occasionally sensation will return in a nerve area, notwithstanding the continued severance of the nerve-trunk. It must be remembered that anastomoses of the sensory branches of the three nerves of the hand exist on the back and palm and on the pulps and along the edges of the digits, and that MM. Arloing and Tripier have proved that the 'persistence of sensibility' in nerve-areas after division of the nerve itself is due to the conduction of sensory impressions (by means of these anastomoses) through the trunks of nerves which remain entire.

There are some purely mechanical conditions in the hand which are liable to lead to misconceptions as to the power of certain muscles. The fingers and the wrist-joint each have their own flexor and extensor muscles, which act independently of each other ; and it is obvious that the state of the wrist-joint, whether flexed or extended, must influence the tautness or slackness of those tendons which, having no attachment to the wrist, pass over it to flex or extend the fingers. When the wrist is in an extreme degree of flexion or extension those tendons which run over the convex side of the joint are taut, while those which run over the concavity are slack ; and this tautness or slackness of the extensor or

flexor tendons of the fingers is, be it observed, quite independent of any action on the part of the extensor or flexor muscles of the fingers. The fingers can be extended with greater ease and completeness with the wrist flexed than with the wrist extended ; and, indeed, when the wrist is fully extended a complete extension of the fingers can scarcely be accomplished, and the attempt causes great fatigue-pain in the palm owing to the efforts of the interossei and lumbricales. The cause of this difficulty is clear enough. When the wrist is fully extended the tendons of the extensors of the fingers are slack, and this has to be overcome before extension of the near phalanges can begin. Hence part of the difficulty. At the same time the flexor tendons are taut, and tend, quite mechanically, to flex the mid and far phalanges. This is a second obstacle to complete extension of the fingers with the wrist extended. It is the interossei and lumbricales combined which extend the two end phalanges, and the lumbricales seem especially designed to overcome the obstacle offered to complete extension of the fingers by extension of the wrist. These muscles arise from the tendons of the flexor profundus, and are inserted with the interossei into the aponeurotic expansion on the back of the mid phalanges. When, therefore, they contract, they tend, by one and the same action, to slacken the far ends of the flexor tendons and to tighten the far ends of the extensor tendons. They thus permit extension by their action on the flexor tendons, and actively produce it by their action on the extensors. I am not aware that this view of the action of the lumbricales has been previously stated. The interossei and lumbricales have also the power of moving the fingers from side to side ; but it must be remembered that, as the tendons of the extensors radiate to a slight extent from the wrist-joint, the fingers tend to separate from each other during the full action of the extensor communis digitorum. This may be mistaken for true interosseal action.

We are now in a position to discuss the effects of diseases at various points of the nervous system upon the form and functions of the hand. Beginning at the highest point, we

will consider first the effect of diseases of the brain-cortex. There seems to be no doubt that a lesion of the upper limb convolutions in one half of the brain is capable of causing trouble in the upper limb of the opposite side, and what are called 'brachial monoplegias' have been reported tolerably often. They are most common in tubercular meningitis and syphilis, which, when it attacks the brain, often affects the cortex. According to Landouzy, who has studied the subject of cortical paralyses, these paralyses are characterised by the following peculiarities : 1. They are partial and limited to any extent—i.e. we may have paralysis of only one limb, or of part of one limb. 2. They are very often incomplete—i.e. weaknesses rather than palsies. (3) They are often transitory. 4. They are variable—i.e. the area of the paralysis tends to get smaller or larger.

Landouzy has collected over 100 cases from various sources which seem to support the theory that motor functions are localised in particular convolutions. Among them, however, are not many cases of arm palsy, and of these most were hemiplegic in the first instance.

In a case of congenital absence of the right hand recorded by Gowers, the right ascending parietal convolution at its middle third was found to be smaller than its fellow on the left side, measuring transversely only ·35 in. as against ·65 in.

In a case of congenital absence of the right hand which I lately had an opportunity of seeing, by the courtesy of my friend Mr. Barwell, in the Cripples' Home, Marylebone, there was a very noticeable inequality in the size of the two halves of the skull, that opposite to the absent hand being very much the smaller. A diagram made from measurements taken with a cytometer showed this clearly, but the difference was far more apparent to the eye than the diagram would lead one to suppose.

In another case of congenital absence of one hand, which I saw a few weeks since at Andover, the head appeared to be perfectly symmetrical, but I had no opportunity of measuring it.

Hahn has recorded the case of a young man of twenty who

had a weakness of the first three fingers of the right hand, for which he sought advice on August 11. He then became epileptic and died comatose on August 30, without any extension of the paralysis. Post mortem a part of the cortex of the left hemisphere 'towards its hinder third' was found hardened and of a yellowish colour over an extent of 2 in. by 1 in.

Cotard in 1868 recorded the case of an old woman in whom an old paralysis of the left arm was found to correspond post mortem with an atrophy of the convolutions at the upper end of the right fissure of Rolando.

Bourdon, Rosenthal, Ringrose Atkins, and others have put similar instances upon record, so that the fact of the connexion of certain convolutions with the upper limb may be regarded as clinically established.

I have only had one case in which I have had reason to suspect a cortical lesion, and in this the hand was mainly affected. A gentleman, aged thirty-eight, actively engaged in literary pursuits, was much occupied in looking over many hundred examination papers. In order to get through this work by a certain time he sat up at nights and goaded his brain with tea, coffee, and other stimulants. He was 'taken ill,' and his queer state attracting the attention of his friends, I was sent for about thirty-six hours after the attack. The right hand was distinctly paretic. The patient could move the right leg well enough, but he afterwards stated that when first attacked there was a difficulty in moving the leg. The right side of the face drooped perceptibly. Speech was unaffected. The patient was quietly delirious, but answered some questions fairly well, and knew me perfectly. He had pain on the left side of the head, and was possessed by a fixed delusion that an insect had taken possession of his left frontal sinus, and was trying to work its way into his nose. (He had studied anatomy.) The urine contained a trace of sugar. In a week or so he was practically well enough to go a long voyage alone. There is now, two and a half years after the attack, not a trace of paralysis about him except that his handwriting is not so firm as it was. I believe this

to have been a congestion of an overworked cortex, possibly accompanied by a minute hæmorrhage or thrombosis.

When, instead of the cortex, the internal capsule is affected, we are confronted with an ordinary case of hemiplegia, which need not be discussed. In ordinary cases the hand suffers more than the leg, and is the slowest to improve.

There are two results of hemiplegia which mainly affect the hand, and which call for some special attention : these are contraction and spasm.

Late contraction or contracture is an almost constant phenomenon after hemiplegia ; its completeness is usually proportionate to the completeness with which the limb is cut off from the will, and it is generally ascribed to a degenerative process descending along the path of the will. This degeneration is best marked after lesions of the internal capsule, and is said by Isidore Straus not to occur after lesions of the cortex ; others, however, hold a different opinion. This degeneration follows the course of all nerve degenerations, and travels slowly down the course of the extinct function. Its descent is completed in about two months, and then the contraction of the hand approaches its maximum. Ordinarily (in two-thirds of the cases) the contracted limb is in an attitude of flexion and pronation. As far as the hand is concerned, the attitude is an exaggeration of the cadaveric position—the fingers and thumbs being flexed upon the palm. It is probably due to the extinction of the will and the abandonment of the limb to reflex stimuli, reaching it through the cord, and the consequent tonic contraction of the stronger group of muscles. As a rule the muscles do not waste and respond to faradism, as in health. The contracted flexors can be made to contract still more by electric stimulation, showing that their contraction is not complete. The cadaveric position of the hand is similarly due, probably, to the tonic contraction of the stronger group of muscles in the interval between death and the dying out of muscular irritability. The 'cadaveric position' is assumed, even though the force of gravity may tend to counteract it. I cannot believe that the late contraction of a hemiplegic arm is due to irritation, inflammation, or any

form of discharge emanating from the brain. I have said that there is no wasting or degeneration of the muscles of a hemiplegic limb. The reason of this is, I believe, that sensory impressions made upon the palsied limb produce their effect upon the muscles and vessels of the limb through the spinal cord ; and that this exercise of spinal function, independent of the will, serves to maintain the healthy nutrition of the limb. The case is very different, as we shall see, when a limb is cut off from both brain function and spinal function.

An apparent exception to this rule, that hemiplegic limbs do not waste, is met with in brain paralyses occurring in early life before development is complete. The wasting in these cases is apparent only, and is due to the fact that the sound limb, which is exercised, grows faster than the palsied limb, and, as it were, leaves its fellow behind in the developmental race. The practical lesson to be learnt from this fact is that if we would prevent that distortion of the body which must result from an unequal growth and an unequal use of the two sides, the palsied limbs of children must be stimulated and exercised artificially and methodically until growth has finished. It is well to bear in mind that the amount of palsy or contraction in a hemiplegic limb may vary in degree from a maximum which those who run may read, to a minimum which leaves only a trifling degree of contraction of the flexor muscles, or a little unsteadiness in the performance of delicate manipulative acts. I have seen some eight or nine cases in which a functional trouble, such as a writing difficulty, was distinctly due to what I may be pardoned for speaking of as the dregs of a long antecedent, and perhaps forgotten, hemiplegia. Hemiplegia is not very uncommon in early life, and one must ever be watchful in after life for the trifling effects of it.

Another, though rarer, result of brain lesion is spasm. I shall deal exclusively with chronic spasms occurring some time after the paralysing lesion, such as have been spoken of as post-hemiplegic spasm, spastic contraction and athetosis. These three forms dovetail into each other, and I think it will be profitable to consider them together. These spasms vary immensely in extent. 1. They may affect all four limbs and

the face, such cases being common enough in idiot asylums.
Dr. Clay Shaw drew attention to them in 1873, and quite
recently, through the kindness of Dr. Langdon Down and Dr.
Grabham, I have been able to inspect several such cases at
Normansfield and Earlswood.   The movements are almost
continuous, varying in form, in the same case, within certain
limits ; and they are increased by attempts at voluntary acts.
They are congenital or begin very early in life.   The intelli-
gence is usually not much impaired, and in idiot asylums these
patients rank high in the scale of intelligence.   This general
spasm is common, it is said, in firstborn children ; and very
often some accident causing pressure and damage to the cortex
cerebri has befallen them during birth, so that they have been
thought to be stillborn and have been recovered by artificial re-
spiration, &c.   Several of these children at Normansfield had
an exaggerated knee-jerk when the patellar tendon was struck.
In some cases there is contraction of the limbs as well as spasm.
2. In other cases this form of spasm follows an attack of hemi-
plegia.   It is then limited to the paralysed limbs, and the arm
is usually worse than the leg.   Gowers has recorded one case
in which there was late rigidity of the arm and spasm of the
leg.   3. In cases of paraplegia from a limited lesion in the
cord, with consequent degeneration in the lateral columns,
there is liable to supervene a rigid state of the legs, and if
voluntary power return to any extent there is a spastic spasm
on attempting to move.   It is in these cases that we get the
most marked exaggerated knee-jerk and ankle-clonus.

Now, it seems probable that the mechanism of all these
varieties of spasm is similar.   In all the forms there is some
amount of voluntary power, and in all there is some severance
of the muscles from the will.   In all, probably, there is more
or less descending degeneration along the path of the will, and
of this the existence of exaggerated knee-jerk is probably an
evidence.   Ankle-clonus and knee-jerk are probably due to a
heightening of the spinal function (reflex or not), and it is
certain that phenomena of this class are most easily observed
in the legs whose reflex functions are more highly developed
than those of the arms. In these conditions of the cord, muscles

are in such a state that when stretched they contract visibly. This is certainly only an exaggerated normal condition. Sir Charles Bell in his classical treatise on the hand pointed out that every voluntary movement implied, not only an active contraction of the muscles which produce the movement, but also a regulated contraction of the antagonist muscles, in order to check the movement and to prevent its being too jerky and sudden. Now, this check movement of a muscle is possibly provoked by the mere act of stretching, such as must necessarily occur in the extensors of a joint when the flexors are made to contract actively. These movements, provoked by stretching, if not true reflex movements, are in some way dependent upon the spinal cord, and in certain diseased conditions they apparently become so exaggerated as not merely to check the movements dictated by a weakened will-power, but even to cause movement in an opposite direction.

For the production of steady motion there must be a proper balance between the amount of muscular contraction caused by the will (the influence of which reaches the muscle, be it remembered, from both sides of the brain, by the crossed and the direct will-path) and the contraction caused by reflex influences and the mere stretching of the muscles. In the cases which I have been discussing one or other of the will-paths is certainly more or less blocked at its source or throughout its whole length; certain spinal functions are exaggerated, the balance of power is upset, and disorderly movement results.

It is interesting to note that these spasms occur most frequently in children, in whom all reflex phenomena are but little under control. Of the seven cases which I have seen six were congenital, or followed a hemiplegia occurring in childhood. In these children the functions of the spinal cord develop with the development of the child, while the brain damage hinders the development of the controlling influence, and thus we get the balance of power very greatly upset.

In one of my cases of spasm, which came on seven years after a hemiplegic attack, caused by a blow on the head at the age of twenty-six, the spasm which affected the hemi-

plegic (right) arm, and to a less extent the leg, was, in the arm, of a very violent character, quite unlike the so-called athetotic spasm, and was, moreover, fixed and invariable in its form. The wrist was bent, and the hand violently twisted to a position of extreme pronation. There was in this case, however, evidence of a paralysis of the lower branches of the musculo-spiral superadded, as it were, to the hemiplegia. There was a characteristic prominence on the back of the wrist, impaired sensibility in the radial area on the back of the hand, and the extensors of the wrist and fingers gave degenerative reactions. The paralysis of the extensors and supinators in this case was probably quite independent of the brain lesion, and I believe it served, if not to start the spasm, at all events to give it its determinate and fixed character. I have observed similar facts in at least two other cases.

Finally, I believe that the prime factor in all these forms of chronic spasm is the cutting off of the full influence of the will from the muscles, and their abandonment to other influences over which the patient has no control ; that they are due, not to any active discharge from the brain, but are brought about rather because the brain fails to discharge its normal amount of voluntary stimulus to the muscles. I need not say that I do not mean to apply these remarks to transient convulsions of an epileptic type.

In one class of idiots there is observed a peculiarity of the hand which is worthy of mention, and which is said to be diagnostic of their mental condition. I saw several of these cases at Earlswood and Normansfield. The skin of the hand is soft, wrinkled like a washerwoman's, and looking too big for the hand, and the finger-tips are tapering and conical. These idiots are often the youngest children of large families. They have a fair amount of intelligence, are singularly imitative, and have the eyes obliquely set, like a Chinaman. The head is round. They never acquire a handicraft, though they are often musical. There is a high arch to the palate, the tongue is fissured, and they usually die of tubercle before puberty.

Before leaving the subject of the influence of brain lesions

on the hand, I must mention a case of a child in whom was found a glioma as big as a small egg, growing from the left lobe of the cerebellum, and largely occupying the fourth ventricle. In this case, which was under my care at the Royal Hospital for Children in the autumn of 1878, there was observed the characteristic tetanic rigid conditions of the limbs occasionally seen in cases of cerebellar tumour in children. For the last month of life the child was continuously in a state of opisthotonos, with the legs extended and rigid, the left toes strongly flexed, and the right toes strongly extended ; no rigidity about the jaws. As to the upper limbs, the arms were powerfully rotated inwards so that the backs of the extended elbows looked outwards and a little forwards, while the hands with the fists clenched were so strongly pronated that the palms looked almost directly forwards.

In dealing with those spinal lesions which cause troubles in the upper limbs I shall try to avoid the repetition of well-understood facts.

The best understood spinal lesion is probably that which is characterised by 'inflammation' of the front horns with destruction of its motor cells. This disease, well known as infantile, spinal, or essential paralysis, or anterior poliomyelitis, begins with a sharp febrile attack. The area of the paralysis is at first extensive, but gradually gets smaller as the patient recovers, leaving one limb, or part of one limb only, paralysed, and finally, in typical cases, a small group of paralysed muscles is surrounded by a larger group of muscles, which are merely weak. The reason for this I have already tried to explain. The paralysed muscles being cut off from every physiological stimulus, whether direct or reflex (the motor cells being apparently the path for both forms of stimulation), rapidly waste and degenerate. Sensation is perfect or, more often, the skin is over-sensitive. There are no trophic lesions, but the affected limb is always cold, and very often blue and congested. The paralysed muscles, tested with electricity, give the degenerative reactions, and sometimes, in cases which are recovering, we may find muscles, lately paralysed but paralysed no longer, which also give the degenerative

reactions.   This curious fact has been noticed also in some cases of lead palsy.

In the following case this phenomenon was observed.

Walter B——, a schoolboy of sixteen, was seen by me on August 3, 1881.   Three months ago, after a game of play, he had been seized with a typical attack of this disease, and at first nearly every muscle in his body was paralysed, and he was in a state of great danger.   In a day or two he began to improve, and has steadily progressed.   At present the legs are weak, but he can walk without much difficulty. Both arms are still partially paralysed, and their condition is almost symmetrical, but the right is rather the worst.   On both sides he can move the shoulders, flex the elbows, and extend the wrist to some extent.   He can flex the left wrist and digits but not the right, and on both sides there is paralysis of the triceps and of the interossei.   The scapular muscles, biceps, and supinators on both sides respond to faradism, as do also the abductor and opponens of the left thumb.   The flexors and extensors of the wrists and digits, and the inter-ossei on both sides, as well as the right abductor and opponens, give degenerative reactions, although some of these muscles (the extensor of the wrist on both sides and the left extensors and flexors of the fingers) respond to the will and are no longer paralysed.

I should mention also that I have seen hopelessly paralysed muscles, which formerly gave degenerative reactions, begin, after months of patient treatment, to respond to faradism, but nevertheless continue to remain as hopelessly paralysed as before.   Thus in this disease, under certain exceptional circumstances, we may find non-paralysed muscles giving degenerative reactions ; while muscles hopelessly and incurably paralysed may respond normally to electricity.

The following case of complete paralysis of one arm is of exceptional interest from the point of view of diagnosis.

Mrs. L——, aged twenty-four, consulted me on August 12, 1880, by the advice of Dr. C. Daniel, of Epsom.   A fortnight previously the patient, who was pregnant, being heated by exercise, had sat with her back to an open window and had

fallen asleep. Two days later she was seized with pain on the left side of the neck and scalp. There was vomiting, and the temperature rose to 102° F. There was noticed some want of power in the left arm ; and after the pain subsided (in thirty-six hours) it was found to be completely paralysed, sensation remaining perfect. When first seen every movement of the arm caused pain, and there were pain and tenderness, but no swelling, up the left side of the neck. The left arm was absolutely powerless. The deltoid and pectoral muscles, and every muscle of the upper limb supplied by the brachial plexus, with the exception of the rhomboids, were paralysed, and gave degenerative reactions ; sensation was perfect. There was no trophic change or change of colour, and there was no tendency for the arm to become cold. The pain soon left her. In December she was confined with a healthy child. The general health has steadily improved, and the patient has gained weight, but in spite of a daily, methodical, and most efficient galvanisation of the muscles, which I have myself seen performed upon several occasions, and the administration of mercury, strychnine, iodides, and arsenic, there has not been as yet, though a twelvemonth has elapsed since the attack, any tangible improvement in the power of the limb, which is still absolutely useless. The arm has not wasted as much as might have been expected, because of the constant galvanisation to which it has been subjected. There has been no difficulty in maintaining the temperature, no trophic change, and no tendency to contraction or deformity, for the very good reason that no muscles are capable of contracting. I have been in considerable doubt as to the diagnosis of this case. The early pain, the tenderness up the side of the neck, the absence of lividity, the ease with which the temperature has been maintained, and the fact that there has been no diminution of the area of the paralysis, have inclined me to think that this is not a case of anterior poliomyelitis, but possibly a compression of the anterior nerve-roots by a rheumatic thickening of the meninges. Two physicians equally eminent for their investigation of nervous diseases have seen this patient with me ; the first, in January, thought with me

that the motor cells were not affected ; the second, in July, with the knowledge that no improvement had taken place in eleven months, considered the case to be one of anterior poliomyelitis. I confess I am still in doubt. It is needless to say that the diagnosis of motor cell (?) or nerve-root (?) is of great importance for prognosis. The regeneration of a motor cell is inconceivable, while the power of regeneration of conducting fibres seems almost unlimited.

For the sake of comparison with these cases I will now bring forward a case of absolute paralysis of the right arm from a bruise of the brachial plexus.

In March, 1877, Miss F. W——, aged fifty, fell down a flight of steps, alighting on her right shoulder. Shortly after the accident the arm was devoid of motion and sensation from the shoulder downwards. When I saw her three months later the arm was much wasted, the deltoid, triceps, biceps, and all the other muscles of the limb gave degenerative reactions. The limb was absolutely useless, and sensation was entirely wanting below the middle of the forearm, and impaired elsewhere. On scratching the forearm it was noticed that vascular reaction occurred far more readily on the sensitive than the insensitive part. The galvanic current applied to the sensitive parts caused an immediate uniform redness, but on the insensitive parts it slowly produced an irregular patchy redness like a lichenous rash. The fingers were scurfy and bulbous at the tips, and the patient complained that they got hot at night. Galvanisation was efficiently and methodically applied to the arm by the patient's sister. By November sensation had returned down to the finger-tips (imperfectly). Wrist movable ; hand clawed and paralysed ; nails furrowed and white. During the winter the hand was covered with chilblains. In April, 1878, the arm was well, but the intrinsic muscles of the hand remained paralysed : the nails were a dead white. In July, 1878, the motion and sensation of the hand were still impaired, and the muscles of the hand gave degenerative reactions. In June, 1879, the intrinsic muscles of the hand responded to faradism. There were glazy spots at the roots of the finger-nails ; nails harsh, hard,

and grooved. In June, 1880, the hand was fairly useful though still liable to chilblains, and the sensation still imperfect. I have a water-colour drawing of the hand at this time. In a letter dated August, 1881, this patient writes, 'It is really quite a natural colour. Nails of thumb, first, and third fingers well; and the others much better. Performance on the piano quite grand. I can reach an octave, but cannot strike it without holding the thumb. The thumb and first fingers are the worst parts, as the thumb will not go out quite far enough, and the first finger is inclined to bend back. The chilblains last winter were nothing to what they were before.'

This case is one of surpassing interest, and is most instructive from the point of view of nerve pathology. First, as to the time of duration of the symptoms. The accident happened in March, 1877, and in August, 1881, her recovery, though still progressing, is yet not complete, so that it has already been protracted over a period of nearly *four years and a half*. During the whole of this time there has been a steady, slow, evenly progressing recovery, so that her improvement was recognisable from month to month. Recovery has taken place in a regular order : sensation returned first, and always preceded the return of motion ; and muscles situated nearest to the trunk recovered before those which were more distant from it. At present two muscles of the hand, the abductor of the thumb and the first dorsal interosseus, alone remain paralysed. From what is known of nervous degeneration and regeneration one could hardly have expected the course of events to have been otherwise. The injury in this case was severe. The nerve-trunks were probably bruised and torn by the head of the humerus, so that immediately after the accident they were completely 'blocked' to upward sensory impressions or downward motor stimuli. A disused nerve degenerates along the line of function, and the motor nerves in this case rapidly degenerated from the point of blocking down to their terminations, for no stimulation could possibly reach them either directly from the brain or be reflected to them through the spinal

cord. Accordingly, within a month or so after her accident, it is probable that each motor nerve had been converted into a degenerated cord some three-quarters of a yard long, as incapable of conducting stimuli as the skin, fat, or connective tissue. Now, as the degeneration of a nerve takes place from trunk to end, so its regeneration takes place in the same direction, for there can be no regeneration unless physiological or artificial stimuli reach the point to be regenerated. Motor stimuli, both direct and reflex, impinging against the block, gradually induce molecular regeneration and the power of conducting stimuli, and thus little by little, as the stimuli are able to travel further, we get a complete regeneration of the nerves (a process which, in this patient, has already taken four and a half years). It is quite inconceivable that the stimulus which seems necessary for the regeneration of a nerve should be able to pass by any unregenerated portion. One might as well expect the makers of a well to begin their work in the middle. Just as a well has to be tediously bored, so is a degenerated motor nerve slowly opened for the traffic of stimuli from above. Provided stimuli can reach the nerve the power of regeneration seems almost unlimited, and, indeed, when pieces are cut out of a nerve-trunk the severed ends seem capable of worming their way, as it were, towards each other, and eventually effecting a junction in spite of every adverse circumstance. It seems probable that the bulbous ends in amputation stumps must be looked upon as an overgrowth due to the arrival of stimuli in the stump of the nerve, which are unable to produce their proper physiological effect. Bulbous nerve ends, according to Weir Mitchell, are physiological rather than pathological, and are almost invariably present in stumps.

The earlier return of sensation in all cases of nerve injury is due to the following facts: 1. That the sensory branches from the periphery to the obstruction are not deprived of their natural stimuli, and presumably do not entirely degenerate, and are consequently ready to resume their full functions directly the obstruction on the up-path is removed.

2. That in the parts above the block the incidence of impressions of various kinds probably serves to keep up in some degree the healthy condition of the nerve ; it is, in short, next to impossible to deprive a sensory nerve of its natural stimuli.

3. That, owing to the anastomoses which exist between sensory nerve branches, impressions have the power to some extent of choosing the path of least resistance. Hence, happily, it results that sensory impressions, so important for the nutrition of the limb, are able to produce their physiological effects in spite of very serious injuries.

A few words as to the trophic troubles observed in this patient. These were of three kinds : 1. Muscle-wasting. 2 A sluggishness or absence of vascular reaction when the skin was stimulated. 3. Scurfiness of the skin, loss of nails, and a tendency to chilblains.

Why does it happen that muscles paralysed from a brain lesion scarcely waste at all, while those paralysed from destruction of the motor cells of the cord or from injury to a nerve-trunk rapidly waste and degenerate ? Because in the first case physiological stimuli still reach it through the cord, while in the second case it is completely cut off from every source of physiological stimulation. So long as a muscle be stimulated it will maintain its size, no matter whether the stimulus come to it from the brain along the will-path, or from the surface or deep parts along the path of reflected impressions, or be artificially applied to it by means of a galvanic battery. If, on the other hand, a muscle be cut off from all sources of stimulation, it will waste. I do not, of course, mean to say that there may not be other causes for muscle-wasting, but I have never encountered an exception to the rule I have enunciated.

With regard to the trophic changes I speak with less confidence, but I must express my absolute disbelief in the existence of special trophic nerves ; and we clearly ought to exhaust every possible explanation before we proceed to do what is far too common in nerve pathology, viz. to give 'to airy nothings a local habitation and a name.' I believe that tissue changes other than muscle wasting are often due to

the cutting off from the vessels which supply those tissues those physiological stimuli which produce the contraction and dilatation of the vessels, and thus exercise a local control over them. Every paralysed limb is deprived of one important aid to circulation, viz. the muscular contraction, which is a material aid to the circulation, especially in driving the blood towards the heart. Every paralysed limb must therefore be at a nutritive disadvantage, but yet in hemiplegic limbs whose connections with the spinal cord are normal no trophic changes usually occur, except a little congestion. When the motor cells of the cord are alone destroyed, no trophic changes occur. When a mixed nerve is destroyed, trophic changes are very liable to occur to some extent. When the posterior roots and sensory paths of the cord are damaged, trophic changes often occur, as witness the joint affections and occasional zona of locomotor ataxy. When a purely sensory nerve, such as the fifth, is damaged, trophic changes are common, but not invariable ; their occurrence, according to some, depending upon the implication or otherwise of the Gasserian ganglion.

Now, in the case we have been considering there were trophic changes. There was impaired vascular reaction on stimulating the insensitive parts of the limb, and it is tolerably certain that cutaneous impressions were not reflected to the vaso-motor nerves of the limb. The local circulation was therefore cut off from the stimulus of muscular motion and from the stimulus of cutaneous impressions. It must not be forgotten, however, that impressions made upon one limb seem capable of being reflected to the vessels of the other, so that impressions made upon the left arm and other parts of the body were probably able to reach the paralysed limb. This fact is very suggestive of the importance which it is in the animal economy that cutaneous impressions should reach the vaso-motor nerves. A sensory impression is felt, if I may be allowed the expression— (1) by the brain ; (2) by the muscles being reflected to them by the spinal cord ; (3) by the vessels being reflected to them by the cord or the ganglia on the posterior nerve-roots. It must be remembered that

conceivably any one of these paths for sensory impressions may be blocked, while the other two may remain open ; that any two may conceivably be blocked, or that all three may be blocked. The question is very complicated and very difficult to study. The dependence of nutrition upon a due connection between the sensory and vaso-motor nerves combined with muscular motion seems to me extremely likely. Its absolute proof or disproof seems almost impossible.[1]

CHAPTER II

PROFESSIONAL NEUROSES [1]

BY 'professional neuroses' I mean those troubles which result from the constant repetition of the same act, the act generally being that which enters largely into the professional work of the patient, and which too often constitutes his means of livelihood. The most common of these professional neuroses is 'writer's cramp'; but I need not remind my hearers this evening that there are many analogous troubles, such as piano cramp, telegraphist's cramp, sawyer's cramp, tailor's cramp, milker's cramp, hammerman's palsy, dancer's cramp, and so forth.

I shall limit my remarks almost entirely to 'writer's cramp,' so called because it is infinitely the most common of all, and because, in the stress of the nineteenth century competition, it bids fair to become still more common. I have chosen the subject for my paper this evening because I think I may fairly claim to have had exceptional experience. It is now twenty-two years ago since my first paper on the subject was published in the *Practitioner*, and in consequence of this first paper and two subsequent papers on the same subject communicated to the Royal Medical and Chirurgical Society I have enjoyed, thanks to the kindness of my professional friends, exceptional opportunities of studying these interesting and troublesome cases. I am sure I have seen and closely examined and studied at least three hundred of these cases, and this must be my excuse for bringing the matter to your notice this evening.

One result of my study of the subject is a doubt whether there is a distinct morbid entity which merits the name of

[1] A Paper read before the Thames Valley Branch of the British Medical Association, 1894.

writer's cramp.   Writing difficulty is common enough, but it
depends on a great variety of causes, and my first duty to-
night will be to answer this question : Given a case of writer's
cramp, how are we to set about diagnosing its cause ?   Let us
first consider the act of writing, an act practised in this
country exclusively with the right hand, and which we learn
to perform fluently only after *many years* of patient labour.
We are none of us born writers, and the children of educated
people do not learn the art with materially greater ease than the
children of the uneducated.   There is no evidence whatever, I
think, that by constant practice of the art of writing we gene-
rate a faculty which is transmissible to succeeding generations.

In writing, the pen has to be held with very great steadi-
ness, and there are distinctly two acts involved in writing, viz.
pen-prehension and *pen-movement*.   The pen is kept steady
(when the art is perfected) mainly by the intrinsic muscles of
the hand, the interossei and the muscle of the ball of the thumb
being chiefly employed.   In order that these muscles may
get a firm hold of the pen the carpal and metacarpal bones
must be held steady, and the wrist-joint must also be
fixed.   This throws work on the muscles of the forearm.
The elbow must be steady, and this throws work on
the muscles of the arm.   The shoulder and scapula must
also be steady, and therefore the shoulder and thoracic
muscles are brought into play.   The trunk has to be kept
firm, which involves contraction on the part of the spinal
muscles, and in order that the pelvis may give a firm support
to the spine the legs have to be firmly fixed.   There is scarcely
a muscle in the body that is not brought into play in the act
of writing, and if we watch a little child at its writing lesson
we generally see that it hitches one leg round the leg of the
chair in order to steady the pelvis and trunk, and so great is
the muscular effect involved that the face muscles contract
consentaneously, and the movements of the pen are, as often
as not, followed by the tongue.   We must always bear the
above facts in mind, and in examining a patient who has a
difficulty in writing we must remember that the seat of trouble
will often be found far away from the hand.   In examining

these cases the first thing necessary is to strip the patient to the waist. Having your patient stripped, let him stand before you, and look first at the shoulders and see whether any muscles are wasted or whether any of them manifest any tremor, fibrillary or otherwise. Turn him round and look at the position of the scapulæ, and whether their relation to the spinal column is normally symmetrical. In my experience I have seen some few cases of writing difficulty which depended on paralysis of the serratus magnus or some other scapular muscle, which, by failing to keep the scapula steady, caused an unsteadiness of the hand. Next examine the arms, forearms, and hands to see if there be any wasting of muscles, particular attention being paid to the interossei and the muscles of the thenar eminence.

Next ask the patient to hold his hands above his head, when any failure of the scapular muscles will be accentuated. Then ask him to hold the arms in front of him, and examine the hands very closely for tremor.

Next examine the joints to ascertain whether there is any stiffness and whether there is any thickening round them, or creaking on movement, or any nodular swellings of the fingers indicative of gout or rheumatoid arthritis.

Further, examine the tendons for teno-synovitis. I have seen several cases of writing difficulty dependent on rheumatism of the joints of the arm or hand, and one case in a distinguished member of our own profession, dependent upon gouty deposits in the tendons of the flexor longus pollicis.

Next ask the patient to write. In extreme cases there may be almost a complete inability to write a single legible word. Such cases are rare, however, and usually the chief detectable difference from health is the slowness of the act and the evident effort with which it is performed. Watch the pen, and you will generally see that it is grasped with almost all the patient's might, and as the letters are formed one can often see daylight through the nibs of the pen, showing how firmly the pen is pressed upon the paper. At other times we notice a good deal of shoulder movement, and these great efforts usually indicate that the pen-prehension is done by the

muscles of the forearm, and the movements of the pen are mainly from the shoulder. This is an indication that the true normal muscles of pen-prehension are not performing their quota of work.

Sometimes the grasp of the pen suggests feebleness and impotence, and, although the poise and movements of the hand are in these troubles almost infinite in variety, it is very common to see the forefinger move away from the penholder, and, becoming flexed, glide upwards. This almost certainly shows that the first dorsal interosseous muscle, the abductor indicis, one of the most important of the muscles of pen-prehension, is not performing its work properly.

The next step in the examination is one of great importance, viz. the search for any distinct tenderness of the nerve-trunks in the arms or hands. In order to detect this, equal pressure should be exercised, simultaneously, on the nerves of both arms, and it is by the comparison of the two and the flinching or expressions of pain on the part of the patient which enable us to detect it. I am accustomed to ask the patient to stand in front of me and rest his two hands, palms upwards, on my shoulders. Then, grasping his arms, I press upon the median nerves, the musculo-spirals, and the ulnars, and it is exceedingly common to find very well marked tenderness in one or other of the nerve-trunks of the affected limb. This is probably to be taken as an indication of slight neuritis, and I have no hesitation in saying that of all peripheral causes of writer's cramp slight neuritis is the most common and the most important. Occasionally some nutrition change, such as glossy skin, congestion, furrowed nails, or herpetic eruptions, affords additional evidence of neuritis.

Having found neuritis, we must look for the cause of it. These are strain, pressure, rheumatism, gout, and diabetes. The urine must be examined for signs of gout or diabetes. Examine the chest with care for evidence of aneurism, or other mediastinal tumour, and I would emphasise this advice by saying that in one patient sent to me for writer's cramp I discovered the trouble to be due to the pressure of an aortic aneurism.

Next, hunt for evidence of hemiplegia. A man may have had an attack of hemiplegia so slight as to have escaped his memory. His power may have practically been perfectly restored, the only evidence of want of power being a slight feebleness in the grasp of his pen and slight shakiness of the handwriting. On careful examination of these patients I have detected the slightest possible deviation of the tongue, the slightest want of symmetry in the face, the slightest want of power in the right hand, and the slightest tendency for the toe of the right boot to wear out before the left. These trifles all point in the same direction, and give a clue to the cause. In this connection the examination of the heart becomes important, as the detection of a murmur tells us of a possible cause of embolism. I would remind you that hemiplegia may occur at birth or before birth as the result of injury of the head, and is not by any means uncommon in infancy as the result of fits. The hemiplegia of children often only becomes manifest when they begin to use their limbs and specialise their movements.

Congenital left-handedness is a not unfrequent cause of writing difficulty, and these patients will tell you that writing has always been to them a labour and a sorrow, and you will learn that, whereas they perform almost all other delicate acts with the left hand, they have been driven to use the right for penmanship by dint of the obstinacy of their writing masters. Left-handedness is not unfrequently due, I believe, to damage to the left hemisphere of the brain in early life or before birth, and in such cases I have occasionally found obvious flattening of the left side of the skull and a want of development of the right half of the body. The left clavicle is longer than the right, and the size of gloves and boots is greater for the left limbs than the right.

There are various chronic troubles of the brain and spinal cord which lead to writing difficulty, and I have found it as an early indication of general paralysis of the insane, loco-motor ataxy, disseminated sclerosis, lead palsy, anterior poliomyelitis, and paralysis agitans. This short review will, I hope, impress upon my hearers how manifold are the causes

which lead to difficulty in writing, and how essential in such cases is a careful examination.

It will be found that the vast majority of cases of writing difficulty are due to one or other of the causes I have mentioned, but I admit there is a small residuum of cases which merit the name of true writer's cramp, in which no peripheral troubles and no evidence of coarse cerebral change is detectable. Some of these (mostly women) are pure neuroses and get well, but the remaining few afford grounds for a very gloomy prognosis *quâ* writing.

In the case of true writer's cramp (and in those due to neuritis) it will be found that one or other of the muscles of pen-prehension manifest a marked loss of irritability when tested by electricity, and it will be found that a current just sufficient to stir the muscles of the left hand has no effect when applied to those of the right. The loss of irritability is the same both for faradism and galvanism, and is probably due to some change in the muscle-tissue and the intramuscular nerves. In some of these cases I have thought that the muscle felt hard and rigid, as though some fibroid change had taken place. This, however, is pure speculation.

I have spoken of such cases as due to chronic fatigue of the overused muscles. There is always a change in the muscle, as evidenced by its inability to respond normally to electricity, but I readily admit that there may be concomitant changes in the whole motor-path from the grey matter of the cerebral cortex through the internal capsule, crus cerebri, spinal cord, and nerve-trunks and branches. It is generally asserted that there is no difficulty in cases of ' writer's cramp ' with any act other than writing. This is true only of a small number of cases, and it will often be found that in delicate acts such as manipulating a pin, winding up a watch, or wielding a salt-spoon there is some slight disability.

It will be at once evident that the question of treatment must depend upon diagnosis, and if the failure in writing power is found to depend upon any of the morbid conditions mentioned above the treatment appropriate for such morbid condition must be adopted.

The most treatable form of writing difficulty is that which is accompanied by tenderness of one or other of the nerve-trunks, which is caused apparently by a mild neuritis.

In order to cure the neuritis, the patient must cease for a time to write, and must be careful not to overstrain the arm by any excessive use of it, and especially he should avoid any exercise which involves a prolonged muscular strain, such as carrying a heavy handbag.

The tenderness of the nerves must be subdued by counter-irritation, and nothing is more useful than a good blister applied over the tender nerve-trunk at a point where it is superficial. In some cases a more chronic form of counter-irritation is useful, and I have found the application of a piece of capsicum plaister of undoubted service. For this purpose I always use the American capsicum plaister, which is very thin and pliable, is perforated with holes, and is excessively adhesive, having, I believe, a basis of indiarubber.

When the nerve-tenderness has been removed I think a thorough rubbing and massage of the whole limb, but especially of the intrinsic muscles of the hand, is very useful. I must give a word of warning as to the danger of practising massage while the nerve-tenderness persists, such a course, in my experience, being always harmful. This, I think, is the commonest mistake made in the treatment of writer's cramp, and therefore I repeat that there must be no massage until the patient has got rid of the nerve-tenderness.

In cases where there is no nerve-tenderness massage may be commenced at once. The application of the continuous electric current is of undoubted service, but I am not prepared to say that it is more efficacious than rubbing, which is a tolerably simple matter and easily applied.

If the patient be unable absolutely to abandon writing for a time, he should do the writing of sheer necessity with a pencil, or, better still, should use a typewriter. Here it may be necessary to add that only those typewriters which work with a keyboard are admissible ; those which involve the *continuous* grasp of a handle are scarcely, if at all, less dangerous than a pen. I must not forget to mention the not

uncommon association of alcoholism with writing difficulties. I have seen cases where the whole writing difficulty was caused by alcoholic tremor, and many more in which the difficulty had been intensified by the fatal practice of drinking spirits to ' steady the hand.' In both of these classes of cases abstention from alcohol has been sufficient to cure or greatly improve the condition of the patient. It is often very necessary to seriously warn the patient against yielding to the fatal practice of taking alcohol. I have not found any drugs of much use in these conditions. In cases which are presumably due to neuritis I have found benefit from the administration of small doses of liquor arsenicalis (ɱij or ɱiij) combined with one grain of iodide of potassium, and it is needless to say that if there be gout, glycosuria, or rheumatism present these conditions call for suitable treatment. The general health must be attended to, as any derangement, such as dyspepsia, constipation, or even a common cold seems to intensify the trouble. The arms should be kept warm. Flannel or merino vests *with long sleeves* should be worn, and in some cases I have ordered an extra sleeve for the affected arm.

One word as to prevention. The frequency of writer's cramp is due, in the first place, to the unwholesome life of a clerk who spends his days in a stuffy office in a crowded city. Secondly, it is due to the fact that the holding of a pen involves the *continuous contraction* of the muscles of pen-prehension, which get tired out, just as our arms get tired when we hold them rigidly extended, and as our legs get tired when we stand in one position for long periods.

The only remedy for this is to teach the child to write with both hands, which I believe might easily be done. The writing would have to be upright instead of slanting, and, with a pen in both hands, the left half of the line might be written with the left hand and the right half with the right. We educate the left hand far too little. Girls in this respect are better off than boys, for such exercises as piano-playing and knitting encourage a great amount of ambidexterity in

D

girls.   Why should not the clerk use either hand alternately, and so give to each its much-needed rest ?

What effect has the exercise of the periphery upon the development of the centre ?   By constantly using the left hand for written language might we not possibly educate our right Broca's convolution instead of letting it lie idle ?

I once gave utterance to these opinions and speculations at the bedside, and at their conclusion a student of more than ordinary intelligence asked me whether the extreme fluency of the female organs of speech might not be due to the fact that the right side of women's brains had been developed by a more constant use of the left hand than is common in males ?

In support of the statements and advice above given the author would quote briefly from the papers published by him in the 'Transactions of the Royal Medical and Chirurgical Society,' in 1878 and 1887, a few cases illustrative of the various classes into which cases of writing difficulty may be divided. A few of the 168 cases reported are in no sense cases of 'writer's cramp,' but it was the difficulty experienced in writing which caused them to seek medical advice.   The numbers refer to the table of cases given with the original papers.

The first group of cases is characterised by definite injury to one or more of the nerves supplying the arm.

1. G. E. (male), aged 65, died in University College Hospital in 1877 from the bursting of a large axillo—subclavian aneurism.   The first symptom of this trouble was a difficulty in writing produced by the pressure of the aneurismal tumour on the nerves of the brachial plexus.

2. A banker, aged 50, who suffered from syphilitic neuritis of the musculo-spiral nerve, leading ultimately to paralysis of the extensors of the wrist and fingers.   One of his first troubles was with the act of writing.

3. A male, aged 34, in whom the median nerve above the wrist had been completely divided by a pane of glass. He could only write when the pen was held between the thumb and ring finger, in which sensation remained, and he was obliged to watch the movements of the pen intently.   The median area

of the hand was anæsthetic and dead white in colour, and he had lost his finger nails on the fore- and mid-finger.

4. A Government clerk, aged 23, who had neuritis of the ulnar nerve at the olecranon, leading to loss of motion and sensation in the ulnar area. His first symptom was difficulty of writing, and his recovery was complete.

6. The Rev. Mr. R—— consulted me in June 1875 for impaired writing power. He was a public preacher and lecturer, and had suffered from nervous exhaustion. Three months previously he had been disturbed at night by a pricking sensation in the hand, and the next morning he wrote with great difficulty. At the time of his visit there was no loss of sensation in the hand, and the muscles of the little finger were acting healthily. There was wasting and paralysis of the interossei and adductor pollicis, and there was characteristic clawing of the fingers. A diagnosis of Cruveilhier's atrophy had been previously made, but the fact that the atrophied muscles all gave the so-called degenerative reactions when tested with electricity made it more probable that definite nerve lesion was the cause of the trouble. This supposition was confirmed by the discovery of a tender point in the palm near the base of the fifth metacarpal bone, and the eliciting of a history of a bruise at this spot sustained while moving furniture. The patient completely recovered, but the act of writing is still an effort. He has since suffered from sciatica with consequent wasting of the calf.

6A (not in the original paper). Mrs. S——, aged 35, October 1885. Paralysis of the deep branch of the ulnar nerve, causing wasting of interossei, adductor pollicis, and the flexor brevis pollicis and two outer lumbricales. Sensory branch is not affected. There is a very tender spot just over the origin of the deep branch, and a history of damage from moving heavy furniture.

6B (not in the original paper). A clergyman, aged 52, who had paralysis of the deep branch of the ulnar nerve, due apparently to a bruise. He had an exquisitely tender point in the palm just below the pisiform bone. There was complete paralysis of the interossei, but the muscles of the little finger

escaped, and there was no sensory paralysis. The case had been diagnosed as ' creeping palsy,' but he made a good recovery.

This and the two preceding cases point to the importance of examining the fine nerve-twigs in the palm, and to remembering that the deep branch of the ulnar nerve in the palm is purely motor, and has not sensory functions.

The next group of cases is characterised by symptoms which are attributable to cerebral lesions.

Two (Cases 7 and 8) of these were instances of ordinary post-hemiplegic movement. In one case (7), a married lady, aged 23, the symptoms had developed subsequent to a hemiplegia which followed diphtheria when she was seven. In No. 8 (male, aged 36) there was a history of a blow on the head, followed by prolonged insensibility (many days). The uncontrolled movements developed when he was engaged in almost constant writing, and have continued now for thirty years in a most extreme form. At first the right arm only was affected, but latterly the unsteadiness has extended to the leg.

In Case 9, a little girl aged 10, the movements began after an attack of convulsions in early infancy. This patient had been taught to write with her left hand, and she produced backward (mirror) writing with absolute fluency.

Case 10 was that of a university student of high intelligence who had suffered from athetotic movements from his earliest babyhood. The act of writing demanded immense mental effort, and these efforts to write had apparently caused a considerable extension of the spasmodic movements of the right upper limb.

I think there can be no doubt that in those patients who are unable to write by reason of a cerebral (motor) lesion, the effort to perform an almost (to them) impossible act is fraught with danger, and is likely to increase their trouble. (See case of hammerman's cramp on p. 97.)

In four of these cases it was observed that certain muscles of the paralysed limbs were especially wasted and degenerated. Thus Cases 7 and 8 had marked wasting of the extensors of the wrist and fingers ; Case 9 had wasting of the tibialis

anticus and peronei, and Case 10 had wasting of some of the ulnar muscles of the left hand with impaired sensation over the area of distribution of the ulnar nerve in the left hand. In these four cases there appeared to be a paralysis within a paralysis, a peripheral lesion superadded to a cerebral lesion, and this fact appeared to determine the direction and form of the spasm, at least to some extent, the wasted muscles not being able to satisfactorily oppose their antagonists.

I have had one case (Case 11), and one only, of tumultuous spasm of the right arm in which there was no satisfactory evidence of any coarse cerebral lesion.

This case was very remarkable. It was the first severe case of writing difficulty which I ever had under my care. I am now inclined to think that it must have been due to some cerebral thrombosis in the cortical area connected with the hand, and that it is distinctly analogous to the case of hammerman's cramp recorded on page 97. Unlike the other cases of this class, it proved strangely amenable to treatment, and in that connection it is in my experience unique. It was reported in the 'Practitioner' for September 1872, and the following is an abstract :

'Case 11.—George Gair, aged 32, single. A small man, well made, rather muscular, and of very healthy appearance. No history whatever of any hereditary neurotic tendencies. Has always enjoyed most excellent health. He has always been steady, and, while able, was hard-working and industrious ; has never had syphilis. He is a well-educated and very intelligent man. He left school when fourteen years old and became a writing clerk in a commercial house in the City. He wrote an excellent hand, and gained rapid promotion in his office. In the year 1859 he left his situation and entered an accountant's office, where he did, on an average, nine hours' writing a day. By reason of his neat style of writing he was constantly employed in copying balance-sheets. In this situation, in which he remained for fifteen months, he earned five pounds a week, and there can be no doubt that the amount of writing which he was called upon to do was something prodigious. He left his situation because of his present illness

which came on almost without warning of any kind. He had been working as well as ever, when one day, towards the close of his day's work, he says, " he had a difficulty in bringing his right hand down upon the paper." He managed, however, for that day and for the three following days to continue his writing by holding his right wrist firmly on the desk with his left hand. At the expiration of three days he found himself wholly incapable of accomplishing the neat work required of him, and he had to leave his employment.

'For about a month subsequently he managed to write a few letters by steadying the palm of his hand against the edge of the desk and only using his fingers. Between that date (October 1862) and January 1872 he has been totally unable to accomplish any writing at all with his right hand. He gradually acquired the art of writing with his left hand, and what writing he has absolutely been required to do has been accomplished in this way. During last year, however, the left hand began to suffer from cramps, and ultimately became as stubborn as the right. His means of subsistence being taken from him, he was obliged to live upon his savings, which were considerable; but his store of money gradually diminished; and about the middle of last year he began to experience real want and to be filled with apprehension for the future.

' About this time, and probably as an effect of his straitened circumstances and anxiety of mind, his right hand, which hitherto had refused to write only, began to be affected with spasms at other times. He gradually had lost the power of using his knife at dinner, and occasionally he found himself unable to accomplish the most ordinary acts by reason of sudden spasms of the muscles of his right arm. Shortly before I saw him he had broken the jug belonging to his wash-hand basin, in consequence of a sudden spasm just as he was about to pour out some water.

' During the six weeks previous to his coming under my observation there had been a further exacerbation of his condition, and the right arm had become liable to sudden spasms even when not called upon to perform any act. It was the seat of

an exaggerated local chorea ; it was always jerking about, and at times would bounce out of the side pocket of his coat as he was walking in the streets. These strange antics naturally attracted attention, and immediately the patient saw that he was observed the spasms became doubly severe. In January of this year he came under my notice at the out-patient department of the Charing Cross Hospital, and the following account of his then condition is taken verbatim from my note-book : " On asking him to strip himself to the waist, he does so without difficulty. There is no evident impairment of the nutrition of the muscles of his right arm. The right arm hangs by his side and is subject to constant twitchings. The deltoid, the pectoralis major, the scapular muscles, as well as the biceps and triceps, are all affected ; but the last-named muscle is the worst offender, and in it the cramps are more constant and more severe than elsewhere. While I am talking to him the arm is forcibly extended, and the triceps is as tense and hard as a board. The fingers and wrist are often flexed, but never extended. The thumb and fingers do not seem liable to spasms individually. At times the spasms subside for a few moments, but any allusion to them seems to bring them back." [This apparently was due to nervousness, and was exactly analogous to the extra difficulties experienced by a stammerer when attention is called to his defective speech.] " On being requested to perform any act, the right arm 'jibs,' as it were, like a stubborn mule, and it is only by main force and by the greatest concentration of thought and determination that the most simple things can be accomplished. Everything is done with the arm extended. On being asked to unlock a box which lay on the table the arm was stiffly extended, and the patient, standing of course at arm's length from the box, managed with great difficulty to unlock it.

'" Of all acts, writing occasions the greatest amount of spasm. On asking him to write his name, he takes the pen in hand, and immediately he does so there is a violent cramp of the triceps ; the arm is forcibly extended, and with great difficulty he manages to write ' Geor ' in a manner scarcely legible, when the hand is twisted off the paper by a violent rotation of the

wrist, and his fingers lose the grasp of the pen. On asking him to continue writing, he is perfectly unable to do so, and every effort even to place his hand on the paper seems to be violently resisted by every muscle from the deltoid downwards.

' " The spasms of the arm never come on during sleep. He states, however, that latterly, owing to mental worry, he has slept very badly."

' The published cases of " writer's cramp " and the articles upon the subject in our text-books gave one very little hope of bringing about any improvement ; but as such cases are of a peculiarly interesting nature, I resolved to try every therapeutic agent which seemed in any way applicable. First, small doses of strychnine were given, but as the patient said very decidedly that the medicine made him worse they were soon discontinued.

' Mental irritation and distress and sleeplessness being marked features, bromide of potassium was given and with excellent results, for it procured good nights' rest, and seemed to diminish a little the spontaneous spasm of the arm. This seemed to be due to his paying less attention to his condition. His power of writing showed no improvement after the bromide alone ; but in consequence of the sleep and mental ease which it seems to give it has been continued in doses of fifteen grains three times a day throughout his whole course of treatment.

' From the very first *rest* was enjoined. In order to make quite certain that the arm could be used for nothing at all, I first attempted to put the whole of the arm in an immovable apparatus of plaster of Paris, but this did not succeed, for the spasms were so violent and constant that it was impossible to get the plaster to "set" properly, and very soon the rough ridges which formed in the bandage began to bruise the arm. The plaster of Paris was therefore removed within forty-eight hours of its application, and the patient was merely ordered to keep his arm in a sling. To this he adhered rigidly for two months or so, and then, the general spasm having much diminished, he was allowed gradually to use his arm more and more, and at the present time he is allowed to use it as much as he likes for every purpose except that of writing.

' I first saw the patient on January 20, and for the first

three weeks the above methods of treatment were alone employed.

'On February 8, in addition to the bromide of potassium and rest in a sling, I commenced the use of the continuous galvanic current. At this date he had improved somewhat in so far as his condition bothered him less, and he always slept soundly at nights. The general spasms of his arm were still as bad as ever, and his inability to write had not in the least abated. The improvement in his mental condition, which served as it were as a solid foundation for his further treatment, was due, in my opinion, entirely to the bromide of potassium, which he still continues to take.

'The galvanic current was used in a peculiar way, and, as a reference to the accompanying facsimiles of the patient's handwriting will show, with the best results.

'One of Weiss's continuous current batteries was employed, and twenty-three cells were used to begin with. The sponges, being well wetted with salt and water, were first placed on either side of the belly of the deltoid muscle (which at the time was the seat of almost continuous spasm). The spasm immediately subsided, and then the man was made to exercise his deltoid while I counted "one, two," "one, two," like a drill sergeant, every time he elevated or depressed his arm, the patient keeping time to the counting.

'Other muscles were then exercised in the same way—the pectoralis major, biceps, triceps, pronators, supinators, and the flexors and extensors of the wrist and fingers, care being taken not to overtire the muscles. Every possible kind of rhythmical exercise has been gone through. The pectorals have been exercised by practising "extension movements" and drawing back the shoulders, and the fingers and thumb have been also drilled by opposing the thumb to every finger in turn, and by making him run along the mantelpiece or the table with his fingers as if playing the piano. It is unnecessary to detail the way in which the galvanic current was employed for every muscle or group of muscles, but the samples quoted will serve to show the principle. The good effects of this plan of treatment were soon manifested. It was commenced

on February 8. On the 9th the patient stated that "his arm had been remarkably quiet since the electricity yesterday"; and on the 11th he said that he had already derived so much benefit that his arm was no longer a nuisance to him, but, on the contrary, he was able to use it for dressing himself; and on the 12th he buttoned his collar, which he had not done for months before. A reference to the lithographed facsimiles of his handwriting will show that this too began to improve in a most remarkable way. The galvanism and rhythmical exercise has been continued every day without intermission from February to the present time, and the patient still continues to make gradual and marked improvement. He can now accomplish everything, except writing, with perfect ease, and even the power of writing has improved in a very great degree. He has written me two or three letters, and has been able to embark in a small business. When he writes, he says, he feels like a schoolboy beginning to learn, and the act of writing is gradually becoming more easy to him. The great bar to his writing hitherto has been the triceps muscle, which, till recently, has taken on a spasmodic action whenever he has taken a pen in his hand; and all his writing has been hitherto accomplished with the arm forcibly extended. This condition of the triceps is being gradually overcome, and the eighth sample of handwriting was written "with the greatest ease, and with the arm bent."

'In addition to the galvanism he has had a daily hypodermic injection of morphia (gr. $\frac{1}{6}$). I do not think that this was of any decided benefit to him. It has been discontinued now for the past six weeks, and his improvement has been, I think, more rapid since. At the beginning of June the galvanism was discontinued for a fortnight while the battery was under repair, and it was gratifying to find that during this time he suffered no retrogression.

' I append the dates and a few remarks concerning some of the specimens of handwriting.

'1. Written on January 20, 1872, before the adoption of any treatment.

1.

Geo

2.

George

3.

George Gaur

4.

George Gair

5.

George Gair
Kennington park

6.

George Gair
77 Lorrimore Street
Kennington Park
London S.E.

April 19th 1872.

7. The flower garden just now begs us on the more for everything is blooming

8. George Your Commercial Road

9. The flower garden just now keeps us on the move for everything is blooming
July 31.st 1872

'2. Written on February 8, after first use of the galvanic current.

'3. Written on February 9. The word "Gair" was written while the sponges were held on the front and back of his forearm.

'4. February 14.

'5. Middle of March.

'6. April 19.

'7. July 6.

'8. July 25. Written with the arm bent and "with the greatest ease."

'9. July 31.'

In a large number of cases the unsteadiness in writing appeared to be the dregs, as it were,' of a long antecedent and perhaps forgotten hemiplegia, or else marked the commencement of degenerative changes in the cerebral cortex.

None of these cases were coarsely hemiplegic, and in all the evidence of previous cerebral paralysis had to be carefully searched for. An attack of hemiplegia, even though it be slight and occur no matter how early in life, always, I believe, leaves its mark. Patients are often said to 'recover' from attacks of right hemiplegia, and are able to follow their occupations without apparent difficulty. It is, however, in the performance of a delicate muscular act such as writing, which requires a maximum amount of steadiness and firmness, that the previous cerebral damage asserts itself. The writing is shaky or slowly executed, or executed with a certain sense of difficulty.

In elderly people who come complaining of writing with difficulty the evidence of slight hemiplegia must always be looked for. Persons also who state that 'they have always had a difficulty in writing' must be examined carefully for evidence of hemiplegia, a trouble which is by no means uncommon in early life, but which, I believe, is often not suspected, because the movements of an infant are too little specialised to enable defects to be noticed.

When I speak of 'hemiplegia,' a term which it is con-

venient to use, it must be understood that I include under it such a condition as brachial monoplegia from cortical lesion.

Among the cerebral cases are included three of congenital left-handedness, because I believe this defect to be often due to lesion or anomaly of the left cerebral hemisphere. Of these cases only one wrote with his left hand, which he did fluently, and gave a normal slope to his letters. He was left-handed for fine work (writing, playing flute, and violin) and right-handed for coarse work (hammer, knife, cricket). The left hand and foot were bigger than the right, and the left boot was the first to wear out. Both this patient and another had injured the left arm (dislocation of shoulder and fracture of arm) in early life, but this had not cured the left-handedness. In two cases the right clavicle was shorter than the left. In these cases of congenital left-handedness, it is, I think, to be regretted that the patients were not taught from the first to use their left hands for writing.

I have seen so many cases of writing difficulty in left-handed subjects that I now look for left-handedness as a matter of routine.

Case 11A (not in the original paper). Mr. E. J. H., a clerk, aged 43, who was rather fat, flabby, and tremulous, and probably took too much alcohol, consulted me in February 1896. I found that he was absolutely left-handed for cricket and left-footed for football. He even dealt cards with his left hand. I told him to give up alcohol and to practise left-handed penmanship. In a fortnight he had learnt to produce most beautiful writing (true left-handed caligraphy). In April he returned to his office and did all his work with the left hand, and said that instead of being the *worst* he had become the *best* writer in his office.

I last saw him in November 1896, and he was still working admirably and rightly regarded himself as the champion left-hand writer. The writing is certainly marvellously good.

A very large number of my patients have been found to be suffering from chronic degenerative changes of the spinal cord or brain, and I have often found that their 'writer's cramp' was merely an early symptom of *general paralysis,*

lateral sclerosis, posterior sclerosis, progressive muscular atrophy, disseminated sclerosis, or *paralysis agitans.*

Of the 168 cases which I reported to the Royal Medical and Chirurgical Society no less than 117 were not to be classified under any of the previous headings, and I accordingly grouped them together and called them the

NEURO-MUSCULAR GROUP.

The most important objective symptoms observed in this group were (in addition to writing difficulty)—

(*a*) Tenderness of one or other of the nerves of the arm.

(*b*) Changed faradic irritability of muscles.

(*c*) Tremor.

The presence of nerve-tenderness is frequently recorded.

As to the pathological significance of nerve-tenderness I shall have something to say hereafter.

In my first series of cases nerve-tenderness was not systematically looked for, but out of the 66 cases of the second series which fall into the neuro-muscular group, there were 13 in which nerve-tenderness existed apart from change of muscular irritability, 21 in which change of muscular irritability existed without nerve-tenderness, 32 in which these symptoms concurred.

The neuro-muscular group is thus divisible into three subgroups.

Sub-Group A.—*Nerve-tenderness without change in muscular irritability* (13 cases).

In 12 the right median nerve was tender.

In 1 both medians were tender, in 1 all the nerve-trunks were tender, in 1 the musculo-spiral and median were tender, and in 1 the nerve-tenderness was very doubtful.

Seven cases suffered from gout, rheumatism, dyspepsia, or alcoholism.

Four cases had tremor.

Three are noted as being crippled in acts other than writing.

The previous duration of the trouble in this group was

relatively not long.   In the 9 cases in which this fact is recorded these periods were eighteen months, thirteen months, twelve months, nine months, six months, 'two or three months,' 'some weeks,' 'a few weeks,' one month.

SUB-GROUP B.—*Change of muscular irritability without nerve-tenderness* (21 cases).

By change in muscular irritability is meant merely a change in the irritability to faradism, tested by a comparison of the effects of equal currents applied to corresponding muscles in either hand or arm.

The hands are placed symmetrically and at absolute rest (usually hanging over the back of a chair).   One rheophore is then fixed to the nape of the neck, while the other (a rheophore of small surface) is applied delicately to the muscles which it is wished to test.   Having determined the least strength of current which will cause the contraction of a muscle (and I usually begin with the first dorsal interosseous) on the left (and presumably healthy) side, the same current is then applied to the corresponding muscle on the right side, and any difference in the effect is noted.   When it is stated that the muscular irritability is depressed or heightened, it means that the muscle responds to the current less readily or more readily than the same muscle on the left side.

To detect the differences requires some care, but not more care than is required to detect the alterations of tone observed in the eye-muscles in cases of diplopia.

Each of these 21 cases had muscles manifesting a decrease of irritability.   In 3 there were muscles manifesting an increase of irritability alongside of others which showed a deficiency.

The first right dorsal interosseous (1st R. D. I.) was affected thirteen times.

The abductor and opponens pollicis eight times.

The second right dorsal interosseous six times.

The fourth right dorsal interosseous and flexor longus pollicis were affected twice, the third dorsal and fourth palmar interosseous each once, and the extensors of the thumb and small muscles of the little finger each once.

In 8 cases there was trouble (pain, enlargement, creaking) in one or other of the joints, and in a ninth there was a family history of rheumatic gout.

In 11 cases there was tremor.

Four are noted as being crippled in acts other than writing.

In 18 cases the previous duration was noted, and it will be observed that it is longer than in the previous group, in which no change in muscular irritability was detected. Thus we have recorded periods of fifteen years, thirteen years, 'many years,' 'some years,' 'years,' six years, six years, three years, three years, fourteen months, 'some months,' one year, 'a few months,' four months, two months, two months, one month.

SUB-GROUP C.—*Nerve-tenderness and change of muscular irritability combined* (32 cases).

Nerve-tenderness occurred in the median only in 28 cases, the ulnar only in 2 cases, and in the median and musculo-spiral combined in 2 cases.

As regards muscular irritability, decrease only occurred in 20 cases, increase only in 8, and a combination of the two conditions in 4.

As regards the muscles affected, we have :

The first right dorsal interosseous affected nineteen times.

Other interossei seven times.

Abductor and opponens pollicis seven times.

Extensors of thumb four times.

Flexor brevis pollicis three times.

Flexor longus pollicis twice.

All the muscles of the hand three times, and supinator longus, supinator brevis, serratus magnus, and pectoralis major each once.

In 2 cases there was a history of sprain of the wrist, and one had had his right hand smashed in a mangle twelve years previously.

Creaking of the right shoulder occurred in 3 cases, 9 are noted as being dyspeptic, gouty, or alcoholic.

Tremor was noted in 18 cases.

In 11 cases there was disability in acts other than writing.

The previous duration was for the most part considerable, in only three cases was it less than a year.

In the whole 66 cases of the neuro-muscular group the previous duration was less than a year in 11, or 16·6 per cent.

In sub-group A the previous duration was less than a year in 6, or 46 per cent.

In sub-group B the previous duration was less than a year in 5, or nearly 24 per cent., and

In sub-group C the previous duration was under a year in 3, or 9·4 per cent.

It is dangerous to form conclusions on averages of small numbers, but these figures point in the direction that some time is, as a rule, necessary for the production of change in muscular irritability.

In the whole series of cases in both papers we find that of the muscles whose irritability was found altered :

The interossei were affected seventy-nine times.

Abductor and opponens pollicis twenty-eight times.

Extensors of thumb twenty-three times.

Flexors of thumb thirty-two times.

Muscles of little finger nine times.

And other muscles only rarely and exceptionally.

In the second series of 93 cases nerve-tenderness (N. T.) was noted fifty times, and in the subjoined table its distribution in the groups and its association or otherwise with altered muscular irritability (A. M. I.) is shown.

	N.T. only.	A.M.I. only.	N.T. + A.M.I.	Neither N.T. nor A.M.I.
Paralytic (13 cases)	4	4	1	4
Degenerative (14 cases)	0	3	0	11
Neuro-muscular (66 cases)	13	21	32	0
Total cases (93)	17	28	33	15

Of the 50 cases in which nerve-tenderness was noted it is to be observed that in all but 8 it occurred in the *median nerve*. I wish to call particular attention to the fact that the *muscles* in which change of irritability has been most frequently noted (the interossei) are supplied by the *ulnar nerve*.

What is the pathological significance of altered muscular irritability ?

We have seen it occurring in every group, cerebral, degenerative, and neuro-muscular, so that it has no absolute value for localising the writing trouble in the brain, cord, nerve, or muscle.

It is probably due to some change in the muscle or intra-muscular nerves, and it seems probable also that such change may be primary, or secondary to a lesion in brain, cord, or nerve.

These cases are strongly confirmatory of the fact that de-pressed muscular irritability in cases of 'writer's cramp' is observed more often in the muscles of *pen-prehension* than in those of *pen-movement*, i.e. in muscles which are subjected to very prolonged contraction, and therefore very liable to get over-fatigued.

It is possible that fatigue in a muscle is produced with undue readiness when the voluntary motor stimulus is impeded at any point. The man with cerebral paresis is very conscious of muscular effort when he tries to write, and the same is true of patients in whom the conducting power of the nerves is interrupted by inflammation, congestion, or pressure.

The following case is very instructive on this point. It is that of a boy, aged twelve, in whom a congenital fracture of the clavicle had caused pressure on the brachial plexus. The case was brought before the Clinical Society by Mr. Barker. The boy had peculiar trouble in writing, so that the case had been spoken of as one of writer's cramp. In this case there was very slight but quite obvious wasting (or non-development) of the pectoralis major and the muscles of the right arm and hand. The electric irritability was, however, normal in all the muscles except the first right dorsal inter-osseous, in which the irritability was depressed. The patient was a lad of unusual intelligence, and had worked hard at his writing lessons, which caused him fatigue and pain. It would seem as though the pressure on the plexus had necessitated increased effort, and had predisposed to the fatigue of the

muscles of pen-prehension and the lessening of the electric irritability in at least one of them, and that probably the most important. The pressure on the nerves of the brachial plexus was relieved by operation, and some weeks later I saw the boy writing without difficulty, and I found on examination that the depressed irritability of the first right dorsal inter-osseous no longer existed. It must be remembered that not only had the pressure been removed, but the boy had had a prolonged period of rest.

Occasionally the irritability of one or more muscles has been found exalted. The reason of this is doubtful. Occasionally I have found a muscle *supplied by a tender nerve* manifesting exalted irritability, but this is by no means invariable. I feel sure that the practice of massage increases the irritability of muscles.

It must be remembered that change in the irritability is judged by no fixed standard, but only by comparison with the opposite limb, so that we are always liable to the fallacy that a relative change may not really mean an absolute change from the physiological normal. For example, when we find the right first dorsal interosseous more irritable than the left, this fact may really be due to an abnormal depression in the irritability of the left first dorsal interosseous.

It must be remembered also that a muscle which manifests excess of irritability is not necessarily more efficient than the corresponding muscle of the opposite limb. Some authors have spoken of a condition of ' irritable weakness,' a condition which is probably a fact. Not only have the muscles shown great differences in their readiness to respond to equal electric currents, but their mode of contraction has often been obviously different to the eye, the muscle which is presumably healthy contracting forcibly and moving its point of insertion vigorously, while the muscle which shows deficiency of irritability contracts in a hesitating, jerky, and unsatisfactory manner.

The feeling of contraction (under electric stimulation) is often very different on the two sides—at least, this is what patients say ; but the fact has not been systematically recorded

because of the great difficulty of making a satisfactory record of such a fact.

For the same reason the record of cutaneous sensibility has not been systematically made, although, to judge from expressions used by patients, change has frequently been present.

What is the pathological significance of nerve-tenderness ? It is recorded fifty times in the second series of ninety-three cases, viz. forty-five times in the neuro-muscular group, five times in the paralytic group, and not at all in the degenerative group. The conditions under which nerve-tenderness occur are very varied.

It is oftenest and best observed in neuralgia of the fifth nerve. Whether the neuralgia be definitely due to central change or peripheral irritation or constitutional cause, we often find every accessible branch of the nerve more or less tender on pressure, and the tenderness, marked during a paroxysm, is absent or scarcely noticeable in the intervals between the attacks.

In paralysis of the facial from exposure to cold we usually find the trunk of this purely motor nerve more or less tender at its point of emergence from the stylo-mastoid foramen.

In 'sciatica' every accessible branch of the nerve is often tender, and even when the sciatica is due to intra-pelvic irritation we find tenderness of the ultimate branches, notwithstanding that the source of trouble may be nearly a yard distant from some of the tender twigs.

I have noticed great nerve-tenderness in cases of lead palsy and in cases of muscular atrophy, but without any other evidence of neuritis.

In true neuritis of a mixed nerve we find not only nerve-tenderness, but changes in sensibility, neuralgia, causalgia, trophic change, muscle wasting, and often distinct evidence of swelling of the nerve involved.

In chronic rheumatic conditions and in gout the nerve-trunks are often tender, and this without any good evidence that they are irritated by thickenings or bony outgrowths.

Nerve-tenderness may certainly be produced by excessive muscular exertion. I have seen such a condition in laun-

dresses, who state that they have strained the arm from wringing clothes, and also in men who have made excessive muscular effort, and notably in one gentleman whose musculo-spiral nerve had become excessively tender from prolonged use of a heavy fishing-rod.

In some of my cases the nerve-tenderness was associated with dyspepsia and alcoholism.

Mr. Barker's case may be again alluded to. In this patient all the nerve-trunks of the arm were very tender before the removal of the pressure on the brachial plexus, but after the successful operation this tenderness quickly disappeared.

From this *résumé* it appears, therefore, that nerve-tenderness may, in regard to its cause, be

1. Functional (neuralgia).
2. Central.
3. Peripheral.
4. Due to cold.
5. Due to rheumatism.
6. Due to inflammation (neuritis).
7. Due to muscular exertion.
8. Toxic (lead, alcohol, gout).

In my cases of impaired writing power the median nerve has most often been found to be tender. Muscular failure is most often evident in the muscles of pen-prehension, and notably in the interossei. When these small muscles of pen-prehension fail, bigger muscles (such as the superficial and deep flexor of the fingers and the long flexor of the thumb) take their place, and I believe that the tenderness of the median nerve is dependent upon the over-use of these (median) muscles.

In the early days of these cases it is possible that one or more of the deep branches of the ulnar nerve may have been tender in association with over-use of ulnar muscles, but this, of course, is only a surmise.

No thickening of any nerve-trunk was detected in these cases.

Trophic lesions were not common. In Mr. Barker's case

the nails of the affected hand grew slowly.   No. 78 was troubled by coldness and blueness of the hand.   No. 102 had a congenital brown mark on the inner side of the right arm. No. 128 had pitting of the right finger nails.   No. 130 had a crop of moles on the right arm and a lichenous rash on the right forearm.   No. 144 had brittleness and furrowing of the right thumb nail, and No. 149 had throbbing of the thumb after writing.

'Gout,' 'rheumatism,' and creaking and thickening of joints were tolerably common.

Tremor was a very common symptom, and is recorded in 87 out of the total of 168 cases.   It is a symptom of varied significance, and appears to be liable to occur with defects at any part of the motor path.

In some few instances the tremor was of very limited extent.   No. 121 had tremor of the thumb only, accompanied by slight wasting of the abductor and opponens pollicis. No. 122 had tremor of the middle finger only.   No. 150 had tremor of the far-phalanx of the thumb only, and in this case there was deficient irritability of the extensors of the thumb as well as of the abductor and opponens.   No. 160 had tremor of the thumb from a similar cause.

Tremor of limited area may thus point to the fact that the muscles moving the joints about which the tremor occurs are especially affected.

Obvious spasm was rare.   No. 82 had slight athetotic movements, but, as a rule, it was hardly possible to say, from merely watching the act of writing, why the writing failed.

There were some exceptions to this rule, however, and these were instructive.

No. 76 suffered from extreme flexion of the far-phalanx of the thumb associated with paralysis of the extensor of that phalanx.

No. 117 suffered from a collapsing of the phalangeal joint of the thumb and rotation towards the index, associated with weakness of the extensor primi internodii and abductor and opponens.

No. 127 (like 76) suffered from flexion of the far-phalanx of the thumb associated with weakness of the extensor. Nos. 140 and 144 were very similar cases.

In Nos. 129, 131, 139, and 140 there was inability to keep the forefinger on the pen, associated with weakness of the first right dorsal interosseous.

No. 156 did not use his thumb while writing, and in this case there was weakness of the flexor brevis pollicis and tremor of the thumb.

In No. 163 the phalangeal joint of the thumb collapsed, and it was noticed that faradisation of the flexor longus pollicis caused tremor of the thumb.

When writing is made impossible by some decided and recurring spasmodic action we may expect to find either *deficient* irritability in the muscles which normally antagonise, or *excess* of irritability in the muscles which normally produce such movement.

More than a quarter of my total cases of impaired writing power have been due apparently to recognised morbid states of the brain and spinal cord, and it must be borne in mind that the act of writing—an act which requires at once so much delicacy and firmness—would certainly afford evidence of slight changes in the nerve-centres, of changes so slight as not to be likely to materially interfere with the performance of coarser and less sustained muscular exercises.

The majority of the cases seem to have been produced by what I have ventured to call ' chronic fatigue ' of the muscles.

Since *Fatigue* is by no means an uncommon cause of disease, it will be well in this place to make some general remarks on the subject.

Work results in fatigue, and fatigue is a regular and constantly returning symptom experienced by all of us. Periods of functional activity invariably alternate with periods of repose, during which the waste caused by the exercise of function is repaired. We are indebted to Sir James Paget for having pointed out in his Croonian Lecture, delivered some years since before the Royal Society, that

'rhythmic nutrition' is a law of nature ; but, although the truth of this dogma is recognised on all hands, and may be said to be axiomatic, it has hardly received that careful consideration at the hands of practical physicians which it deserves.

Our whole life consists of a series of vibrations—periods of tension alternating with periods of relaxation ; and, although the rapidity of these vibrations varies immensely, they are recognisable in all our acts, be they voluntary or involuntary. Let us look first to the 'vibrations' of an organ the movements of which are placed entirely beyond our own control—the heart. Now, the rate of vibration of the heart is 72 per minute, and if the total period of each vibration be divided into ten parts it will be found that four of these parts are devoted to the process known as *systole*, which may be looked upon as labour ; three are occupied by the *diastole*, which, although hardly labour, is nevertheless exercise of function ; and the remaining three parts are occupied by the *pause*, during which the heart apparently enjoys absolute rest from any exercise of function, and may be considered to be in a condition analogous to sleep. May we not apply the rate of action of this organ, which has been regulated for us, to our voluntary acts, and may not the heart be made to give the key-note, as it were, to many questions, personal and social ? If we divide the day of twenty-four hours into ten equal parts, and give four of these to active work, three to functional exercise of other kinds, and three to sleep, we shall find that nine and a half hours' work, seven and a quarter hours' 'relaxation,' and seven and a quarter hours' sleep is what a normal man may, and, as a rule, does, perform without injury to himself.

To continue with the consideration of our 'vital vibrations,' we may remark that it is universally ordained among civilised nations that once in every seven days there shall be a remission of labour and a change of occupation ; and we further recognise the fact that it is highly advisable for those who are occupied in monotonous pursuits to break away from them at least once a year and indulge in that variety of

work which we call amusement. Monotonous repetitions of the same act are acknowledged, on all hands, to be the most potent causes of fatigue.

Fatigue occurs directly we attempt to alter the rhythm of our vital vibrations by prolonging the periods of tension at the expense of the periods of relaxation, or by demanding for any length of time a quickening of the normal rate of vibration; and it is not unreasonable to suppose that every organ of the body has its 'normal rate of vibration,' if we could only determine what it is.

We recognise the fact that athletes who 'over-train' run risks of cardiac troubles and loss of wind ; that the man who from any cause is unable to sleep runs a serious risk of permanent impairment of health ; and when we find patients pursuing their avocations too zealously we know that, if such offence against the laws of nature be persisted in, general paralysis or other form of 'break-down' is likely to be the result.

Fatigue may be general or local, and both forms may be either acute or chronic.

That fatigue in all its forms is due to impaired nutrition there can be little doubt, and we shall find that the symptoms of chronic fatigue are often the prelude of definite and recognisable degenerative changes.

As to *general fatigue,* it is recognisable with care both in its acute and chronic forms. There is a disability for performing either mental or physical work, and this disability is noticed first in work requiring attention and sustained effort, and, lastly, in those acts which have become automatic. The symptoms of general fatigue are referable usually to the brain and nervous system.

As to *local fatigue,* this, again, may be acute or chronic, and the symptoms of it are referable usually to the muscles ; but we must always bear in mind that muscle and motor nerve are one and indivisible, and that recent experiments have given great probability to the idea that every muscle is connected with a certain definite spot in the brain. When, therefore, we speak of a sense of fatigue we must necessarily

be in doubt, notwithstanding the fact that the symptoms are referred to the muscle, whether brain, nerve, or muscle, one or all of them, be really at fault. The symptoms of acute local fatigue are (1) *loss of power* to a greater or less extent. By too frequent or too prolonged stimulation the irritability of muscular tissue becomes exhausted, and it either refuses to respond or responds but feebly to the stimulus of the will ; our power of adjusting the force of contraction to the act to be accomplished is lessened, and accuracy of movement and delicacy of co-ordination become impossible. (2) *Tremor* is a symptom of acute local fatigue which everyone who has been called upon for extraordinary muscular effort must have experienced. (3) *Cramp-like* contraction is the symptom of local fatigue which disturbs our rest after a hard day's walking or riding, or muscular efforts in the ball-room or elsewhere. (4) The *pain* of fatigue is familiar to most of us, and is readily distinguishable from other forms of pain.

Local fatigue is caused far sooner by prolonged and sustained muscular effort than by repetitions of short muscular efforts having due intervals of relaxation between them. Anyone who has attempted to hold out a weight at arm's-length knows the impossibility of a long continuance of the effort, and it is proverbially true that standing in one position is to most people far more tiring than walking, the reason apparently being that in standing the muscles which support the body are subjected to a prolonged strain, while in walking we use the muscles on either side of the body alternately. The great increase of working power which we obtain by this alternating use of the muscles would seem to be one of the chief reasons for the bilateral symmetry of the body. Not only is sustained effort a far more potent cause of fatigue than repeated effort, but we find that, when fatigue supervenes, actions requiring sustained effort, be they physical or mental, are the first to fail, while automatic actions endure the longest, and in this respect local fatigue resembles general fatigue. It is quite possible to exhaust a muscle by artificial stimulation, and if one of the small interossei muscles of the

hand be continuously faradised it will be found, as before stated, that in a short time its power of contracting under any form of stimulus will be absolutely abolished. It is tolerably certain that the brain can have no share in artificial fatigue thus produced, and there seems good reason to suppose that in some people of energetic temperament the irritability of a muscle may be exhausted while the power of mental stimulation remains unimpaired. Although the author dwells chiefly on the peripheral phenomena of **fatigue**, he readily admits that these must be generally accompanied by central changes. What these latter are must remain a matter of speculation, but in the peripheral phenomena we have something recognisable and certain, and deserving of our most careful attention.

Chronic local fatigue has causes and symptoms similar to those of acute local fatigue, and there can be little doubt that this condition is a common cause of many of those chronic maladies which seem to result from overwork, and which are characterised by irregular muscular action. That some cases of writer's cramp, torticollis, and hammer palsy are due to chronic fatigue of the muscles employed there can be little doubt. Duchenne and Mr. Brudenell Carter have pointed out how, in cases of short sight, the prolonged strain of the internal recti has caused troubles of vision and even cerebral symptoms ; and Taylor, of Nottingham, and Snell, of Sheffield, have shown reason for including in the category of ' fatigue diseases ' a peculiar form of nystagmus occurring amongst miners, who try their eyes by working in the dark, and the author has little doubt that, attention having been directed to the symptoms and effects of fatigue, additional light on the subject will be forthcoming.

We are now in a position to apply these theories to the act of writing. The act of writing is primarily divisible into (1) *the act of prehension*, and (2) the act of moving the pen ; and the act of movement may be again subdivided into (*a*) the stroke-making movement ; (*b*) the movement of the hand from left to right ; (*c*) from right to left ; and lastly (*d*) the ink-dipping movement. Besides the act of prehension, there

is (3) another muscular act : this is the poising of the fore-arm and hand, which is ordinarily kept about three-quarters prone, the hand being balanced upon the pisiform bone and little finger. Thus it will be seen that writing is divisible into three acts—the *prehension of the pen, the poising of the hand and forearm*, and the *movement of the pen*, and there is probably no muscle between the shoulder and the fingers which is not brought frequently into action during writing.

The muscular action to which we wish to direct very particular attention is that of prehension. The pen is normally held between the thumb and the first two fingers. The thumb and the index finger form an oval ring through which the penholder passes, being held by the distal and resting on the proximal end of the said oval. The distal ends of the metacarpal bones of the thumb and index fingers are widely separated ; the first phalanx of the thumb is abducted ; the phalangeal joint forms an angle which is more or less acute in different writers ; and the pulps of the terminal phalanges of the thumb and index finger are, but for the intervention of the pen, almost directly opposed to each other. With regard to the first two fingers, the proximal phalanges are flexed, and the two terminal phalanges nearly straight. The muscles which keep the thumb and fingers in this attitude of prehension are, we believe, with one exception, *intrinsic muscles of the hand*, in proof of which, if the rheophores of a faradising apparatus with big sponges be placed, the one on the palmar surface of the hand between the thumb and index fingers, and the other on the dorsal surface over the metacarpal bone of the index finger (so as to influence more or less the special muscles of the thumb and first two fingers), the thumb and first two fingers will assume an attitude of pen-prehension (saving only the flexing of the phalangeal joint of the thumb), and a pen held between them will be tightly grasped.

The muscles chiefly concerned in the muscular act are, we believe, as follows : The *interossei* of the first two fingers which flex their respective first phalanges (the dorsal muscles further helping the act of prehension by dragging the first two fingers towards the thumb) ; the *abductor pollicis*, which

abducts the first phalanx of the thumb, an action without which proper opposition of the pulps of the thumb and index finger would be impossible. The *opponens pollicis* and *flexor brevis pollicis*, as their names indicate, are also important muscles in the act of prehension. The phalangeal angle of the thumb is maintained, in a great measure, if not entirely, by the action of the *extensor primi internodii pollicis*.

The muscular effort of *poising the hand* is thrown chiefly on the supinators. The hand is three-quarters prone, and in this position the weight of the hand tends to make pronation complete—a tendency which is checked by the supinator longus, the supinator brevis, and possibly the extensors of the thumb.

The stroke-making movements are accomplished by the long flexor of the thumb, and the extensor secundi internodii, the flexor profundus digitorum, and the extensor communis digitorum. The up-strokes are in part dependent on an increased action of the interossei.

The movement of the arm from left to right depends chiefly on the triceps extensor, and that from right to left on the pectoralis. The muscles concerned in the ink-dipping movement scarcely require naming.

The five muscular acts above enumerated, which, taken together, produce the complicated act of writing, are divisible into two classes. *In the first class (consisting of the prehension of the pen and the poising of the hand) the muscles concerned are subjected to prolonged strain, and (especially those concerned in prehension) are kept in a continuous state of contraction often for inordinately long periods.* In the second class of muscular acts the contractions of the muscles alternate with periods of repose.

Now, as long as a muscle remains contracted it is in a state of exercise, a condition which is attended with impairment of composition such as can be repaired only during repose. The muscles by which the prehension of the pen is effected need not of necessity obtain any interval of rest for hours together. During all the several acts which constitute writing these muscles remain in a state of contraction, for

the pen, in stroke-making, in horizontal movement, and in ink-dipping, cannot be released from the grasp of the fingers and thumb.

These muscles, whose function has been thus abused, soon begin to respond but sluggishly, or refuse entirely to respond to the stimulus of the will, and the scrivener finds that his grasp of the pen is faltering and uncertain. He finds that he is obliged to take a tighter grasp of the pen and (unconsciously, of course, as far as he is concerned) he deposes the proper muscles of prehension and substitutes others to perform their office. He begins to grasp the pen, not with the intrinsic muscles of the thumb and first two fingers, but uses for that purpose the big flexors of the thumb and fingers situated in the forearm. The *flexor longus pollicis*, the *flexor sublimis digitorum*, and the *flexor profundus digitorum* are now the muscles of pen-prehension, and the stroke-making movements of the pen are accomplished by the flexors and extensors of the wrist, or the writer finds himself incapable of resting his wrist upon the desk because the movements of the pen have to be accomplished by the movement of the entire forearm by the muscles of the shoulder (the forearm being kept by means of its flexors at right angles to the arm). These new muscles of pen-prehension are kept in a condition of prolonged contraction just as were their predecessors, and in time become similarly sluggish, obstinate, and irritably weak, and the scrivener has to resort to new methods of pen-prehension, which generally consist of some mechanical contrivance, either entirely artificial or such as interlacing the pen amongst the fingers. The flexors of the elbow-joint (should they be used) soon strike work from a similar cause, and the triceps extensor is brought into use. The man writes with the pen mechanically fixed in the hand and the arm rigidly extended ; the stroke-making action being accomplished by the move-ment of the entire body.

Other muscles which are very prone to suffer in writer's cramp are those which poise the hand, and which, like those of pen-prehension, are subjected to prolonged strain. It is a common observation that the poising muscles suffer in writer's

cramp, and the hand is very liable to roll over in the direction of pronation (owing probably to the weakness of the supinators), or more rarely to be jerked in the direction of supination (as in a case recorded by Dr. Buzzard[1]) by the irritability of those same muscles. Many persons affected with writer's cramp become unable to poise the hand, and are obliged to write with the entire forearm supported and the hand in a state of complete pronation.

To illustrate our meaning, we have purposely supposed an extreme case. The disease not unfrequently does not spread beyond the fingers, but its rapid progression, as depicted above, is occasionally observed. When once the disease has commenced (showing itself perhaps merely as a trifling awkwardness in writing) its progress is liable to be very rapid if writing be persevered in. If, on the other hand, writing be abandoned on the first appearance of awkwardness, and the patient is able to give himself rest while his exhausted muscles of pen-prehension recover their tone, he rapidly regains his lost power.

Further, as to the act of writing, it must be borne in mind that it is one of the most complicated possible, perhaps the most complicated muscular act which is ever performed by the body. The act of writing takes years of patient labour to acquire, and although children begin to learn very early in life, it is seldom before adult age is reached that their writing loses those evident marks of juvenility which we all know how to recognise. Perfect writing should be an act accomplished without effort, and almost without thought, or, in other words, it should be a purely automatic act, and one accomplished by an expenditure of mental stimulus so small that we can scarcely recognise it. For the accomplishment of the act of writing a very large number of muscles is required, and when we consider the light yet firm grasp of the pen which is necessary, the poising of the hand in the semi-prone position, the stroke-making movements of the pen accomplished by the flexion and extension of the fingers, the travelling of the hand across the paper and back again, and the journey of the hand

[1] *Practitioner*, August, 1872.

to the ink-pot, we see that nearly every muscle between the shoulder and the finger-tips is brought into play, and we cease to wonder that years are required for educating these muscles to work accurately and harmoniously together.

There may or may not be a 'co-ordinating centre' whose function it is to control the act of writing; this is a matter of speculation. It is, however, tolerably certain that, should one or more of the muscles which have been so laboriously educated exceed or fail in its work by an increased or diminished response to stimulation, the harmony of the complicated act of writing is interfered with, concord is converted into discord more or less marked, and that which had become a purely automatic act by dint of years of study relapses again into an act which requires a greater or less amount of attention.

Now, directly an act which should be automatic begins to demand our attention for its execution the difficulties of executing such act are increased a hundredfold. Fear of failure, especially before others, is ever present to the mind, and it would seem as if a certain proportion of that mental stimulus which ought to animate the muscles suffered what we have called emotional diversion, and thereby caused increased muscular impotence. In every case of writer's cramp that the author has seen there has been an emotional factor. Those who have had the most obvious physical cause for their troubles have complained that their troubles are worst in the presence of others, and especially when they have been called upon for official signatures before official witnesses. Emotional natures are those which are most prone to suffer from derangement of educated actions. We have seen one case, and only one, in which no cause, save an emotional one, could be discovered for the failure in writing, and it is right to state that there was nothing objectively wrong with the handwriting, the patient merely stating that 'he felt a difficulty.' In two or three others emotion, coupled with very trivial causes, such as alcoholism or slight neuralgia, had caused the difficulty, and in these patients again there was very little amiss with the writing. In some cases rheumatic stiffening of one or other of the joints of the thumb had rendered certain combined movements im-

possible, and had thus destroyed the automatic character of writing.   In others the troubles seem to have originated in stiffness of the shoulder and weakness of the deltoid (insidiously following rheumatism).   In four cases a general strain of the arm had produced (through implication of the nerves) general weakness of many of the muscles.   In the remainder of the author's cases the difficulty in writing has been occasioned by excessive use of the pen, and has arisen for the most part in persons of energetic temperament who have written against time to accomplish some task.   In these the failure of writing was undoubtedly due to chronic fatigue of some of the writing muscles.

Now, immediately one small muscle—such as the first dorsal interosseous—fails, the act of writing ceases to be automatic.   The attention of the scrivener is required, mental effort is necessary, and mental effort for the accomplishment of muscular acts means excessive stimulation of the muscles employed.   When once the patient becomes conscious of his troubles the disease is sure to spread rapidly ; and, for the same reason, if he uses his left arm it is sure to give out very quickly.   In these extreme cases the patients invariably suffer from the acute pain of over-fatigue (sometimes accompanied by headache) whenever they attempt to write.   The fatigued muscles will be found to respond less thoroughly to faradism than the healthy muscles of the opposite limb, and the patient either finds that voluntary action of these muscles is next to impossible or, if possible, is very quickly arrested by fatigue. In the author's experience it is not true, as is stated in most text-books, that all acts save that of writing can be perfectly accomplished.   It must be borne in mind that for the accomplishment of most acts we have a great choice of muscles, and hence muscular failure may be difficult to detect ; but it will be certainly observed that *all acts involving the affected muscles* are either impossible or performed in a clumsy manner. The phenomena of writer's cramp vary somewhat.   They generally consist of mere impotence, without evident spasm or paralysis.   Occasionally, however, there is cramp in the fatigued muscles, and sometimes spasm of the muscles which are anta-

gonistic to them is observed. One definite cause of spasm is undoubtedly faulty antagonisation, and whenever we investigate local spasmodic action we must be careful to eliminate this cause.

CASE 58.—*Writer's Cramp. Reported by the Author in the* 'Practitioner,' Vol. 2, 1873. Henry Millerd, aged 40, in the employ of the General Post Office, came to consult me at the Charing Cross Hospital on October 5, 1872. He is a 'nervous man, has suffered from neuralgia, and has one sister who suffers from paraplegia. Has done an immense amount of writing during the last thirteen years, often writing 'against time.' States that in the summer of 1868 he felt that he had lost control over his pen, and was obliged to grasp it tighter. He managed, however, to continue writing tolerably well till April, 1872, when 'he lost control over the fore-finger,' and was obliged to begin holding his pen by interlacing it among the fingers. He cannot rest his wrist upon the desk when writing ; if he does so, the fingers and thumb 'start away from the pen.' He complains of deep-seated pain in the wrist-joint and in the carpo-metacarpal joint of the thumb. He has now completely lost the power of writing with his right hand, and has learnt to use the left ; but, strange to say, he is quite unable to write with the left hand without making spider-like movements with the fingers of the right.

Any attempt to use the right hand causes agonising pain from the shoulder downwards. The pain is the pain of intense over-fatigue, and does not follow the lines of the nerves. On stripping him it was found that there was nowhere any detectable wasting of any muscle, but that both arms were soft and flabby, and the right rather smaller than the left. On being asked to write, the whole body seemed to take part in the exertion ; the pen was grasped as tightly as possible, the forearm raised off the table, the shoulder elevated. An incomplete signature was all that could be effected, and the attempt caused great pain in the limb. On testing with faradism it was found that the interossei, the abductor pollicis, the flexor longus pollicis, and the extensor primi internodii on the right side contracted far less readily and less

forcibly than their fellows of the opposite limb. Voluntary movements of these muscles were performed with the greatest difficulty, as was also the movement of supination, which could only be effected four or five times in succession, notwithstanding the strongest efforts to do so. The phalangeal joint of the right thumb has a curious habit of 'snapping' audibly at intervals, which is due, apparently, to the spasmodic and unequal action of the muscles moving it. The nails of both hands are very bad, being 'pitted' in an extraordinary manner.

His condition is ever present to his mind, and his mental state is one of great depression, his writing power being his source of income. Lithographed facsimiles of this patient's handwriting are given, which show how rapid was his improvement under treatment. On the first application of the current the pain in his arm disappeared, and within a week of its first application he could manage to sign his name with tolerable ease. His writing from this time rapidly improved, and at present his handwriting, when he is allowed to use his right hand (which is very seldom), is, as far as appearances go, as good as it ever was. Samples of his handwriting are given in the accompanying lithograph. The first three samples were written respectively on October 5, 12, and 21, and the fourth on November 1. The other two samples are dated. Although the handwriting is now very good, the pen-prehension is still faulty, and I have forbidden him to write until he can do so with the pen held properly and lightly between the thumb and first two fingers. Pen-prehension has gradually very much improved, and it was soon noticeable that the effort of writing had much decreased, and such efforts soon ceased to cause fatigue. First the elevation of the shoulder disappeared ; then the forearm could be rested on the table during writing. Next the phalangeal angle of the thumb no longer gave way, and now he can write very comfortably if he holds the pen only between the thumb and first finger (at one time the first finger could not be placed on the penholder), but any attempt to use the middle finger bothers him very much. On testing the muscles with faradism, it is now found that the dorsal interossei on either side of the middle finger

1. *Henry M.*

2. *Henry Miller*

3. *Henry Millerd*

4. *Henry Millerd*   2 Oct 1872.

Henry Mullerd
14/12 [ ]

s:

173 Manor St Claptham

the weather is fine but cold

Henry Mullerd
2 x9 march 172

6

Written while awaiting my removal to
the death

respond *far less* readily than their fellows of the left hand, but that the other interossei, which were weak in the first instance, have recovered their normal irritability. Earlier in the case it was evident that the dorsal interosseous muscle on the radial side of the middle metacarpal bone was less irritable and more weak than the one on the ulnar side ; and by placing a rheophore on the metacarpal bone so as to influence equally the muscles on either side the first phalanx was flexed, but dragged by the stronger muscle towards the ulnar side.

The galvanism has been used in the manner stated at p. 41, and the variety of exercises which he was made to practise was very great. Especially he has been made to hold a pen or pencil, or my finger, and to perform with it the movements of writing a hundred times in succession, while the current has been applied alternately to the nerves supplying the muscles implicated. At first the difficulty of this exercise was extreme, the pen frequently slipping from the grasp of the fingers. At present it can be performed without any difficulty whatever. The only movement at present in performing which there is the slightest difficulty is the waggling of the middle finger from side to side by means of the interossei. It was noticeable very early in the case that the forearm had got firm and muscular, and that the pitting had disappeared almost entirely from the finger nails. The patient's enjoyment of the current is quite remarkable, and during its application he frequently uses such expressions as ' That's comforting,' ' That seems to give me strength,' &c. The general health has much improved, a fact which is attributable in great measure to the removal of the cause of his mental depression as well as to the mixture of strychnine, perchloride of iron, and nitro-muriatic acid which he has been constantly taking. The snapping of the thumb has almost disappeared, and is now far less frequent and less loud.

He has continued to use the left hand for writing while he has been under treatment. The movements of the right fingers while writing with the left hand have quite disappeared. Once or twice he has complained of a feeling of awkwardness with

the left hand, but this has always been immediately removed by the current. The amount of writing which he has done during the treatment has been about two hours a day, but this amount had to be performed often rapidly and *against time.*

The following cases are of sufficient interest to bear quotation. Those not included in the original papers are distinguished by letters only.

It is important to remember that not a few cases of neuritis are traumatic, or distinctly due to injury or to excessive muscular effort, and not infrequently the history of injury or strain is only elicited on cross-examination.

CASE A.—An under-gardener, aged 18, strains his arm while wielding a spade. The next morning he cannot milk the cow, and has all the symptoms of neuritis of the right median nerve.

CASE B.—*Neuritis from strain.* M. B., 39, surgeon, in August, 1883, while pulling at a rope in a small yacht, fell backwards, in consequence of the breaking of the rope, and bruised the right axilla against the edge of a hatchway, and generally strained the muscles. Felt very little inconvenience at the time, but later in the day, after exposure to cold, felt 'rheumatic' pain in the arm, and the following day the arm was stiff and painful. The acute symptoms passed off in a day or so, but in a fortnight's time the musculo-spiral nerve was distinctly tender over the head of the radius, and the use of the extensors of hand and wrist caused pain, and the jolting of a railway carriage caused much jarring and discomfort. For many months this gentleman suffered from some disability of the right hand after prolonged writing or during any strong effort. The musculo-spiral nerve remained tender also for months. The symptoms were much relieved by blisterings and applying a piece of capsicum plaister over the tender nerve. Twice, when the arm was apparently very nearly well, the symptoms redeveloped, once after playing lawn tennis and on another occasion after making strong muscular efforts to reduce an old dislocated elbow.

The patient recovered completely, except for some slight numbness in the area of the distribution of the radial nerve,

and also at the top of the palmar surface of the *middle* finger. These regions have been numb throughout. The numbness of the middle finger probably indicates that there must have been some damage to the median as well as to the musculo-spiral nerve.

CASE C.—*Neuritis from pressure.* (Kindly communicated by the patient, March 17, 1881.) Dr. N. M. travelled from Scotland in a *coupé lit* on the night of February 9, 1881, and slept with his right arm 'extended between his body and the side of the couch.' On awaking the right forearm was 'asleep,' and this condition continued. On February 10 a bruise was noticed on the upper part of the arm, and the musculo-spiral nerve was distinctly tender. For some days this pain increased, and was sometimes almost severe when the arm was in a position causing pressure of its muscles on the nerve. Sensation was perfect, and all the muscles responded perfectly to faradism. After a fortnight much inconvenience was felt from the fatigue caused by writing, and also after making post-mortem examinations, which was the patient's almost daily occupation. By March 10 the writing power began to improve, and by March 17 (36 days after the injury) it was almost perfect again. Writing with a quill pen was less fatiguing than writing with a steel pen. General conditions affected the local trouble, and one day, when the patient spent 'about five hours in the post-mortem room with a good deal of decomposed material the arm was so inclined to "pins and needles" and so fatigued that he was able to do very little work in the evening.' 'I have had,' he says, 'precisely the same kind of pain (only in both arms and back) as the first symptom of an attack of intermittent fever. Though I took quinine, I feel sure that the affection I have described was not neuralgia of malarial origin, as my temperature was always normal.'

CASE 21.—*Neuritis from strain.* Mary Anne C., sempstress, 27 (U. C. H., October, 1875), was kindly sent to me by Mr. Morrant Baker. In June, 1874, she was working inordinately hard, 'often nearly all night,' and one day, after using a very big pair of scissors for 'cutting out,' thought she had

strained her wrist. She then lost the power of plying her needle, and the arm became comparatively useless for all acts, whether coarse or fine.

When first seen (October, 1875) there was marked *tremor* of right hand, *tenderness* over the median, ulnar, and musculo-spiral nerves, and a great tendency for the hand to 'die away' and to get red and mottled. Using the arm or hand gave her great *pain*. She had, she said, been faradised, but this had most distinctly made her worse. She improved *very slowly*, blisters over the tender nerves seeming to do her most good combined with tonics and iodides of iron and potassium. By June, 1876, she was able to do a little work, and the hand had ceased to 'die away' and get mottled, but in the following October she returned because the weakness of her hand had come back again. In November, 1876, she had a crop of *five boils*, almost simultaneously, scattered over the right arm. She has since been lost sight of.

CASE D.—*Neuritis from strain*. A. C. S., 50, barrister, July 29, 1885, complained of a numbness and want of power in the right hand, which made him nervous and apprehensive of a paralytic attack. Had recently been trout fishing for many hours daily and wielding a rather heavy fishing-rod. On examination the right musculo-spiral nerve was found excessively tender and the right median slightly so. There was numbness and tingling over the area of distribution of the right radial nerve in the hand, and in this area the sun had produced spots of sunburn in well defined patches of a size from a shilling to a threepenny-piece and of a rich leaf-brown colour.

A piece of capsicum plaister was ordered to be worn over the tender nerves, and the patient was soon quite well.

CASE E.—*Neuritis from strain*. A laundress, aged 34, was seized with neuritis of the right median and musculo-spiral nerves after very hard wringing of clothes. Both nerves were tender and sensation was impaired over their respective sensory areas in the hand.

There was burning pain, coming on in severe paroxysms, accompanied by throbbing and swelling of the thumb and a

tendency for the hand to close. Rest, blistering, some purgation and abstention from beer brought about a complete cure.

CASE F. — *Compositor's Cramp.* James H., 31, compositor, November 30, 1880. Has a difficulty in filling his 'stick.' When the right arm is moved away from the side and he attempts to move the right hand *from* the case towards the 'stick,' held in the left hand, *the elbow is forcibly jerked up and rotated inwards.* There is pain round the right scapula, and when, being stripped, he performs the movement in which he finds a difficulty, the *rhomboids,* supra and infra spinatus, and the upper fibres of the pectoralis major are seen to contract forcibly, while the base of the scapula projects unduly from the side. There is no difficulty in pushing the arm forwards, which requires a strong contraction of the serratus magnus. On faradising the muscles there is found to be some want of irritability in the front fibres of the right deltoid, and he says that the sensation (to faradism) over the right deltoid and pectoralis major is less acute than on the left side.

His difficulty seems to be to maintain the humerus, with the elbow bent, away from the side. He is equally powerless to comb his hair with his right hand. States that he could 'clean his boots' with his right hand, and this, at first sight, was a movement very similar to that of combing the hair or 'filling the stick.' On inquiry, however, it was found that he invariably cleaned his boots on his feet, and this is a very different movement. It is probable that some muscle necessary for maintaining the proper position of the humerus has become fatigued (possibly the anterior fibres of the deltoid or the serratus or supra spinatus).

CASE G.—*Compositor's Cramp.* J. P., aged 45, compositor, came to Charing Cross Hospital on April 19, 1873, complaining that during the past nine months he had been obliged frequently when at work to lay aside his 'compositor's stick' in consequence of 'the jumping of the nerves of his left arm.'

He had had frequent 'hysterical fits' with attacks of

giddiness. There was obvious tremor of both hands and a doubtful 'lead-line' on the gums. This patient was lost sight of.

CASE J.—*Cramp from tying pickle jars.* Neville, J., aged 31, traveller (U. C. H., October 13, 1875). This patient was sent to me by Dr. Cooper. It has been his business to cover pickle jars with bladders. Used to tie down 800 or 1,000 a week, and sometimes has done as many as 200 in a morning. When the knot is tied and 'hauled taut' the top of the jar is grasped with all possible force with the left hand. In February last he began to suffer from a twitching of the left hand, and in July complained of pain at the lower part of the cervical spine.

When seen there was marked tremor in the left trapezius, biceps, deltoid, and the flexors and extensors of the arm and fingers. There was marked tenderness over the sixth and seventh cervical and first dorsal vertebræ. The tremor was not continuous, but came on during exertion and immediately he imitated the act of pickle-jar tying. No pain in the arm, no paralysis, no impairment of sensation.

Ordered a blister to back of neck, rest from his work, and tonics, and in six weeks' time he was far on his road to recovery. The tremor had gone, and he could pick up a scrap of paper with the left thumb and forefinger without tremor or other difficulty. This he could not do when first seen.

CASE K.—*Paralysis of both hands from exposure to cold.* J. H., aged 43, master mariner (U. C. H., September, 1882). In June 1882 made a very bad passage from Boulogne to Folkestone. Was three hours on the bridge of his boat in a biting east wind and rain. He had left his warm gloves at home, and was grasping the iron rail of the bridge the whole time. The next day he found his hands clumsy at writing, and they have got weaker ever since.

All the intrinsic muscles of both hands (median and ulnar) are wasted, and give degenerative reactions. Extrinsic muscles of the hand (long flexor of thumb, flexors and extensors of fingers) are not affected.

No fibrillary tremor observable.

Had a very strong hand, and states that he used to be able to squeeze a pewter pot flat.

CASE L.—The following case seems to be similar to one recorded by Mr. Brudenell Carter in the 'Clin. Soc. Trans.,' and illustrates the occurrence of fatigue in muscles other than those of the hands.

Mr. A. C., aged 21, watchmaker (May 8, 1879), was sent to me by Dr. Mitchell Bruce. Complains that after working with a watchmaker's lens he gets a pain in the inner corners of his eyes and that the sight becomes dim and blurred. On asking him to hold the frame of his lens, with the lens itself removed, he could do so without difficulty. His trouble was not therefore in the orbicularis. When the eyes were made to converge by looking at a near object the pain was soon developed. Mr. Brudenell Carter saw him with me in consultation, and ordered a pair of prismatic glasses with the bases outwards. This quite removed the difficulty. It should be mentioned that the trouble had come on after a severe fall from a bicycle and while the patient was in second-rate health.

CASE M.—A 'cello player, aged 26, who experienced difficulty with the bow. This was found to be due to rheumatism of the extensor tendons of the right hand with tender nerve-trunks. A small ganglion ultimately developed on the back of the right wrist.

CASE N.—A lady, aged 25, violinist, had trouble with the bow hand. She had been overworked and underfed. On examination she was found to be *extremely left-handed*, and could write as well with the left hand as the right. The left hand and foot were both larger than the right, but there was no want of symmetry about the face. The right arm, however, was $\frac{3}{4}$ inch longer than the left. This she attributed to her profession, and asserted that it was 'literally universal among violinists for the right arm to be longer than the left.' This she attributed to the constant stretching of the right arm with bow exercises—especially in early life. Since this assertion was made I have only had an opportunity of measuring the arms of one professional violinist, and certainly

found the right arm, measured from middle of sternal notch to tip of mid-finger, to be $\frac{3}{4}$ inch longer than the left.

CASE O.—Mr. J. P., aged 36, violoncello player, October 28, 1879. In the spring of 1877 felt an aching pain down the left thumb and the tip of the left forefinger while practising the violoncello. There is now a numbness and fulness in the tips of the left forefinger and mid-finger when they hang down, and a feeling of 'pins and needles' when he fingers the instrument. He is in the habit of playing solos on the violoncello, and makes the nut with the radial border of the phalangeal joint of left thumb (at which point there is a corn), while he does the fingering with the left fingers. The abductor pollicis is very notably less irritable to faradism on the left side than the right, and the sensation of the whole of the left hand is perceptibly impaired.

CASE P.—A violinist, aged 45, could not 'stop' the string with his left little finger without consentaneous movement of the other fingers. There was numbness of the left hand, and he was found to be suffering from gouty neuritis.

CASE Q.—Miss F., aged 33, consulted me in 1893 with rather indefinite signs of neuritis of the right arm. She was neurotic and *extremely left-handed*. Wrote fluently with the left hand, and backwards (mirror fashion). This she accomplished without the slightest effort, and seemed amused that I should think her performance in the least strange.

CASE R.—In connection with left-handedness, allusion may be made to a gentleman, aged 39, who consulted me for writer's cramp. He had 'never been a good writer,' and he was aware that his right arm and hand, leg and foot, were smaller than the left. This was obviously the case, *but he was not left-handed*.

CASE S.—A silk weaver, aged 29, consulted me because he could not release the shuttle from his left hand. 'If he thought about it,' he said, 'the fingers closed instead of opening.' A careful examination revealed very little amiss with this patient. The left scapula was a little lower than the right, the left rhomboids were slightly wasted and the left palpebral fissure looked smaller than the right.

CASE T.—A lady post-office clerk, aged 29, consulted me for writer's cramp in 1890. It appears that in 1883 she got painful contractions of palmar fascia and neuralgic pains in fingers. In 1885 her ulnar nerve had been stretched, and in 1887 her mid and ring fingers were removed. She now wrote with her thumb and index, and was found to have painful nerve-trunks and interossei muscles deficient in irritability on both sides.

CASE U.—A gentleman, aged 54, consulted me in September 1893 for rheumatism of the right arm and shoulder. He suffered great pain, which was often periodic and came on at the same time every day. In November 1894 a piece of a sewing needle nearly an inch long and quite black worked out of his arm near the insertion of the right deltoid muscle. How the needle got there he never knew, but with the escape of the needle his symptoms disappeared.

[It is interesting to record that about this time another patient of mine developed an ischio-rectal abscess, from which a crooked black pin was liberated by the surgeon.]

One case of 'writer's cramp' which was sent to me I found to be caused by marked thickening of the first metacarpal bone, the result of injury.

It is interesting that I can only find notes of two telegraphists who have consulted me. These were both middle-aged men, both beer drinkers, both had tremor which affected all acts performed with the right hand. They were both, in short, cases of gouty neuritis and general nervous degeneration.

I have never yet seen a true case of 'telegraphist's cramp.'

CHAPTER III

A CASE OF TAILOR'S CRAMP, AND OTHER TROUBLES AFFECTING THE FUNCTIONS OF THE HAND [1]

THERE are many diseases which are caused by the occupation of the patient. These might be divided into at least three classes, viz. : (1) *Irritant*, in which the mechanical or chemical irritation of the materials with which the workman is brought in contact is the cause of trouble. Examples are found in the eczema of the baker or grocer, or the phthisis of the stonemason or knife-grinder. (2) *Toxic*, in which some poisonous matter is absorbed into the blood. Such are chronic mercurialism and the lead poisoning of plumbers and painters. (3) *Fatigue diseases*, in which the constant repetition of some muscular movement which constitutes the very essence of the trade or handicraft produces an inability to perform such movement. The most common example of this last class is 'writer's cramp.'

CASE 1.—The first case to which I wish to direct your attention to-day belongs to the class of fatigue diseases, and may be shortly spoken of as a case of 'tailor's cramp.' The patient is a tailor, aged 57. He has lived in London for the last forty years. He is well built and temperate, and is free from disease other than that for which he seeks advice, and there are no facts in the family or personal history which throw any light upon his condition. About a year ago the patient began to experience a 'weakness' of the right thumb and forefinger, which made it difficult for him to hold his needle, and this difficulty increased until about eight weeks ago, when he was obliged to give up work. The patient is here, and we will ask him to demonstrate his trouble ; and it is quite characteristic of this class of malady that there is

[1] Clinical Lecture, U.C.H., 1890.

very little to see. We talk of writer's cramp when there is no visible cramp or spasm, of writer's palsy when there is no obvious paralysis, and you will observe in this case that after a few stitches the needle eludes the grasp of our tailor, and it is rather difficult to say why. The needle is held between the thumb and forefinger. The metacarpal bone of the thumb is abducted and partly flexed on the palm, the near phalanx is slightly flexed on the metacarpal bone and slightly rotated towards the radial side, while the far phalanx is fully extended, so that the phalangeal knuckle makes an approach to a re-entrant angle. (See fig. 1.) This is well shown in a drawing made for me by Mr. Wells. The needle is held between the pulps of the index finger and thumb, and in order that these points may be in apposition the middle and far phalanges of the index are both slightly flexed, and pressure appears to be maintained by the flexion of the near phalanx. After working for a short time the thumb and forefinger give way, the needle is no longer grasped with sufficient firmness to push it through the fabric, and in this failure the most noticeable fact which an observer can detect is the giving way, as it were, of the phalangeal joint of the thumb, the extension of the far phalanx not being maintained.

We will now proceed to examine the patient to ascertain why the failure takes place, and in order to do this satisfactorily we must have him stripped to the waist. Having done this, we first determine that there is no sign of hemiplegia, however slight. The movements of the tongue and face are normal and symmetrical, the pupils equal and reacting normally, the grasp of the two hands firm and equal, the gait without a trace of limp. There is no sign of any coarse cerebral lesion, whether old or recent. It is important to determine this fact, because it often happens that the failure to execute some delicate manipulation constitutes the one lingering sign of an otherwise scarcely noticeable hemiplegia. We next look carefully to the movements of the scapula, because any want of firmness in the scapula necessarily entails a corresponding want of firmness and certainty in the use of the hand. We ask the patient to shrug his shoulders,

to extend his arms in front of him, to raise his arms above
the head, to try to make his elbows meet behind his back, and
by this series of movements we ascertain that all the muscles
acting on the scapula act normally on the two sides. The
movements of the arm and forearm likewise appear normal.
There is no wasting of muscles, no change in the knee-jerks,
no sign of ataxy in the legs or arms. There is no tremor of
the hands or arms, or lower limbs. Asking the patient to
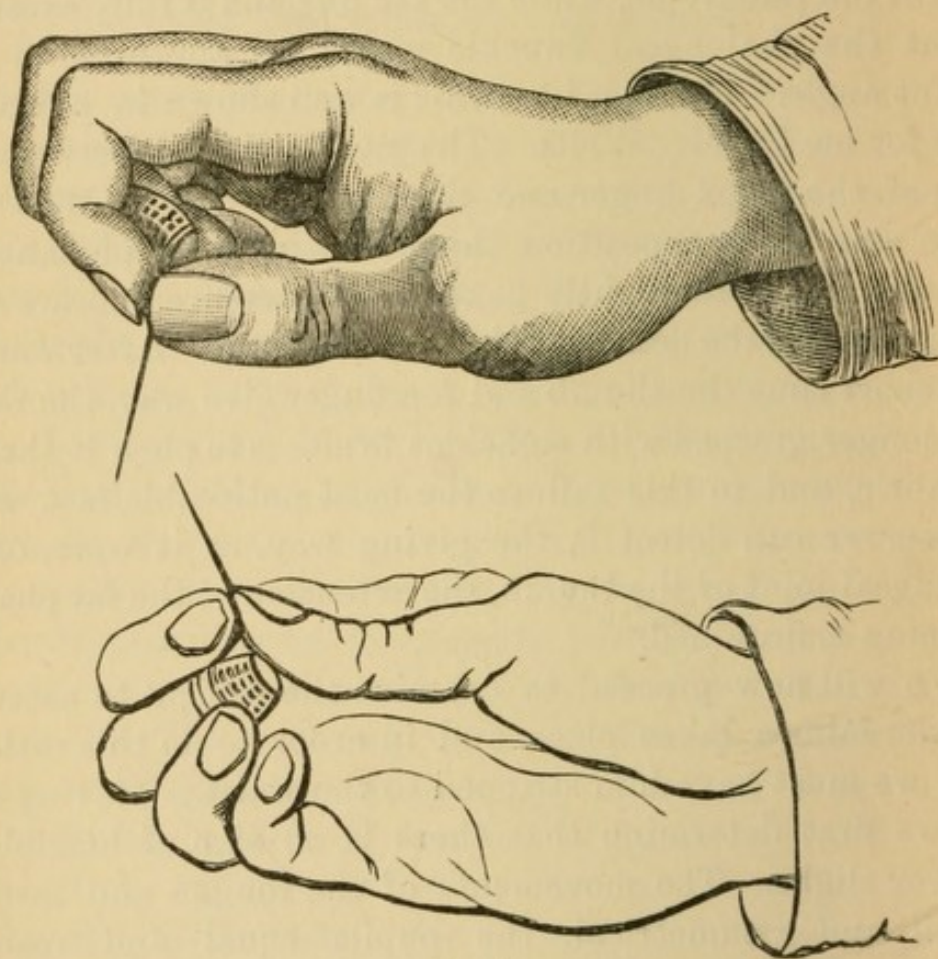
Fig. 1.

stand facing me and to rest his arms upon my shoulder, with
the palms uppermost, I cross my own arms, and, grasping his
elbows, make gentle pressure with my thumbs over the median
nerve as it lies to the inner side of the biceps tendon. There
is very distinct tenderness on the right side, but not on the
left. *The trunk of the right median nerve is distinctly tender.*
I find also that there is a very tender spot in front of the
right wrist just where the median enters the palm, and a

third tender spot in the palm itself at the base of the thenar eminence. There thus appears to be well marked tenderness over the trunk and branches of the median nerve in the fore-arm and palm. There is no similar tenderness over the musculo-spiral or ulnar nerve. The sensation of the right hand appears to be duller (front and back) than on the left, but this blunting of common sensibility is especially marked in the 'median' area of the palm, and you will observe that the patient distinctly states that sensation is far more acute on the ulnar side of the right ring finger than on the radial side. This impairment of sensation is slight in degree, but, if carefully looked for, is perfectly evident. We have so far shown that the right median nerve is tender, and that its sensory functions are impaired. Looking more closely to the hand, we find no wasting of muscle, no impairment in the strength of rapid movements, no enlargement of joints, no creaking about the wrist, no teno-synovitis, or other trouble interfering with the freedom of movement of the tendons. The muscles of the ball of the right thumb are perhaps harder to the touch than those of the left side, but the difference is not very marked. Proceeding now to test the muscles with faradisation, I ask the patient to sit on a common Windsor chair with the wrists resting on the back of a second chair, upon which I sit astride for the purpose of testing the muscle. One rheophore of the faradising battery is made fast to the nape of the neck in order that the current to reach the hands may pass through equal lengths of the body on either side. The muscles of the hand are then tested with a small rheo-phore, the skin being well moistened with hot salt-and-water. I begin with the left hand, taking the muscles singly, and, finding what is the least current which will produce a contrac-tion, I then watch the effect of the same current applied to the corresponding muscle of the right hand.

The result of this investigation is to show a very distinct impairment of irritability of the first dorsal interosseous and the muscles of the thenar eminence on the right side. A current which produces a quick and vigorous contraction on the left side has no appreciable effect on the right. When the

strength of the current is increased, the right hand muscles
contract, but with less vigour than the corresponding muscles,
and, especially in the ball of the thumb, there is a little hesi-
tancy about the contraction, owing apparently to a lengthening
of the latent period of stimulation. This difference in irrita-
bility was very much more marked a month ago than it is at
present, rest and other measures having done much to restore
the normal condition of the muscle. Now it is obvious that
the muscles in which we are able to demonstrate this impaired
irritability are precisely those which are essential for holding
the needle ; for keeping the needle firm between the pulps of
the thumb and index the muscles of the thenar eminence and
the first dorsal interosseous are precisely those which are
employed. Duchenne pointed out that the muscles of the ball
of the thumb are analogous to the interossei muscles in the fact
that their action is to flex the near and extend the far phalanx.
The radial head of the short flexor and the abductor pollicis
may be regarded as functionally one. These maintain the
thumb in the needle position ; they are markedly deficient in
irritability, and the ' giving way ' of the phalangeal angle is
due mainly to the failure of these muscles to remain con-
tracted. The first dorsal interosseous is similarly deficient in
irritability, and there is no doubt that the impaired condition
of these muscles as demonstrated by electricity is amply suffi-
cient to account for the ' professional impotence ' of our tailor.
We have also as an interesting fact in this case the well-
marked tender spots along the course of the median nerve, the
nerve supplying those muscles of the thenar eminence whose
loss of irritability is especially well marked, and also the
markedly impaired sensibility in the median area of the palm.
There is no well marked tenderness along the course of the
ulnar nerve (supplying the first dorsal interosseous), although,
be it remembered, there is some dulling of common sensibility
in the hand as a whole. Having found that our patient
has the muscles which are essential for holding his needle
functionally impaired, and also well marked evidence of
impaired functions in one of the nerves supplying those
muscles, his condition ceases to be a mystery, because we

can put our fingers on the precise anatomical seat of his troubles.

We have made an anatomical diagnosis. Can we go a step further and make a pathological diagnosis? If we could see this patient's median nerve and the muscles which have their irritability impaired should we be able to see anything amiss either with the naked eye or the microscope? The median nerve is tender and the involved muscles are perhaps a little harder than their corresponding muscles of the left limbs. Is the nerve slightly hyperæmic, and is there a slight increase of cellular tissue in the muscle? Possibly, but in the absence of any observations it is wiser to say we do not know. The condition is brought about by excessive work, and the electrical reactions are those of fatigue, so that we are quite justified in talking of the condition as one of 'chronic fatigue,' especially as such a term does not involve any assertion as to the histology of the implicated muscles or nerve.

Next a few words as to treatment, and as this patient has greatly improved since he has been under observation I will recount what has been done for him. In the first place, he has been ordered to desist from his occupation, rest being the most essential part of the treatment. Rest from the occupation which has brought about the trouble is sufficient. There is no need to put the arm in a sling, as is occasionally done. No harm will result in this case from the use of the arm for all ordinary purposes so long as the fatigue of the thenar muscles and the first dorsal interosseous be not intensified by excessive use of the needle. After rest the next step in treatment is to diminish the tenderness of the nerve-trunks, and to this end we placed three blisters over the tender points along the course of the median; as a result of this the tenderness has now almost completely disappeared. My experience of these cases has led me to consider that it is very undesirable to use electricity or massage for the muscles so long as the nerve-trunks be tender, and if this be done I believe there is no little risk of making the patient worse rather than better. When, however, any nerve-tenderness which may have been present has been removed by blistering,

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we may have recourse to electricity or massage with a fair chance of benefiting the patient.

The use of electricity is attended by good results in a fair proportion of these cases. The galvanic current must be employed, and not the induced current. The latter produces strong tetanic contractions of the muscles, and is apt to intensify the fatigue which it is sought to allay. It is possible that an occasional application of the faradic current may do good by quickening the blood and lymph currents in the muscle, but anything like a continued employment of this form of electricity is calculated to do harm. With the continuous galvanic current, used of a strength sufficient to redden the skin and produce a slight movement in the muscles, it is otherwise. The refreshing effect of this current when applied to acutely fatigued muscles is very remarkable. You will remember that I demonstrated this fact a few days ago, when I asked the patient to keep his arm extended at right angles to his body for as long a period as he could ; and you will remember that the passage of a galvanic current through the arm just when the fatigue pains became almost too intense to bear removed the pain, refreshed the muscles, and enabled the patient to continue the extension of his arm for a very long time. The same refreshing effects are observed when the current is used for the relief of these chronic fatigue diseases, and I strongly advocate its employment in cases where the irritability of the muscles is lessened and the nerve-trunks are *not* tender. The employment of massage is very beneficial, but it must be used with judgment, and, like electricity, it must not be used while there is any tenderness of the nerve-trunks.

The massage should be directed to the whole of the implicated limb, and especially to those groups of muscles in which the irritability is lessened. Massage, like electricity, reddens the surface and causes a flow of blood to the limb, and it further quickens nutrition by increasing the current in the deeper blood-vessels and lymphatics. If fatigue be caused by the accumulation of fatigue products in the blood, and if this condition, which we call chronic fatigue, be due to the same cause, then massage and kneading of the

muscles must commend themselves as means of cure likely to be beneficial. In any case the result of treatment in this case has been very encouraging, and the combination of rest, blisters, electricity, and massage has had the effect of bringing the muscles to something like their normal condition again, and the patient's power of sewing is very greatly increased.

Internal remedies are not unimportant in these cases. The general health must be attended to, and any derangement in the functions of internal organs be rectified if possible. There is one specific remedy which I very often prescribe, and from which I think I have seen good results. This is arsenic combined with iodide of potassium. Our patient is taking one grain of the latter combined with two minims of liquor arsenicalis three times daily. It is difficult to say which of our remedial measures has been most beneficial, but there is no doubt as to his very great improvement. I believe this to be the only true case of 'tailor's cramp' that I have ever seen. I have seen many tailors who had a difficulty in the use of the needle, but in all of the cases the difficulty was only an accidental complication, so to say, of some other trouble, such as alcoholism, hemiplegia, paralysis agitans, or locomotor ataxia.

I have often had occasion to remark that all 'fatigue diseases' are prone to occur in those professions which entail prolonged rather than intermitting muscular contraction. The scrivener may not be able to lay down his pen for hours, but the tailor must relax his grasp of the needle between every stitch. Hence it is that 'writer's cramp' is very common and 'tailor's cramp' is very rare.

The next case is that of a goldbeater by trade, who comes to us complaining that he is unable to wield his goldbeater's mallet with his accustomed force or precision.

Case 2.—B. H. is fifty-seven years old, and has been engaged at his present occupation for eleven years. He has been a soldier, and served in the Crimea. He has enjoyed good health, and there are no facts in his personal or family history which apparently have any bearing upon his present condition. From the account he gives of his trouble we

gather that he beats gold with a mallet weighing about sixteen pounds ; that his difficulty consists not in any cramp or spasm of the hand, but rather in a general weakness, which makes it necessary for him to shift his mallet at frequent intervals into the left hand, which obliges him to grasp the mallet with abnormal firmness, and which inter- feres at once with the force of the blow and the exactness of aim. The patient is thin and emaciated, and has a worn look, as though he had worked hard and had not fed very well. On asking him to strip to the waist, we notice he is thin, but we are unable to detect any morbid condition of the muscles of the upper limbs. There is *well marked tremor* of both hands, especially when they are extended in front of him. This tremor is rather more marked in the right hand than in the left, and on looking attentively at the right hand we are able to say that the tremor is more marked in the thumb than elsewhere. This intensification of tremor in certain places is important, and not infrequently gives us a clue to the muscles which are especially implicated. I have occasionally noticed that tremor in a limb is not noticeable until some group of muscles which is morbidly weak is brought or is attempted to be brought into action. Then the tremor begins sometimes in the limb as a whole, and sometimes only in some small section of the limb, which is moved by a muscle which is especially weak. It is important, therefore, to notice that not only is there tremor of both hands, but that the tremor is more marked in the right hand than the left, and most marked of all in the thumb of the right hand. The tongue is tremulous as well as the hands. The tremor of the hands is not intensi- fied by the attempt to perform any delicate act such as the picking up of a scrap of paper. The palm of the right hand has several corns upon it, but these, he says, are not cha- racteristic of his trade. 'Not one goldbeater in five hundred,' he says, 'has such corns.' 'They are due to my being obliged to grasp the mallet so tightly.' There are one or two corns on the left hand also, which is due to the fact that he is obliged to use his left hand when his right hand gets tired.

Both arms are tender, especially tender over the nerve-trunks. The right arm is more tender than the left, but he shrinks as if in considerable pain when very moderate pressure is made on either arm, especially over the trunk of the median nerve as it lies to the inner side of the biceps tendon. There are no tender points in the palm. On testing the muscles with faradisation it is noticeable that all the muscles respond equally on the two sides, with the exception of the muscles of the right thenar eminence and the right flexor longus pollicis, in which, especially the latter, there is observed a well marked depression of the normal irritability—i.e. a current which causes a forcible contraction of these muscles on the left side has little or no effect upon those on the right side. It is obvious that in the grasp of a goldbeater's mallet the muscles of the thumb are all-important. It is very interesting also to note this depression of irritability, especially in connection with the distribution of tremor, upon which I have just been remarking. Now although we have made in some sort an anatomical diagnosis in this case, and although we have demonstrated that certain muscles of the right thumb, muscles essential for a goldbeater's work, are abnormally weak, I am in doubt whether we ought to regard the case as a true fatigue disease or professional ailment. The patient is nearly sixty, and is emaciated and tremulous, the tremor being quite independent of his work, and noticeable, be it observed, in the tongue as well as the hands. I regard the case as one of senile tremor occurring in an over-worked and ill nourished man, whose arteries are certainly degenerating, and whose nerve-centres are beginning to be imperfectly nourished. When senile tremor commences it is not surprising that it should show itself most and earliest in the muscles which are most used. This seems to be the case in the patient under observation. The man is very feeble, and especially feeble at his trade, which is a laborious one. Such cases must not, I think, be classed with the true professional ailments caused by fatigue, in which an otherwise strong and healthy subject finds himself incapable of some one delicate manipulation. The treatment in the present case must be

directed to the improvement of the general health, food and warmth, rest and tonics.

Case 3.—The next case I have to bring to your notice is one in which a man has been rendered unfit for following his trade owing to the toxic effects of the trade which he follows. It is, in short, a case of mercurial tremor. The patient is an Italian aged sixty-two. He came to this country when he was seventeen years old, and for the past thirty-four years he has been a looking-glass silverer. His duty is to 'run' the mercury over tinfoils spread upon sheets of glass. He is therefore constantly handling mercury, and is exposed to its fumes. He is a strongly-built, dark-haired, olive-complexioned man, of cheerful manner and well nourished. He is hard working and temperate, and there are no facts in his imperfect family history which have any bearing upon his present condition. A careful medical examination leads to the conclusion that, with the exception of the tremor, the patient is in perfect health. The tremor affects the hands mainly, and the right hand more than the left. When the patient is at rest the hands and arms are steady, but the instant that he attempts to use the hands the trembling and shaking become excessive. He cannot pick up a scrap of paper, he cannot feed himself, and is obliged to drink through a glass tube owing to the impossibility of holding a cup steadily to his lips. There is some slight tremor of the legs, but this is scarcely noticeable, and does not prevent him from walking freely about. There is no tremor of the face or tongue, and no marked change of speech. The eyesight is good ; no nystagmus ; no change in the discs. Turning to the hands, it is noticeable that the tremor is very coarse, and that when he attempts to use his hands they jerk to and fro through several inches of space. There is no wasting or paralysis of any muscle, no rigidity or contraction of any muscle, and no deformity of the hand. There is no marked tenderness of any nerve, no joint affection, no trophic change, and the muscles respond normally to electricity. This apparently is the third attack of 'the trembles' from which the patient has suffered. The first was twenty-six years ago, and his arms, legs, and head shook

equally. He was treated for thirteen weeks in the London Hospital and completely recovered. About eight years after the first attack (i.e. eighteen years ago) he had a second attack. In this the legs were mainly affected, and occasionally trembled to such a degree that he was obliged to crawl upon his hands and knees. The arms were also affected, but less so than on the present occasion, for he could always feed himself. At this time he lost several of the teeth, the gums were ulcerated, and there was tremor of the tongue and an affection of the speech so that he could hardly get his words out. From this second attack he also completely recovered and returned to his work. He was able to follow his occupation (except during a short attack of 'lumbago' some eight years ago) until Christmas, 1888, when his arms began to tremble, and, his troubles increasing, he ultimately sought advice here, and after some three weeks' attendance in the out-patient department he was admitted as an in-patient at the end of February, 1889. Our patient was treated with hot sulphur baths and iodide of potassium, and left the hospital much improved and able to feed himself, but still far from well.

Cases of mercurial tremor are of rare occurrence in this country, although cases are met with among workmen whose occupation brings them in contact with mercury or its salts. The danger appears to be greatest in those cases where (as in 'water gilding') heat is employed to volatilise the metal; but, now that electro-plating has supplanted gilding by means of mercurial amalgams, such cases are rarely seen. Our patient is a mirror maker, and therefore belongs to a class of workmen who have always been recognised as liable, but not very liable, to chronic mercurial poisoning. Mirrors are, however, made largely now by means of chemical methods, in which the salts of silver and not those of mercury are employed, and mirror-makers by the old-fashioned method of using a tin-mercury amalgam are a dwindling class. Rare as these cases are, they are likely, therefore, to become more rare. The point of main interest in the case is the *diagnosis*. This is made for us to a large extent by a knowledge that the patient is exposed to mercurial fumes in

his business, and by the patient's own experience, this being the third attack of ' the trembles ' from which he has suffered. But, without the history to guide us, should we feel sure as to the diagnosis ? The tremor in this case is the only symptom of mercurialism, and is the tremor sufficiently characteristic to warrant us in attributing it to that cause ? The tremor began in the hands, and is mainly limited to the hands. This is said to be characteristic of mercurial tremor, although it is no absolute rule, for even in this patient's previous attacks the legs mainly suffered upon one occasion, and upon another the head trembled and speech was affected, which is not the case now ; for, although the patient speaks English like an Italian, there is no other peculiarity of articulation. The tremor is so coarse, jerking, and peculiar that it does not suggest tremor from alcohol, paralysis agitans, senile weakness, or ' general paralysis.' It only occurs when the patient attempts to use the hand for the purpose of a voluntary act. In this particular it resembles the tremor seen in cases of so-called ' disseminated or insular sclerosis,' and the diagnosis, judging only by the tremor, would be between that condition and mercurialism. That the tremor of disseminated sclerosis and mercurialism may be practically identical in form seems to be admitted by Charcot and other observers. The age of our patient is against the diagnosis of disseminated sclerosis, that disease seldom commencing after forty, and terminating fatally within a few years of its commencement. The absence of nystagmus is also against the diagnosis of desseminated sclerosis, as is also the fact that the superficial and deep reflexes are in the present case practically normal instead of being exaggerated, as is often the case in disseminated sclerosis. The absence of any speech difficulty is against the diagnosis of disseminated sclerosis, but since its absence is, so to say, merely accidental, and his present attack differs from his last in this particular, this is a point of not much value. Speech is often affected in both these conditions, so that changes in articulation are of doubtful value in the differential diagnosis of the two conditions.

Case 4.—The last case is that of a mat-maker, aged forty, who was admitted to the hospital with his left arm stiff,

contracted, and absolutely useless, so that he is quite unable to follow his occupation. On examining the arm it was found to be contracted and stiff. The humerus was adducted to the side of the chest, the elbow flexed, and the wrist and fingers flexed. On attempting to move the limb passively it was found that all the joints were very stiff, and creaked audibly. The power of voluntary movement was almost *nil*. Sensation but little impaired. The state of nutrition of the left arm and hand, especially the latter, was obviously defective. The limb as a whole was emaciated. All the muscles were small, but there was no muscle or group of muscles which was atrophied in any special degree. The left hand was very markedly smaller than its fellow, the skin harsh and dry, the finger nails deeply grooved, pitted, and incurved. The state of the hand and the contrast in the two hands are well shown in the accompanying drawings kindly made for me by Mr. Wells, M.B. (See figs. 2 and 3.) The sensibility of the left arm and hand is slightly impaired. All the muscles of the left hand and arm respond normally to faradisation. With the exception of the arm the patient appeared healthy. He was somewhat thin, but all the organs appeared to be in a normal condition. It is not possible to form a judgment as to the pathology of this man's condition without reference to the history. The patient has had four attacks of rheumatic fever, viz., in 1876, 1879, 1881, and 1885, and it was after the last attack that the left arm and hand became stiff and powerless. The history of his last attack is very important, and I may add that it was elicited only after careful questioning— questioning which was instituted because (as will be presently explained) the condition of the left hand and arm was sugges- tive of cerebral mischief. The patient lives at Sudbury in Suffolk, and he states that on March 18, 1885, he felt 'dizzy' before going to his work, that he had to leave his work because he felt ill ; and that on his way home he sat by the roadside, and *lost consciousness for two hours* in consequence, as he says, of faintness. This attack was followed by the 'rheumatics,' which kept him in bed. The left arm was swollen, and re- mained so for three weeks, and when the swelling subsided the

arm was stiff and the fingers contracted. The patient further states that at this time he had some loss of taste on the left side of the tongue, some numbness of the head, and some impairment of vision. It is all-important to bear in mind that the condition of the left arm from which this patient is suffer-
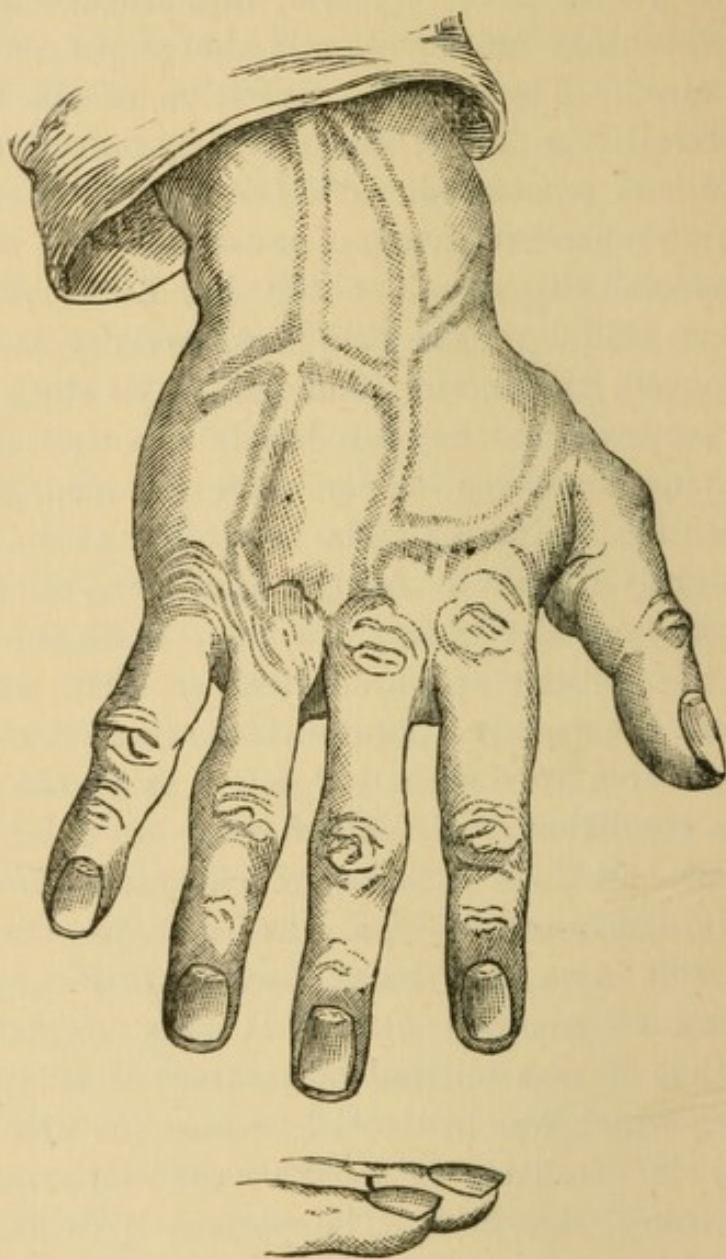
Fig. 2.

ing, although originating in a condition of obvious rheumatic swelling, came on after an attack of loss of consciousness, which was immediately followed by sensory troubles. I do not think that the cause of the arm trouble in this case is purely local and rheumatic, but I am of opinion that there is cerebral

mischief as well, and that one element (perhaps the most important element) of the condition is hemiplegic contracture; and I say this notwithstanding that the evidence of hemiplegia other than the state of the arm is practically *nil*, and there is no cardiac murmur, which might suggest the
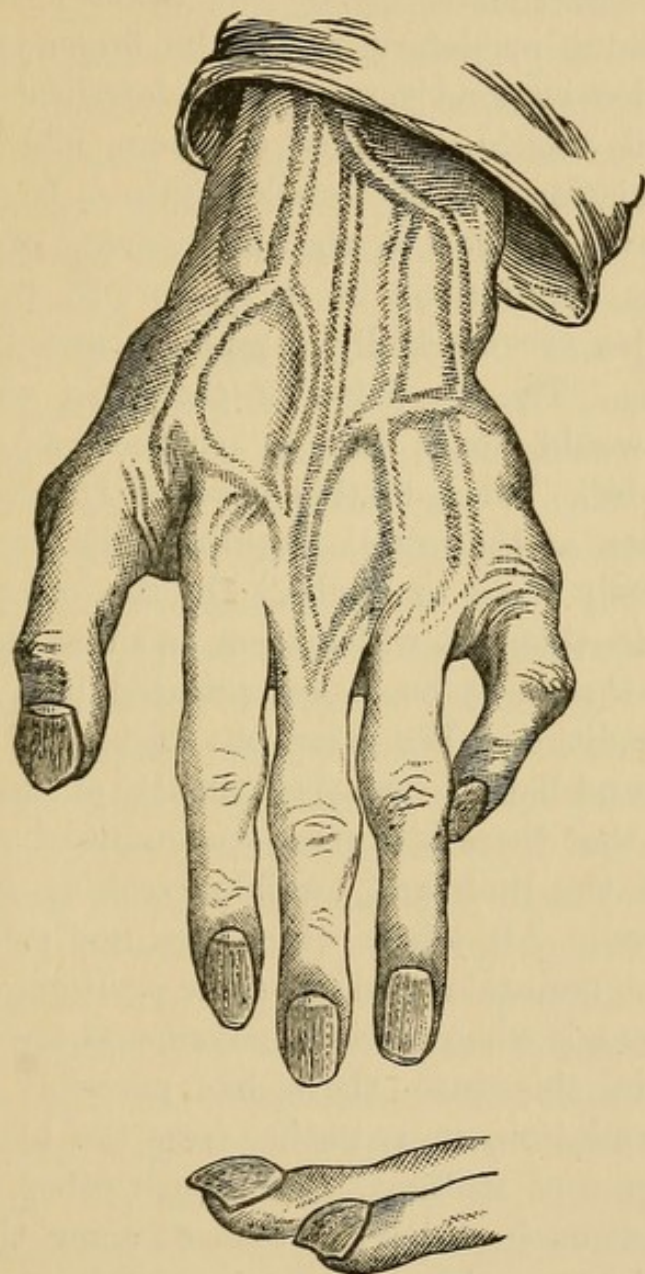
Fig. 3.

probability of embolism as a cause of the trouble. This question is of great importance from the point of view of prognosis. If the trouble be local and rheumatic there are possibilities of absolute cure, which can hardly exist if the voluntary centre for the arm be destroyed. Why, it will be asked, do I

think that there is cerebral mischief ? Because the left
upper limb has all the appearance of 'contracture' after
cerebral lesions. The arm is adducted, and the whole limb
is uniformly flexed, and an examination of the hand shows
that the fingers can be easily straightened, the contracture
being due to a shortening of the flexor muscles, and not
to any stiffening or deformity of the finger joints. Again,
all the muscles respond normally to faradisation, and there
is no marked wasting of any of them, although there is
general emaciation of the whole limb. In cases of old
rheumatic paralysis due to the involvement of nerve-trunks
in rheumatic exudations it is usual to find that some
of the muscles are markedly atrophied and give degenera-
tive reactions. The condition of the arms without a word
of history would lead one to suspect a central cause
for the mischief. When to this is added 'faintness' and loss
of consciousness as the initial feature of the trouble, I think
there is scarcely room to doubt. On the other hand, there
are features in this case which seem to show that hemiplegic
contracture, if such it be, is complicated by a distinct local
rheumatic condition. We have the history of a swelling of
the left arm and hand in his last attack of rheumatism, and
we find now that there is a marked amount of stiffness of the
big joints of the limb and audible creaking when they are
forcibly moved. All joints which are not sufficiently used,
and which are maintained in the same position, are apt to get
stiff, and to creak when forcible movements are imposed upon
them ; but in this case there has probably been definite
rheumatic exudation, as we gather from the history and infer
from the degree of stiffness. There are other considerations
which warrant us in believing that at some time the nerve-
trunks which cross the scapulo-humeral joint were to some
extent involved in and compressed by rheumatic exudation.
My chief reason for this belief is the condition of the hand.
The wasting of it is greater than is usually observed in cases
of hemiplegia ; and the condition of the finger nails, which
are markedly grooved and pitted and incurved, is very
suggestive of a true tropho-neurosis or a change in nutri-

tion brought about by pressure on or injury to a mixed nerve. There is some harshness and dryness of the skin of the left arm and hand, and also some slight impairment of common sensibility which point in the same direction. We must remember that the last rheumatic attack was more than four years ago, and that time enough has been allowed for rheumatic exudations to be absorbed and for recovery of damaged nerve to take place. I therefore regard the condition of the nails and the excessive wasting of the hand as evidence of what has been rather than of what actually exists at present.

The case is not without its difficulties, but, to sum up, I may state my belief that the condition of this patient's left hand and arm is partly due to a cerebral damage and partly to a local rheumatic trouble. What was the nature of the cerebral mischief? As it occurred at the commencement of a fourth attack of acute rheumatism, it very possibly was embolic, although at the present there is absolutely no evidence of any morbid condition of the heart. We are free to imagine a slight degree of hyperæmia and swelling of a valve sufficient to give rise to a deposit of fibrin, and we can imagine such deposit being washed off and lodging in one of the branches of the Sylvian artery, giving us a 'brachial monoplegia' and leaving the heart practically sound. I say we can imagine this, and there is no harm in doing so if we distinguish between imaginings and proved facts. The case presents difficulties from every point of view, but these speculations are by no means idle, because they have a most important bearing on the prognosis, as I have explained. The results of treatment in the case under consideration have been most excellent up to a certain point. When we have to do with a limb which is stiffened, wasted, and beyond, so to say, the reach of the will, our chief aim should be to replace voluntary exercise by every means which is available, and passive movements, rubbing, artificial warmth, and electricity will be found of service. With a limb in the condition of the one we are considering the worst course to pursue is to leave it alone, for it will inevitably result that the disused joints will get, so to say, glued up, and the limb will soon assume a position of stiffened con-

tracture, as has happened in the case under consideration.
Every joint must be methodically worked in every direction so
as to maintain absolute freedom of movement. I do not
think that, unless the stiffness be very excessive, much good is
got by forcible movement under an anæsthetic. If, after such
forcible movement, daily exercises and passive movements be
not persisted in the joint will soon be glued up as tightly as
before, and since the daily exercises are in any case necessary
it is generally advisable to trust to these alone, and to a mode-
rately forcible and judicious coaxing of the stiffened limb,
rather than a too forcible extension. This has been done in the
present case with admirable zeal by my clinical clerk, and with
the best results, as the stiffness and creaking of the joints
have disappeared, and the freedom of movement of shoulder,
elbow, wrist, and finger joints has been greatly increased. The
next point is to improve the nutrition of the limb, and this is
done by causing a ' determination of blood ' to it. The passive
movements of the joints have done much to quicken the blood
stream. In fixed limbs the venous current does not receive the
help of movement, whether articular or muscular, and the passive
movements of the joints and the kneading of the muscles do
much to counteract the evils of this deficiency. Artificial warmth
is all-important in improving the nutrition, and to warmth
should be added a stimulation of the skin sufficient to produce
a thorough redness of the surface. Such a limb should be kept
in a flannel sleeve, and the hand should be covered with a warm
glove so as to run no risk of its getting chilled. Warm bath-
ing is also of great use, and when the trouble is strictly local,
as in this case, a soaking of the limb in hot water is of service,
the soaking being followed by passive movements and massage.
It is of some use, probably, to add salt to the water, as thereby
its stimulating effect upon the surface is increased. Had our
patient been rich he would very probably have been sent to Bath,
Aix-les-Bains, or some similar place, where the machinery for
bathing and rubbing and douching has been elaborated to the
utmost ; but I would have you observe that it is possible to
do a great deal by similar methods employed *with a will* at
home.

Electricity is of great service in these cases. If the irritability of the muscles is lessened the application of faradisation is often very useful, and it should be employed until the irritability reaches the normal. There has been no noticeable diminution of irritability in this case, and none of the muscles give the 'degenerative reactions,' so that the good effects of faradisation have not been as noticeable as sometimes is the case. The good effects of faradisation are, in cases like this, often very marked at first. By its use we succeed in 'stirring up' the disused muscles, and greatly increased freedom of movement is the result. The effect of the first application is often very marked, occasionally it is astounding, while subsequent applications produce hardly any noticeable effect. The continuous current is also extremely serviceable. Large sponge rheophores should be used, and these should be moistened with salt-and-water made as hot as it can be borne. I know of nothing equal to this as an effectual skin reddener, and it is undoubtedly to its power in this direction—the causing a 'determination of blood' to the limb—that much of the utility of the continuous current is due. Cases of this class are often very chronic. Chronic rheumatic affections are very prone to last (with considerable exacerbations and remissions) for a lifetime, and contractures following upon cerebral lesions will certainly do so. The patient's discomforts are very much lessened if we can maintain the nutrition and the suppleness of the limb. This is done by the methods I have indicated, and it is very advisable to use one method at a time and not to exhaust your resources all at once. Human nature loves a change, and will have a variety, and far be it from me to say that change and variety are not of solid use. We all strive to get, and all enjoy, a change in the matter of diet, and that much in excess of what the physiologists tell us is necessary. So the chronic invalid, the nutrition of whose paralysed or rheumatic limb has to be artificially maintained, will have a change of method. It is therefore advisable to use one method at a time. Massage, hot bathing, electricity, liniments, &c., are all means of maintaining nutrition, and when the patient has tired of one

method we may try a second. As soon as the limb is left to itself it will stiffen and get glued up and fixed. The great use of liniments in such cases is found in the fact that they are all, so to say, vehicles for friction. The only thing to bear in mind about them is that they should be oily and not too stimulating. Great care must be taken that no excess of zeal in the matter of rubbing produces any abrasion of the skin, and it is in order to avoid the risk of skin abrasion that I recommend the use of oily liniments, which serve to lubricate the surface. It is needless to say that the patient should be encouraged to use the damaged limb as much as possible, and the exercises pre-scribed will of course depend upon the amount of voluntary power. In order to keep the shoulder- and elbow-joints supple it is a good plan to get the patient to work the limb by means of an elastic band with a stirrup handle, which must be hung to the ceiling or the wall of his room. With methods like those I have been describing our patient has improved very rapidly indeed *up to a certain point*. The joints move easily, and the contraction of the arm and fingers has disappeared. His power of using the limb has not kept pace with the greatly improved local condition, and it is this fact which strengthens and confirms me in the diagnosis of a central cerebral lesion in addition to his local rheumatism. By local measures we may succeed, as we have done, in loosening stiffened joints and in improving local nutrition. There is, however, the scar of an old injury in the area for the arm in the motor region of the cortex cerebri on the right side. This is not to be reached by our rubbings, frictions, and electricity, and, although the local improvement has been immense, there has not been and never will be any commensurate improvement in the voluntary use of the limb. It is easy, therefore, to see the great importance of diagnosis and its influence on prognosis.

CHAPTER IV [1]

CASE OF HAMMERMAN'S CRAMP

THE case which I wish to bring to your notice to-day is one belonging to the class of 'professional ailments,' in which the constant repetition of the same movements begets an inability to perform that movement—a movement which often constitutes the profession of the sufferer, and is his sole source of income.

Careful examination of these cases has shown me that the breakdown is generally most evident in those muscles which are subjected to prolonged strain rather than in those in which the periods of contraction and relaxation are more equally divided. Thus in writer's cramp it is the muscles of pen-prehension rather than those of pen-movement which are liable to fail. In the comparatively few cases of pianist's cramp which I have seen the extensors of the wrist and the near phalanges have been at fault. In a packing-case maker who could not wield his saw the chief trouble seemed to be in the supra-spinatus muscle, which during the act of sawing is subjected to prolonged strain. 'Dancer's cramp' would probably be unknown if rhythmical movements, such as constitute dancing proper, alone were executed. It occurs only in those who abandon the rhythm which is the very essence of dancing for the performance of an acrobatic *tour de force*.

The following history of our patient is summarised from the very excellent record which has been made by Mr. L. Barnett, house physician.

J. M——, aged 25, nailmaker, admitted February 2, 1886. He is a piece-worker—i.e. the more nails he can make the more money he will earn. He has worked from 7 A.M. to

[1] Clinical Lecture, U.C.H., 1886.

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7 P.M., with about forty minutes' interval for meals. He has worked in a hot place indoors, and has been in the habit of taking three or four pints of beer per diem with occasional spirits. There is nothing in the family history of definite import, but the death of his parents at 54 and 50 and the loss of eleven brothers and sisters (out of thirteen) in infancy make it probable that our patient's 'constitution' is not very robust. His work consists in forging nails with a moderately light hammer. The iron is seized with pincers in the left hand, placed in position, and then forged into shape by a rapid succession of blows dealt by the hammer in the right hand. The onset of his trouble was almost sudden. He states that on Monday, June 8, 1885, after he had been at work for about two hours, he was seized with an inability to direct the blows with his right hand. The trouble increased during the ensuing three hours, when, in consequence of the intense feeling of fatigue, he was obliged to 'knock off work' for the day. On June 9 he managed, by dint of holding the elbow close to the side, to get through his day's work. On June 10, 11, 12, and 13 he worked as well as ever. On the following Monday (June 15) his trouble returned, but not to the same extent, and he got through his week's work fairly well by holding his elbow close to the side. After this the feeling of loss of aim in the arm recurred every Monday, and gradually wore off during the week, but the period it took to wear off got greater each time. By September the constant pressure of the elbow against the side produced soreness, and when attempts were made to keep the arm against the side the limb jerked, so that further work became impossible, and on September 4 he had to leave it off. The patient states that after this everything he did with his right arm was done clumsily. He was then admitted into the Royal Free Hospital under Dr. Harrington Sainsbury, by whose kindness I first was enabled to see him.

Leaving the Royal Free Hospital, he next went to St. Bartholomew's, and from the latter institution he came here. On admission the 'present state' was taken with great care and minuteness by Mr. Barnett. On the chest are a few

small scars, the result of burns from sparks; otherwise the skin is normal. The circulatory, respiratory, digestive, and genito-urinary systems present no abnormalities of any kind. The temperature during his stay in hospital was always normal, but the pulse, when recorded, was found to be rather infrequent, being 62 on one occasion and 56 on another.

As regards the nervous system, it is noted that the intelligence was good, that there was no headache, and that the special senses were normal. The third, fourth, fifth, and sixth cranial nerves were apparently normal. The right nasolabial fold was rather less marked than the left; the mouth dropped a little on the right side when at rest and moved a little less vigorously than the left side. The tongue is slightly tremulous when protruded, and the median furrow deviates very slightly to the right side. The right upper limb, if carefully observed, will be noted to be seldom or never quite still. When sitting up with the arms hanging by the side the right arm is the seat of constant slight movements—elevation, and adduction of the humerus, and flexion of the elbow. When an attempt is made at voluntary movement the spasmodic action of the muscles of the upper limb is increased. The arm is held fixed to the side, the wrist is extended, the hand supinated and closed. The spasmodic action is seen to extend to the deltoid, trapezius, and pectoral muscles. The right sterno-mastoid is also slightly affected. When an attempt is made to use the hammer the spasm reaches its maximum, and affects the right arm and shoulder to an extent which makes analysis impossible. It is evident that some of the muscles attached to the scapula are in an abnormal condition. I have followed my invariable plan and have carefully outlined both scapulæ with a blue pencil. By this means we see that the inferior angle of the right scapula is at a higher level than the left, that the distance between the mid-vertebral line and the base of the scapula is 5 inches on the right side and $4\frac{3}{4}$ inches on the left. The spinal border of the right scapula is somewhat tilted, and stands away from the surface of the back. The mass of the rhomboid muscles is very distinctly larger on the right side than the left. Looking to the lower

axillary region, it is evident that the mass of the serratus magnus muscle is distinctly less evident on the right side than on the left. There is nothing wrong with the lower limbs except that the plantar reflex is more marked on the right side than the left. Sensation is everywhere normal. The muscles were tested electrically. When I first saw this patient at the Royal Free Hospital, with Dr. Sainsbury, the irritability of the right serratus magnus to both forms of current was distinctly diminished. Now, however, the difference in irritability of the two serrati is scarcely noticeable. The patient is free from pain. After attempts at voluntary use of the arm there is a sense of fatigue and some aching, especially beneath the prominent rhomboid muscles on the right side, but beyond this his condition causes no suffering. *None of the nerve-trunks are in the least tender.* It is very evident that the spasm is not now special to the performance of his professional act of hammering. When he is asked to write he picks up the pen with his left hand and then transfers it to the right, and his attempt to sign his name is interrupted by repeated slight shocks of spasm, and the resulting signature bears evidence of the jerking of the hand. No treatment has been of any service to him. He has previously been in two hospitals without benefit. In this hospital he has had electrical treatment, and in addition he took one-sixteenth of a grain of extract of physostigma from February 6th to the 15th, and twenty minims of paraldehyde three times daily from February 15th to the 21st. He is no better.

There are some points about this patient to which I desire to direct your attention, because they seem to me to place his case in a category different from some of the other professional ailments to which I have alluded. First, as to the mode of onset ; it is to be noticed that it was comparatively sudden, and the patient fixes the date absolutely, and he states that before June 8, 1885, he had no trouble at all, but since then he has never worked satisfactorily. In most other 'professional ailments' the onset is gradual, the loss of function increases by scarcely perceptible degrees, and

for a patient to be able to fix the exact day of onset is infinitely rare. I took occasion to tell you in my preliminary remarks that these professional ailments most commonly affect movements which necessitate, as in writing, a prolonged contraction of muscles. Now, in hammering it is difficult to say where (if anywhere) this prolonged contraction occurs. It would be most likely to occur in some of the muscles which steady the shoulder, and the permanent slight displacement of the right scapula in the case we are considering rather favours such an idea, but I am unable to say anything more definite.

The act of hammering is probably less automatic than many other acts of a similar kind. The action of the right hand depends to a certain extent upon that of the left, which places the metal in position for being struck. The action of the left hand in selecting and placing in position the nails to be forged is far from being a mere monotonous repetition of the same act, and hence the position of the nail when receiving the blows from the hammer must be to some extent uncertain. Hence it follows that each blow with the hammer involves the taking aim and the exercise of no inconsiderable amount of judgment. The act is as much mental as it is automatic, and both arms and the eyes have to act in harmony for its accomplishment. It seems to me such an act as would be likely to produce a considerable amount of mental worry, which would be redoubled on the first appearance of any failure of accuracy. The repetitions of the act were very numerous, and during the eleven hours of work very many thousand blows with the hammer must have been delivered. Dr. Frank Smith of Sheffield, who published a very able paper on this subject in the 'British Medical Journal' of October 31, 1874, estimated that a pen-blade forger who suffered similarly to our patient delivered about 28,000 blows per diem, and if we assume that the hammer weighs 3 lb. and is lifted 1 foot for the delivery of each blow, this amounts to 35 foot-tons per diem. If we assume that the work of delivering the blow is equal to that of raising the hammer, and if we add to the weight of

the hammer the weight of the arm, we must allow that the work done is about 150 foot-tons per diem. This is by no means a very excessive day's work.

You will observe that, as I have previously said, the trouble is by no means limited to the act of hammering. It very obviously interferes with the accuracy of all voluntary efforts of the right hand, and, indeed, the spasm is noticeable to some extent when the patient is making no voluntary effort with it. It is, or used to be, stated in text-books that in these cases, beyond the professional trouble, there is nothing detectably wrong, and that all movements, excepting the professional movement, are perfect. I dissent from both these statements ; but, nevertheless, the present case differs from the great majority of professional troubles in the very large amount of peripheral evidence of mischief which is present and the rapidity with which the motor trouble has spread, as it were, from the professional act to the involvement of other acts. Indeed, one cannot escape from the belief that the motor trouble is part of a hemi-plegia, for added to the disorderly movements of the upper limb we have evidence of slight failure of the right side of the face and the right half of the tongue, and probably the exaggerated plantar reflex on the right side tells of diminished cerebral control over the right leg. These trifling signs of hemiplegia, added to the fact of comparatively sudden onset, make it highly probable that the lesion, whatever it may be, would be found in the motor area of the cerebral cortex on the left side. In addition, notwith-standing that I can find no defect of intelligence of any definite kind, I confess that there is something vacuous in the expression of his face which, added to a certain listlessness of manner and utterance, leaves upon me the impression that mentally he is below the average. He further admits that since his trouble he is more easily moved to anger than formerly was the case.

There is now loss of control over a large group of muscles, and the history of the case makes it probable that at the beginning of his trouble the area of diminished control was

smaller than at present, and possibly was limited to one or two muscles, or perhaps a single muscle. That the serratus magnus was the first, or one of the first, muscles to be affected seems to me very likely. It certainly is smaller than its fellow ; it certainly was at first less irritable to both forms of the electric current than its fellow ; it was the only muscle that I could definitely say was not in a healthy condition ; it certainly is brought into play whenever the arm is raised in the act of hammering, and most certainly the plan adopted by the patient of holding the elbow close to his side in the act of hammering would enable him to do without scapular movement, and thus save the muscle over which control had been lost. Again, the slightly faulty position of the right scapula points to some weakness of the serratus magnus. The slight tilting backwards of the vertebral border and the slight elevations of the lower angle are both indications that the serratus is weak, and the large size of the rhomboid muscles on the right side looks as if they had been employed to compensate for the weakness of the serratus and had been used for steadying the scapula. The prominence of the rhomboid muscles, even when the scapula is at rest, coupled with the fact that the vertebral border of the right scapula is $\frac{1}{4}$ inch further from the middle line than its fellow, led me to think that possibly a bursa or abscess had formed beneath the muscles, and at my request Mr. Godlee very kindly inserted an aspirator at the spot where the prominence is most marked, but with no result.

This case is of interest because of its rarity. It is the first of its kind that I have seen, and for me its chief interest lies in the fact that in many points it offers a striking contrast to all the other cases of professional ailments (and they are not few) which I have seen. In every case of professional trouble that I have seen I have been able, as in this one, to detect peripheral evidence of mischief, but this one is exceptional in the fact that the *peripheral* evidence is evidence of *cerebral* mischief, and enables us to conclude that the lesion is in the brain. This is the only case of hammerman's cramp that has come under my notice, and the only record of

similar cases which I can find is in the short paper by Dr. Frank Smith previously alluded to. He was physician to the Sheffield Infirmary, and had seen, naturally, a very large number of hammermen among the patients of the infirmary, and he has placed upon record eight cases analogous to the one we are discussing, and which he called cases of ' hephestic hemiplegia or hammer palsy,' the word ' hephestic,' it is perhaps necessary to state, being derived from the Greek word Ἥφαιστος, Vulcan. Of these eight cases six were distinctly hemiplegic in character, while two were cases of brachial monoplegia. In one there was spasm with wasting of the forearm. In one other case there was spasm. In the other cases the symptoms were paralytic, and in four of the cases there was derangement of tactile sense. The ages of Dr. Frank Smith's patients were thirty-one, forty, thirty-two, eighteen, twenty-four, thirty-five, twenty-nine, and fifty-five. He alludes to the points of similarity between these cases and those of writer's cramp, but says that the disease positively differs from writer's cramp in the fact that the cerebral centres are implicated.

It is the difference from rather than the similarity to cases of writer's cramp which constitutes the interest of the case. Cerebral symptoms in writer's cramp and allied professional ailments are infinitely rare, and when present are very vague and consist at most of some slight dizziness during attempts to exercise the deranged function. In the case we are considering and in Dr. Frank Smith's case cerebral symptoms have been prominent, and, once established, persistent, and in no sense intermittent. Why should this be ?

I would offer the following explanation : Muscular acts differ enormously in the amount of cerebral effort they demand. In some muscular acts we are unconscious of any cerebral effort. This is notably the case in walking, which appears to be almost a purely reflex act, and is often accomplished unconsciously. The woman who walks about gossiping and knitting will do an extraordinary amount of work without apparently giving a thought to her knitting-needles.

Even writing—i.e. quill-driving and copying—may be done almost automatically, and so, indeed, may any act in which the tactile sense is largely employed to guide the limb in its movement. An incident which once occurred to myself will serve to show how very automatic a delicate act may become. Some years ago I got a small flake of iron rust into my eye while railway travelling. This caused slight annoyance at the time, but in a day or so I began to suffer from catarrh of the eye, which was excessively painful at times, causing a spasmodic flood of tears and the forcible closing of both eyes. It was in the days when I used a razor, and one morning I was shaving my chin before a looking-glass when suddenly the eyes filled with tears and I closed them ; but, nevertheless, I unconsciously finished my shaving, and shaved my chin quite smooth with my eyes shut. There must have been very little cerebration in this act ; in fact, had I appreciated what I was doing I should inevitably have cut myself. My shaving on this morning must have been almost a pure reflex, brought about by constant daily repetition of the same act.

Other acts, again, can never become automatic, and this is notably the case when the movement of the limb is not guided by tactile sense in the performance of the special act. When, for example, did anyone ever thread a needle automatically or unconsciously ? During such an act as this there is evidence of strong cerebral effort, as is shown in the face of the sempstress. The taking aim—whether it be at a needle's eye, a bull's eye, or the bit of iron to be forged into a nail—involves the bending of the mind towards the object aimed at ; and the aiming at an object against time, as occurs to the pieceworker forging nails, is an act, one would think, which is calculated to cause brain-fag rather than muscle-fag. And the limited number of clinical facts at our disposal seem to point to the correctness of this assumption. When the work done by the muscle is great, and the cerebral effort involved is small, we may well expect the muscle, rather than the brain, to show signs of failure. When, however, the cerebral effort is out of proportion to

the mechanical work, as in the repeated act of aiming with a hammer, it is not surprising that, if breakdown occurs, it should occur in the brain. It might be urged that this is not a 'professional' trouble—that it is merely a hemiplegic attack followed by post-hemiplegic spasm occurring accident-ally in a nail forger. Against this supposition we have to put the fact that his first sign of trouble was in connection with the professional act ; that the spasm of the arm is only seen at its maximum when he attempts to perform the professional act ; and that the case does not stand alone, two of Dr. Frank Smith's cases having been very similar in character.

In the average case of writer's cramp I have always con-tended that, although peripheral evidence of mischief is always present, the evidence does not enable us to say that the lesion is central, although, of course, I very well know that brain cell, nerve fibre, and muscle are practically one. Ordinarily professional ailments never spread to other func-tions, and certainly the man with writer's cramp is not more likely to become hemiplegic than anyone else. In the case we are considering the trouble, from being merely pro-fessional to begin with, quickly spread. This in itself is evidence that in one case the lesion is peripheral and in the other case central, for at the centre, where the nerve fibres converge almost to a point, any *sudden* lesion is not likely to be limited to the cells connected with one small function only. The nervous system may be compared to a railway with branch lines and a 'central' terminus. Now, a fire at Holyhead would not cause any disturbance at Carlisle, not-withstanding that they are both stations on one system of railway, but a fire at Euston would certainly derange the functions of both these outlying stations, and of many others.

The lesion in our case probably began in the part of the centre corresponding to the serratus magnus muscle (for as each limb has its controlling 'centre' in the brain, so, we must assume, has each muscle of each limb), and thence quickly spread until the right upper limb, the right side of

the face, the right half of the tongue and the right leg (?) became involved.

Seeing that the leg is very slightly and doubtfully involved, the lesion, whatever it may be, is apparently almost limited to the left ascending frontal convolution, to an area to which the blood is brought by the ascending frontal branch of the Sylvian artery. Seeing that the onset of the trouble was comparatively sudden, it is not probable that the change which has taken place in the brain is of the nature of a slow degeneration or sclerosis. It is to the vessels of the part to which we must look for an explanation, to hæmorrhage in the area of the ascending frontal artery or embolism or thrombosis of it. There are manifest difficulties in accepting the theory of hæmorrhage or embolism, and against the latter supposition is the absence of any source of emboli. On the whole, I adhere to the belief that the physical basis of our patient's trouble will be found in thrombosis of the ascending frontal and parietal branches of the Sylvian artery. In favour of this theory is the fact that his trouble at its commencement was always worse on Monday morning, when the overworked area of his brain had had a period of rest, and when the circulation through it had been, presumably, slow. If we imagine a clot forming in one of the smallest branches and gradually spreading backwards, so as to involve the larger branches of the ascending frontal branch of the Sylvian branch of the middle cerebral artery, we have conditions which, automatically and pathologically, would account for the clinical facts. Thrombosis on the side of the veins leading from this area might conceivably lead to a similar condition, and cause such derangement of nutrition as to prevent the proper control of movement and lead to such a state of things as we are confronted with.

The prognosis in this case is, I fear, not good. Between September, 1885, and February, 1886, his progress has been *nil*, in spite of complete rest, good nourishment, and the therapeutic skill in at least three of our metropolitan hospitals. The deficient irritability of the serratus magnus has disappeared, but even this has not caused more benefit than a

similar recovery of normal irritability causes in ordinary hemiplegia. With the exception of this diminished irritability, there have been no indications for treatment in the affected limb. No tender nerves have seemed to demand a blister (which is often of very great use in cases of professional trouble), and no massage or shampooing of the limb would be likely to influence the brain. Rest, if anything, should lead to recovery or improvement ; but six months' rest has done nothing, and I very much fear that his trouble is likely to be permanent, and that his loss of control over the affected group of muscles will continue.

CHAPTER V

ON CERTAIN CONDITIONS OF THE HAND AND ARM WHICH INTERFERE WITH PIANO-PLAYING [1]

GENTLEMEN,—I wish to-day to direct your attention to a group of patients who have sought advice because of some difficulty which they have experienced in playing the piano, a condition which has in many instances been apparently brought about by the over-exercise of their professional functions as pianists. We might speak of this condition (as indeed it has been spoken of) as 'pianist's cramp,' but I hesitate to do so because I think such a word would be misleading, and might tend to encourage the belief that a failure in piano-playing is something more than a symptom, and might tend to make you believe that you had made a diagnosis when, in fact, you had only used a word. You will find that a failure in playing the piano may depend upon a variety of causes, a fact which I believe to be true in the great majority of professional ailments. Your reason, backed by your knowledge of anatomy and physiology, will tell you that this must be so. The machinery involved in the delicate manipulations necessary for a pianist is, indeed, so complicated that the wonder is breakdown does not oftener occur.[2]

[1] Clinical Lecture, U.C.H., 1886.

[2] Sir James Paget, in a public address some years ago, stated that: 'He remembered once hearing Mdlle. Janotha play a presto by Mendelssohn, and he counted the notes and the time occupied. She played 5,595 notes in 4 mins. 3 secs. It seemed startling, but let them look at it in the fair amount of its wonder. Every one of those notes involved certain movements of a finger, at least two, and many of them involved an additional movement laterally as well as those up and down. They also involved repeated movements of the wrists, elbows, and arms, altogether probably not less than one movement for each note. Therefore

In playing the piano all forms of sensation (cutaneous, articular, muscular) must be perfect; from peripheral nerve-endings to brain the sensory path must be free from all defects. The motor path must be equally free from defect from the cerebral cortex to the motor end plates on the muscles, and it must be borne in mind that a trouble affecting a fine nerve-twig supplying some little muscle of the fingers is as capable of upsetting the harmony of a delicate act like piano-playing as is a damage to one of the chief cords of the brachial plexus. The muscles involved in the act must one and all be healthy. We do not know much of the minor pathological changes which affect the muscles. Fatty and fibroid change may occur, and we know that a muscle may be painful, or tender, or stiff, as the result of cold, gout, rheumatism, or fatigue, and we speak of such conditions as 'muscular rheumatism,' or myalgia, without having anything very solid behind the expression. Not only must the fibres of the muscles be healthy, but the tendons and their sheaths also; too much or too little of the fluid which lubricates the tendon in its sheath, or the deposit of a small amount of urate of soda in the sheath, must necessarily interfere greatly with the performance of a delicate act.

there were three distinct movements for each note. As there were 24 notes per second, and each of those notes involved three distinct musical movements, that amounted to 72 movements in each second. Moreover, each of those notes was determined by the will to a chosen place, with a certain force, at a certain time, and with a certain duration. Therefore there were four distinct qualities in each of the 72 movements in each second. Such were the transmissions outwards. And all those were conditional on consciousness of the position of each hand and each finger before it was moved, and, while moving it, the sound of each note and the force of each touch. Therefore there were three conscious sensations for every note. There were 72 transmissions per second, 144 to and fro, and those with constant change of quality. Let them imagine it in telegraph wires. And then, added to that, all the time the memory was remembering each note in its due time and place, and was exercised in the comparison of it with others that came before. So that it would be fair to say that there were not less than 200 transmissions of nerve force to and from the brain outwards and inwards every second, and during the whole of that time judgment was being exercised as to whether the music was being played worse or better than before, and the mind was conscious of some of the emotions which the music was intended to impress.'

I would direct your attention to the great number and very wide extent of the muscles used in piano-playing, and also to the fact that for the performance of delicate acts we need steadiness quite as much as agility. For piano-playing the scapula must be steady, the shoulder must be steady, the elbow must be steady, and the wrist must be steady in order that the fingers may act with certainty and precision on the keys. When I use the word 'steady,' I mean, of course, steady in movement, and not necessarily fixed. In examining such cases you must therefore bear in mind that the shoulder may demand examination equally with the hand and fingers.

It is needless to say that a sound condition of all the articulations of the upper limb is necessary for piano-playing.

The frequency of piano-failure is not very great. I find I have seen only twenty-one cases in a period extending over many years. In the same time, I have seen about eight times as many cases of writing-failure. The comparative infrequency of piano-failure is due to the fact that the number of persons who are obliged to play the piano excessively is infinitely smaller than of those who write excessively. The class of clerks is decidedly larger than the class of pianists. The pianists belong mainly to the female sex, and hence it is not surprising that out of my twenty-one patients all but two were females. There is another and more important reason for the comparative rarity of piano-failure as compared with writing-failure—namely, that in piano-playing there is no prolonged strain upon the muscles, at least, to the same degree, as there is in writing. I have again and again pointed out that in writing it is in the muscles of pen-prehension rather than in muscles of pen-movement that failure is liable to occur, and that in all of these 'professional' ailments breakdown most often takes place, or, at least, is most apparent in muscles which are maintained in a state of contraction for long periods.

In examining the cases of professional failure, we must always try to discover which muscles, if any, are likely to be most subject to fatigue in performing the professional act, and

we must be very careful to investigate the condition of these muscles.

Now, in piano-playing, it is, at first sight, very difficult to say which muscles, if any, are subjected to prolonged strain. The very essence of piano-playing, as of all musical perform-ances, is rhythm, and rhythm in movement means that in the muscles producing the movement, periods of contraction alternate with periods of relaxation with regular intervals, and therefore breakdown from fatigue is scarcely more liable to occur than in the heart itself, the untiring prototype of rhythmical movement. Piano-playing does not, however, consist entirely of rhythmical movement, as might appear to be the case. Professors are very exacting with their pupils as to the position of the hand. There is a way of holding the hand in piano-playing which, I am told, is called the 'Stuttgart method,' in which the extension of the wrist is most rigidly maintained during the whole performance, and, except during the instant when the finger is depressed on the key, the extension of the near phalanges of the fingers is also maintained. In this method of holding the hand there is considerable strain thrown upon the extensors of the wrist and the extensors of the near phalanges (extensor communis digitorum, extensor indicis, extensor minimi digiti). In order to be sure that this orthodox manner of holding the hand is maintained, I have known young ladies practise their scales with a coin balanced on the back of the hand, the falling off of the coin showing at once that the absolute level of the carpus and metacarpus has not been maintained. It is obvious that this position of the hand has the effect of keeping the finger-tips constantly as near their work as possible, and that it must conduce to great rapidity of finger-ing. If the 'Stuttgart method' were not of practical value, one may conclude that it would not be insisted on. Certain it is, however, that a large proportion of my patients have had symptoms referable to the extensor muscles of the wrist and fingers and the musculo-spiral nerve. I will briefly give you the facts of a case which will serve as an illustration.

Case 1.—Miss R——, aged 24, consulted me in November

1875. She had been working at the School of Music in Berlin, and had practised as much as eight hours a day, besides using a digitorium for the exercise of the fingers. She had found the Stuttgart position very trying. Her trouble was in the left hand, and was mainly experienced in playing staccato passages, necessitating a sudden extension of the wrist and fingers ; and further, there was a tendency, during the exercise of runs and scales, for all the fingers of the left hand to 'run together,' as she said, owing apparently to a failure of the extensor communis digitorum steadily to maintain the extension of them. There was some tenderness of the left ulnar nerve at the elbow, and excessive irritability (to faradism) of the interossei muscles. There was, further, this peculiarity that, when the first left dorsal interosseous muscle was faradised, all the left interossei muscles responded to the current. This phenomenon was not observed in the right hand, and disappeared in the left hand when she recovered, as she eventually did. This peculiarity was due, probably, partly to the irritable condition of the left ulnar nerve and interossei, and partly to the weakness of the extensor communis digitorum, which in part antagonises the interossei muscle. It was very interesting to see that faradisation of one interosseous produced, as it were, the very trouble of the 'running together of the fingers,' which was one of her chief difficulties when playing. Although the disability of the left hand was most marked in piano-playing, it was not limited to this act. The limb was generally inefficient in the performance of all acts, and readily got fatigued. It often 'died away,' and was sometimes burning hot, and at other times was blue, cold, and mottled. She was worse in cold weather, and her trouble was intensified by emotional excitement, such as was caused by playing before an audience or her music-master. There was well marked tremor of the left hand. This patient was in depressed health, due to overwork ; and also, as she said, to the fact that the diet and mode of life in Berlin had not agreed with her. She was treated by rest, tonics, a blister over the tender ulnar nerve, and friction for the arm. In February, 1877, she called upon me to say she had recovered.

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Case 2 was similar in many respects. Miss L., aged 33, a teacher of music, had been practising the 'Stuttgart method.' Piano-playing had become painful and impossible. All the nerve-trunks of the left arm were very tender. She suffered from great discomfort in the arms at night, an uneasy feeling, and a difficulty in getting them into a comfortable position in bed. Her hands were crippled for all acts, and she was worse in cold weather. She was worried and sleepless. With rest, tonics, and blisters to the painful nerve-trunks, she recovered.

Case 3.—Miss F., a music teacher, aged 33. Had been practising the 'Stuttgart method.' Marked tenderness of the right musculo-spiral nerve and over the extensors of wrist and fingers. She was anæmic, and attributed her trouble to catching cold.

Case 4.—Miss O., aged 20, in delicate health, had 'strained her arm playing the piano.' There was pain, tremor, tenderness of the right musculo-spiral nerve, and excess of irritability to faradism of the extensor muscles. There was discomfort in the arms at night, as in Case 2. After rest, tonics, and blisters to the tender nerves, she completely recovered.

Case 5.—Miss W., aged 20. Marked tenderness of the median and musculo-spiral nerves and tremor of the hand. She was in ill-health and dyspeptic.

Case 6.—Miss G., aged 19, a pupil at the Royal College of Music ; and

Case 7.—Miss M., aged 35, were in a condition almost exactly similar to the preceding, so that I need not trouble you with further details.

Case 8.—Miss Y., a governess, aged 22. Her difficulty was mainly with the left hand. Extension of the wrist and fingers caused her pain, and she had marked tenderness of the median and ulnar nerves. Tremors of both hands, highly nervous, and a history of family trouble and loss of rest.

Case 9.—Miss F. complained that 'the left forefinger would not keep off the keys.' There was marked deficiency of irritability of the left extensor indicis and extensor primi

internodii pollicis. No nerve-tenderness. The patient attributed her trouble to a whitlow which she had had on the left forefinger two years previously. She was nervous and sleepless.

These nine cases are of interest as showing a distinct involvement of the extensor muscles or the musculo-spiral nerve in each case. They also serve to illustrate other points which many of these cases of piano-difficulty have in common.

First, there is the nerve-tenderness which accompanies almost all of these cases. This may definitely be brought about by over-use of the muscles; it may also occur in connection with a wrench or strain of the arm, or may follow a bruise (as we shall see in connection with a case to be presently recited), and it seems very liable to occur in persons of delicate organisation who are in depressed health or who have been exposed to cold. We have no right to assume that it, in all cases, indicates neuritis, but it may do so; or it may point to hyperæmia or congestion of the nerve. There are four conditions which may accompany nerve-tenderness; these are (*a*) muscular disability which, although it may be specially manifest in one particular act, will generally be found to affect other acts as well; (*b*) more or less tremor; (*c*) pain or early fatigue during muscular exertion; (*d*) a sense of discomfort in the limb (not exactly pain), which is always worse at night and makes it difficult to put the limb in a comfortable position in bed and often keeps the patient awake. This latter symptom is both interesting and curious, and one which is often complained of. This sense of discomfort is an effect, probably, of the tenderness of the nerves, whereby they become sensitive to the slight degrees of pressure which are inseparable from almost every position. Not only, however, are the nerves sensitive to pressure, but also to slight degrees of stretching. This fact was well illustrated by a case of so-called multiple neuritis due to alcohol which was in Ward 3 last summer. This patient's nerve-trunks were all tender (median, ulnar, and musculo-spiral), and I had frequent opportunities of showing you that when either of these nerve-

trunks were put upon the stretch, the patient (who was habitually drowsy and in a state of partial mental incapacity) invariably manifested pain, and when asked where the pain was, invariably pointed to the position of the trunk of the nerve which was being stretched. Thus, with the elbow bent and the wrist forcibly extended, if the ring and little fingers were pulled back so as to stretch the ulnar nerve to the utmost, the patient pointed to the back of the olecranon as the seat of the pain. If, with the elbow straight and the wrist extended, the thumb and forefinger were pulled back so as to stretch the median nerve, the patient pointed to a point internal to the biceps tendon at the bend of the elbow as the seat of pain. If, with the elbow straight and the wrist forcibly flexed, the thumb and forefinger were bent strongly into the palm so as to stretch the musculo-spiral nerve, the patient complained of pain in the position of the trunk of the nerve above the outer condyle of the humerus. We tried these experiments again and again, and always with the same result. If, then, the nerves are sensitive both to pressure and tension, it is not surprising that trouble should be experienced in finding a comfortable position for the affected limb.

Another fact worthy of notice is the common implication of the left hand, either alone or to a greater extent than the right. The left hand is less expert and more difficult to train than the right, and it is not to be wondered at that it should be the most prone to suffer from overwork in acts which call upon both hands equally or nearly so. Because of the difficulties experienced with the left hand, it is often inordinately worked while practising exercises.

The next four cases are similar to those already given, except in the fact that the extensors and the musculo-spiral nerve were not specially involved.

Case 10.—Miss G., aged 20, experienced pain and difficulty in playing, and to a less extent in all acts with both hands. All the nerve-trunks were tender. She was dyspeptic, and there was a strong family history of gout. Cured by rest, diet, and change of air.

Case 11.—Mrs. A., aged 34, complained of pain and 'creepy

feelings' after playing the piano. There was a distinct tenderness of the right median nerve, and the cervico-dorsal spine was also tender.

Case 12.—Miss A. B. experienced difficulty in lateral (inter-osseal) finger movements, and to a less extent in all acts, both coarse and fine. It was found that the median and ulnar nerves on both sides were distinctly tender.

Case 13.—Miss B., aged 21, had been working hard at the Leipsic Conservatorium. The right median nerve was exquisitely tender. The patient was anæmic and dyspeptic. Completely cured by rest, iron, blistering the tender nerve, and massage.

The next two cases are of interest as examples of difficulty caused by trouble apparently limited to the region of the shoulders.

Case 14.—Miss E. B., aged 27, a teacher of music, suffered from pain in the shoulder after playing—or, indeed, after any work with the right hand. There were no tender nerve-trunks, but a very tender spot was found close to the insertion of the levator anguli scapulæ, at the superior angle of the right scapula. She improved after blistering the tender spot and the administration of arsenic.

Case 15.—Miss R., aged 29, a governess, came complaining of an ill-defined awkwardness of the right arm, which inter-fered not only with piano-playing, but with other acts, such as cutting her dinner. The elbow, she said, had a tendency to stick out. There was no nerve-tenderness, and no well-marked change in electric irritability. On removing the clothing and faradising the right deltoid, it was noticed that the vertebral border of the right scapula was tilted away from the thorax, whereas nothing of the kind occurred when the left was faradised. This was due, apparently, to a relatively weak condition of the right serratus magnus. There was a disturbance of the normal balance of power between the right deltoid and right serratus, which became manifest to the eye, not during forced voluntary acts so much as during the forcible faradisation of the deltoid. When the right scapula was tightly bound or held against the ribs, the clumsiness of

the right hand was much lessened. This weakness of the serratus was of long standing ; its cause was not evident, and, when last seen, the condition had not materially improved.

The next two cases were, in a mild sense, traumatic.

Case 16.—Miss E. E., aged 23, complained of a trouble with her left hand when playing. The metacarpo-phalangeal joint of the left thumb was found to be weak and tender, and on being questioned, she remembered that she had strained the joint a few weeks previously, while struggling with her brother.

Case 17.—Miss F. B., aged 31, bruised the right arm eighteen months ago, and since then she has had pain and disability of the limb. All the nerve trunks were tender, and she was ordered to counter-irritate the skin over the tender nerves, and rest the limb.

The next case I have to quote is one of considerable interest, in which the primary trouble seems to have been in a tendon.

Case 18.—Miss Ada H., aged 21, pianist, complained of weakness in the right hand while playing, and in all acts. There was special difficulty with lateral (interosseal) finger movements, and in turning the hand and little finger towards the ulnar side, and she further complained of a quivering in the first right dorsal interosseous muscle after playing. There was found a considerable thickening on the tendon of the flexor carpi ulnaris muscle, which not only interfered with the use of that muscle, but also, by pressing on the ulnar nerve, seriously hampered the use of the muscles in the palm supplied by that nerve. The thickened tendon was blistered, and the patient was ordered to take a tonic and go for a holiday. She made a good recovery.

Case 19.—Miss L. H., a pianist, whose performances were hindered by a troublesome cramp of the flexor minimi digiti. The cause of this cramp was not very evident ; but she was dyspeptic, and this fact, combined with excessive use of the muscle, may be regarded, perhaps, as sufficient to account for the cramp.

I have only seen two males who have come to me complaining of difficulty in piano-playing.

Case 20.—Mr. P., aged 60, a professional pianist of intemperate habits. He had all the symptoms of senile tremor and degeneration. His eyesight was failing, and there was grey atrophy of the optic discs. He went gradually from bad to worse. Tremor was most marked in the left arm, which was very tender, so that he could hardly bear to have it touched. The musculo-spiral and ulnar nerves of this arm were especially tender. He said that he could not strike the keys with any certainty, and he complained that the left ring-finger always gave him the most trouble. The left ring-finger is the finger which pianists always find the most difficult to educate. This case is of interest as showing how a well-recognised pathological condition may first show itself in parts which are especially overworked.

Case 21 was that of a professional pianist, aged 23, who for the last few months had experienced an ill-defined difficulty in playing in both hands. The right little finger and the back of the left hand were particularly painful. There was slight rheumatic thickening of some of the small joints of the fingers. Each nail of both hands had a white mark above the middle. These must have been formed at about the same time. The patient stated that, about three months previously, he had kept the house for a feverish cold. This may have entailed some failure of nutrition, and it was subsequent to this that he experienced his difficulty in performing. I could make out nothing definitely wrong with nerves or muscles, and I have no knowledge of the subsequent course of this case.

The short summary of these twenty-one cases shows us that in no two cases was the cause of breakdown precisely the same. These cases afford no justification for using such a term as 'pianist's cramp,' to imply a morbid entity having a special cause, and a pathology of its own. Confronted with a pianist who has broken down, our first duty is to make a diagnosis, and answer the questions, Where ? and Why ? The most common symptom in these cases was nerve-tenderness (indicative possibly of congestion or mild inflammation), which affected different nerves, and seemed to depend, not only on overwork, but on such accidental causes as strain or cold, and

to be predisposed to by various bodily conditions, such as anæmia, rheumatism, gout, and dyspepsia.

You will have noticed that, although the act of piano-playing was most seriously affected, there was usually some general disability of the limb as well, which accompanied the 'professional' trouble in almost every case. You are aware that, with regard to 'writer's cramp,' it is often asserted that, with the exception of the act of writing, there is nothing wrong. Although I cannot quite endorse this statement with regard to 'writer's cramp,' still I must admit that these cases of pianists' trouble offer a contrast to 'writer's cramp' in the large amount of disability which generally existed for acts other than piano-playing. This is due, I believe, to the fact that the muscles, the derangement of which causes breakdown in piano-playing, are larger, more numerous, and in more common requisition for general purposes than are the muscles (most commonly the interossei) whose breakdown causes writer's cramp.

The prognosis necessarily depends upon the diagnosis. In cases where nerve-tenderness and local muscular weakness are the only troubles, I believe the prognosis to be good. Cases of this kind occurring, as very often is the case, in young girls, nearly always recover, although I am bound to say that recovery is sometimes very tedious.

Treatment.—The most important point in treatment is rest. The excessive use of the hand must be discontinued, and it is often necessary to insist upon this rather forcibly. Piano-playing, if not prohibited altogether, must only be practised to a degree short of that which causes pain or annoyance. It is often difficult to restrain the ardour of these patients in the matter of playing. Directly they feel in a small degree better, they fly to the piano ; and I have known the progress of more than one case very seriously retarded by the undoing, as it were, of the good effect of rest by an hour's injudicious and prohibited 'practising.'

I need hardly say that constitutional treatment is all-important. The body must be healthy in order that the arm may recover. Anæmia and dyspepsia must be attended to

and counteracted by diet, medicines, and change ; and no change is better than to a mountainous climate, where the advantages of a chalybeate water may be enjoyed. St. Moritz in the Engadine, Rippoldsau in the Black Forest, Schwalbach, and Spa have a well-deserved reputation for benefiting these cases. Any indications caused by gout and rheumatism must be met, and if there be evidence of gastric irritation, you may be sure that the arm will not be well until this trouble has been cured.

In many cases the administration of arsenic has seemed to be of decided benefit. The most important local measure is the employment of counter-irritation over the tender nerve-trunks. This may be accomplished by means of blisters or by the application of strips of capsicum plaister. Douching, friction to the arm, and systematic massage are often of great use, but the last-named mode of treatment is not to be adopted as long as the nerve-trunks are tender, for under these circumstances it seems decidedly to do harm rather than good. You must remember that the professional *masseur* is not infrequently over-zealous, and that the mere passive act of being rubbed is very exhausting to the patient. You must take care that the massage is neither too long nor too vigorous. If massage be well timed, and be reasonably and sensibly carried out, it is undoubtedly of great value. It seems necessary, in view of the sort of fashion which has set in towards this mode of treatment, to warn you not to recommend a patient to place himself in the hands of a 'professional rubber' unless you know something of his or her character and capabilities. While on the subject of treatment I may allude again to the fact that pianists have special difficulty in educating the ring-fingers, which have less power of independent movement than the other fingers. This is partly due to the absence of special extensor muscles, such as are provided for the index and little fingers, and partly to the fact that the extensor tendon of the ring-finger is joined to its fellows on either side by a fibrous band, near the far end of the metacarpal bone. With the hand in the pianists' position it will be found that the power of raising the ring-finger while

the middle and little fingers are resting on the keys is not great, less than that possessed by either of the other fingers when similarly circumstanced. It cannot be raised more than about three-quarters of an inch, and hence the power of striking the key is small. To remedy this it has been proposed to divide, by means of a tenotome, the bands connecting the extensor tendon of the ring-finger with the extensor tendons on either side of it. This operation has been performed in America and in this country, and it is said with good results. How the reunion of the divided band is prevented I do not know. I confess that the operation seems to me to be one likely to produce weakness at first, and to be followed by greater fixity in the end, when the cicatricial tissue of the divided bands contracts and draws them tighter than before. It must not be forgotten that the ring-finger has its special use, which appears to me to be to grasp firmly in the palm, while the other fingers of the hand are otherwise employed. The great characteristic of the ring-finger is its power of firm grasp, and we should not rashly do anything which is likely to lessen this firmness.

CHAPTER VI

ON A CASE OF 'SAWYER'S CRAMP' [1]

THE patient whose case is recorded in the following short paper was sent to me by my friend Dr. de Watteville, who was kind enough to remember that I took an interest in 'professional' ailments.

W. W., æt. 38, came as an out-patient to University College Hospital on March 8, 1883. He was a maker of packing-cases and had been engaged in piece-work, making his packing-cases against time, as it were, for 10 or 11 hours a day, and occasionally for as much as 14 hours. He was a strongly-built man, but looked a little worn about the face, and had a certain earnestness of manner which I have often seen associated with genuine professional ailments. He did not drink, and there were no facts in his personal or family history which need to be recorded. His only trouble was an inability to use the saw. As soon as he took his saw in his hand, his efforts to use it were thwarted by disorderly movements which he could not explain.

In order to better understand the case, I asked him on the next occasion to bring his saw with him to the hospital, together with a piece of wood such as he habitually used in his trade. This was done, and the patient was stripped to the waist, and was made to go through the act of sawing.

On taking the saw in his hand, there was noticed a slight tremor of the blade, due to a little shake of the hand. The saw was thrust forward through the wood fairly well, but the back stroke could not be accomplished, and the attempt was accompanied by abnormal elevation of the shoulder, scapular

[1] From 'Brain,' 1883.

movement, adduction of the elbow, rotation outwards of the forearm, flexion of the wrist, and swaying to and fro of the body.   These movements were difficult to analyse, the general impression left upon the observer being that the whole of the muscles of the right upper limb behaved tumultuously when the patient attempted to saw.

With the left arm he could saw perfectly well.

From the character of the movements I thought it not unlikely that some of the scapular muscles were at fault, and my first step in the examination was to outline both scapulæ with a blue pencil, to see if there was any want of symmetry in the position of the scapulæ on the two sides.   This examination gave a negative result.

On close inspection, there was seen to be distinct flattening of the right supra-spinous fossa as compared with the left. That this was not due to any wasting of the trapezius was shown by putting the trapezii in action, when the muscles on the two sides appeared to be equally developed.   The first positive fact with regard to this patient then was—

(*a.*) Diminutions in size of the right supra-spinatus muscle.

On close inspection of the chest, there was noticed a slight degree of flattening of the right infra-clavicular region, and I came to the conclusion that there was—

(*b.*) Diminution in size of the right pectoralis major, especially the clavicular portion.

The muscular wasting in the above situations, though very slight, was undoubted, and when pointed out was readily admitted by all who saw the patient.

I was almost inclined to think that there was a little want of plumpness about the right infra-spinous fossa, and that the muscular mass between the spinal border of the scapula and the spine was more flabby on the right than the left side ; but of these two facts I could not feel at all sure.   No other muscles except those in the situations named were in the least degree wasted.

The next step in the examination was to determine if any of the nerves of the brachial plexus were tender.   There was no tenderness above the clavicle, nor over the situations of

the great nerve-trunks (musculo-spiral, median, or ulnar) in the upper limb.

(*c.*) There was a markedly tender spot in the right pectoral region, in the second interspace, midway between the mid-sternal line and the point of the shoulder.

(*d.*) An equally tender spot beneath the spine of the right scapula, just where the supra-scapular nerve enters the infra-spinous fossa of the scapula.

The next step was to test the irritability of the muscles to faradism. It was found that the muscles of both upper limbs manifested equal degrees of irritability, with one exception.

(*e.*) Both portions of the right pectoralis major (clavicular and sternal) showed a marked degree of excessive irritability, and contracted readily to a current which produced no effect on the left side. The right muscle was far more sensitive to the current, and manifested what Duchenne would have called excessive amount of electro-musculo sensibility.

It must be remembered that the position of the supra-spinatus (which was wasted) prevented its being accurately tested, since the movement of the trapezius effectually masked any movement of the muscle beneath it.

The patient stated that he could perform every movement and every act with the right hand with perfect readiness, with the exception of the act of sawing, which, unfortunately, was the one upon which his livelihood largely depended.

Upon examining the individual movements of the limb with great care, it was found that the approximation of the scapula to the mid-vertebral line (by means of the rhomboids) was not so readily accomplished on the right side as on the left ; and that when the latissimus dorsi was put into extreme action, he complained of pains in the pectoral region. I am inclined to think that the difficulty of the rhomboidal movement, and the pain during the action of the latissimus dorsi, were both in part attributable to the stretching of the pectoralis major which these movements caused.

Questioned as to the origin of his trouble, he stated that it began in March, 1882, and that the first symptom was a pain in the pectoral region during the back stroke of the saw

(during which movement the pectoralis major was certainly put on the stretch).

It will be observed that the case lends no support to the statement which is generally found in text-books, that in professional ailments of this kind there is nothing the matter except the derangement of a special co-ordinated movement.

In this patient distinct evidence was obtained of derangement of special muscles and special nerves, but this derangement was not of a kind to attract the attention of a careless observer. On the contrary, it was only discovered after a very diligent search.

This case, again, lends no support to the theory that ailments of this kind are due to the failure of some co-ordinating centre.

Two muscles were found slightly wasted (the supra-spinatus and the pectoralis major), and the nerves supplying them were distinctly tender ; at least two sharply defined tender spots were found over the anterior thoracic and the supra-scapular nerves.

It will be at once conceded that an irritable condition of the pectoralis major would be likely to seriously interfere with the act of sawing.

The connection of the supra-spinatus with the act of sawing is, however, not at first apparent.

In my papers on ' writer's cramp ' I have insisted on the fact that the muscles which are subjected to prolonged strain are more likely to be affected than those in which contraction and relaxation quickly alternate, and accordingly we find that the muscles of pen-prehension more often suffer from over-use that the muscles of pen-movement.

In the act of sawing, what muscles, if any, are subjected to prolonged strain ? Not the muscles of the hand, for the saw is alternately thrust and pulled, and during the former act the grasp is released. Not the muscles which move the elbow, for in this joint flexion and extension alternate with each other during the act of sawing. Not the muscles which pull the humerus backwards and forwards (among which are the two halves of the deltoid), because when the back-pulling

muscles are contracted the forward-pulling muscles must be relaxed, and *vice versâ*. Clearly the muscles which are most likely to suffer from prolonged strain in this act are those which maintain the head of the humerus securely in the glenoid cavity, for if the head of the humerus were not firmly held during the powerful swaying to and fro of the bone, the act would certainly become unsteady. For sawing, also, the humerus must be held slightly removed from the trunk. Now, the maintenance of the head of the bone in the glenoid cavity, and the slight separation of the humerus from the trunk, which are essential for the act of sawing, both demand the contraction of the supra-spinatus.

Duchenne (*Physiologie des Mouvements*, p. 73 *et seq.*), when speaking of the actions of the supra-spinatus muscle, says that it (1) moves the humerus away from the trunk and slightly forward, and at the same time rotates it inwards very slightly, and (2) it depresses the external angle of the scapula. It usually co-operates with the deltoid and serratus magnus in raising the arm, and in the absence of the former muscle the supra-spinatus and serratus magnus combined can raise the arm very considerably. He further says, 'The supra-spinatus does not merely help the deltoid in its action by its slight power of raising the arm, but its assistance is needed for maintaining the head of the humerus in the glenoid cavity during the raising of the arm. I have noticed, indeed, in my experiments, that when the deltoid contracts by itself, the head of the humerus has a tendency to escape from the glenoid cavity and to be dislocated downwards.' When summing up the actions of the supra-spinatus, Duchenne speaks of it as an 'active ligament of the scapulo-humeral joint.' It is easy to understand how necessary the supra-spinatus must be for the act of sawing, not only in holding the head of the humerus firmly in the glenoid cavity, but also in holding the humerus slightly removed from the side.

It may be noted that the clavicular portions of the pectoralis major, which was wasted in this case, and amongst the fibres of which there was a tender spot, is also indispensable for the act of sawing, not only by helping in the to and fro

movement of the humerus, but also in holding the head of the humerus in the glenoid cavity.

In this case of 'sawyer's cramp,' then, there were found two muscles wasted, both of which were indispensable for sawing and both of which were presumably subjected to *prolonged contraction* during the act.

What was the nature of the pathological changes ? and what was their order in time of occurrence ? Were the nerves affected before the muscles, or *vice versâ* ? Was the nervo-muscular condition merely the expression of central (spinal cord) change ? These questions remain to be answered. The peripheral changes were certain.

CHAPTER VII [1]

THREE CASES OF SPINAL MYELITIS

GENTLEMEN,—The only place to study clinical medicine is in the ward ; and those of you who wish to make sure of passing your examination must recognise that medicine is not to be learnt from books alone, or from lectures alone. Cases of disease which you have *seen or tried to understand* will never be entirely eradicated from the mind. They constitute the real foundations of medical knowledge, and I think it is not too much to say that every case which you have really studied will help you in greater or less degree to understand every other case which you will see in the future ; and I am quite sure that you will assimilate the knowledge conveyed in your text-books with far greater ease in proportion as your experience enables you to conjure up mental pictures of actual cases to illustrate the text.

Nevertheless, books and lectures form a very necessary adjunct to clinical observation. In the lecture-room we are able to talk with more freedom than is always possible in the presence of the patient, and I have asked you to meet me to-day in order that we may discuss three cases of spinal myelitis which have lately been in my wards. I shall compare these three cases, and you will find that although the pathological process was in each case very similar, they differ very widely from each other as to course, symptoms, and causes, and I trust that the remarks I shall have to make will not only throw new light upon the cases which form the subject of these remarks, but that a study of these cases, slightly more methodical than is possible in the wards, will enable you to

[1] Clinical Lectures, U.C.H., 1896.

understand many of the symptoms which arise in other diseases of the spinal cord.

Case 1.—*Unilateral focal myelitis in the lower cervical region implicating the cervical sympathetic nerve.* J. E——, a painter, aged 32, was admitted to the hospital, on the recommendation of Dr. Hackney, of Hythe, on October 3, 1895, complaining of weakness of the left hand and foot, a tingling sensation the left side of the head, palpitation of the heart on exertion, and sleeplessness. His trouble began in May with weakness of the hand and leg, and he states that there has been some recovery of power in the leg, but otherwise his condition remains *in statu quo.* He had influenza in 1891, and again in the spring of 1895, and he has never been well since the first attack.

He is fairly temperate, and has a healthy complexion. There is no blue line on the gums, and there are no facts in his history which point directly to lead poisoning ; no history of syphilis ; no pyrexia ; urine of normal specific gravity, and otherwise healthy. On examination we found—

1. No paralysis of the face or tongue, but the left palpebral fissure narrowed, and the left pupil smaller than the right. Both pupils reacted to light and accommodation. Sight good ; no contraction of the field of vision as tested with the perimeter ; optic discs normal. He complains of some headache, principally on the left side. No giddiness ; no vomiting ; no staggering during locomotion ; no deafness.

2. There is distinct wasting of the interossei of the left hand, and also of the muscles of the thenar and hypothenar eminence, more marked in the latter than in the former. The left arm and hand are small and flabby as compared with the right, but there is no marked wasting of any muscles other than those named. The muscles of the hypothenar eminence give degenerative reactions. The interossei, the muscles of the thenar eminence and the extensors, respond feebly to both currents as compared with the corresponding muscles on the right side. There is no contracture or rigidity of the left hand or arm.

3. The left leg is weak, and he drags it very slightly in

walking. There is no wasting or absolute want of power in any of the muscles. The knee-jerk is increased on the left side.

4. Sensation is everywhere normal—touch, pain, and temperature.

5. No circulatory troubles, functional or otherwise, were detected during his stay in hospital.

6. No tenderness or deformity of the spinal column.

Here we have a history of a moderately acute onset, and objectively we find symptoms referable to the left side of the face, the left arm, and the left leg ; and in seeking to make a diagnosis, and localise the lesion which gives rise to the symptoms, we shall do right to take the brain into consideration in the first instance. Now, although the onset was moderately acute, it was not sudden, as are the majority of cerebral lesions giving rise to hemiplegia ; and we must note that there is no paralysis of the lower part of the face or tongue, as is common in hemiplegia. Further, it is evident that although the palpebral fissure is narrowed on the left side, there is no ptosis, and no evidence of paralysis of the third or any other orbital nerve. The left pupil is indeed contracted, which might be taken as evidence of paralysis of the sympathetic or irritation of the third nerve.

As there is no implication of any other muscle supplied by the third nerve, we are forced to the conclusion that paralysis of the left cervical sympathetic is the cause of the contraction of the pupil and the narrowing of the palpebral fissure in this case. The sympathetic is supposed to give tone to certain unstriped muscular fibres in the orbit which keep the lids to a certain extent retracted, and maintain the due prominence of the globe. Certain it is that division of the sympathetic in the neck causes precisely the condition of the eye and palpebral fissure which we have here. The condition of the palpebral fissure is very like what is seen in old cases of facial paralysis, in which the paralysis of the orbicularis muscle is followed by contracture with narrowing of the palpebral fissure.

Leaving for a time this condition, which is probably caused by paralysis of the sympathetic, let us turn our attention to

the condition of the left arm and hand. It is to be noted that while the nutrition of the arm is below par, due probably to want of use, there are certain muscles, such as those of the little finger and the interossei, which are definitely wasted; and we find that the muscles of the little finger (hypothenar eminence) give degenerative reactions when tested with electricity, and that the interossei and other muscles of the hand and forearm respond somewhat feebly to both forms of current, but do not give degenerative reactions (i.e. response to galvanic and no response to faradic current).

In the absence of any sensory change, or any evidence of disease or injury of the ulnar nerve in the hand, we are bound to assume that the wasting and degeneration of the muscles of the hypothenar eminence is due to the destruction of motor cells in the anterior cornu of the left side, and we have to answer the question, ' At what level has this destruction taken place ? '

The muscles of the hypothenar eminence are supplied by the ulnar nerve, and the ulnar nerve is derived from the intricate interlacement of nerve-fibres called the brachial plexus, and this plexus is formed by the last four cervical (5, 6, 7, 8, c.) and first dorsal (I D.) nerve-roots, which have their origin in the cervical enlargement of the spinal cord.

A consideration of this plexus led me to say in 1881 (Bradshawe Lecture on ' Nervous Affections of the Hand ') that whereas the median and musculo-spiral nerves possibly had connections with all the nerve-roots forming the brachial plexus, the musculo-cutaneous nerve represented not more than the three upper nerve-roots (5, 6, 7, c.), and the ulnar nerve not more than the two lower nerve-roots (8 c. and I D.) ; [1] and this speculation has been largely justified by the laborious researches of Dr. Herringham, who traced out the fibres, and in a communication to the Royal Society in 1886 showed that the musculo-cutaneous nerve was derived from the two upper roots and the ulnar from the two lower roots, while the median was derived from the four lower roots, and the musculo-spiral from the four upper roots. Considering that the formation of

[1] See p. 3.

the brachial plexus varies considerably, Dr. Herringham's research very closely confirms my speculation on the matter, a speculation founded on considerations with which I need not trouble you now. Thus, on anatomical grounds alone, the muscles of the hypothenar eminence must be supplied by one or other or both of the lower two roots of the plexus.

The experimental investigation of the function of the nerve-roots going to form the brachial plexus in the monkey, made by Ferrier and Gerald Yeo in 1881, showed that the small muscles of the monkey's hand only moved when one or other of the two lower roots (8 c. 1 d.) was stimulated, so that there is a perfect agreement between the anatomical and physiological facts established by dissection and experiment.

We are therefore justified in coming to the conclusion that, in the case which we are considering, there has been a destruction of the motor cells of the left anterior cornu at the level of the eighth cervical and first dorsal roots.

The muscles of the hypothenar eminence alone gave definite and undoubted degenerative reactions, but it was found that the extensors of the fingers and interossei responded very feebly (as compared with the muscles on the opposite side) to both forms of current, and we are justified in concluding that these muscles still received sufficient nerve impulses to prevent their complete degeneration; and we found that the extensors, receiving their supply from the musculo-spiral (having connections with the four upper roots), responded to electricity more readily than the interossei which are supplied by the ulnar.

It very often happens in anterior polio-myelitis that a small group of muscles which is completely degenerated is surrounded by others which are much weakened, but not completely degenerated; and it is, I think, evident that the interlacings of the brachial plexus not only facilitate the purposive co-ordination of physiological acts, but by widely scattering the influence of the motor cells in the cornu they lead to the semi-preservation of muscles which, by means of the plexus, receive their nerve-supply from several nerve-roots.

The next point to which I would direct your attention is the weakness of the left leg without marked wasting or degeneration of any of the muscles, and the exaggeration of the knee-jerk on the left side. This points to a descending degeneration of the left pyramidal tract, and we have a right to assume that the process which has damaged the motor cells at the level of the eighth cervical and first dorsal roots has also blocked the pyramidal tract at that point, and has caused it to degenerate below.

Looking to the fact that the onset of the case was comparatively sudden, that it followed an attack of influenza, and that there has been no increase and no great improvement in the symptoms from the first, we are justified in concluding that the trouble is caused by a patch of myelitis involving the anterior cornu and pyramidal tract, and limited to the left side of the cord on a level with the eighth cervical and first dorsal segments of the cord.

Having, by means of the muscular paralysis, localised the patch of myelitis, we are now in a position to account for the symptoms which we have presumed are due to a paralysis of the cervical sympathetic. The lower cervical region of the spinal cord has long been remarkable for the fact that injury of it has often been accompanied by contraction or dilatation of the pupil, and also by symptoms referable to vaso-motor disturbance, such as dilatation of the blood-vessels, abnormal body temperature, and occasionally priapism. The ciliary muscle derives its sympathetic supply from the superior cervical ganglion, which, according to the researches of Gaskell, receives its cerebro-spinal filaments from the upper dorsal nerves, or, according to other authorities, from the seventh cervical to the first dorsal segments, this region being spoken of as the *cilio-spinal centre*.

Our anatomical diagnosis is thus complete, and we are in a position to say that the contraction of the left pupil and the left palpebral fissure, the weakness of the left arm, and the wasting and degeneration of the muscles of the hypothenar eminence, and also the weakness of the left leg and the increased knee-jerk on the left side, are all caused by a

localised damage (probably myelitis) on the left side of the spinal cord at the level of the eighth cervical and first dorsal segments.

There are points in the history of this patient which make it probable that in the earlier days of his illness he presented evidence of vaso-motor disturbance, for he tells us that he has suffered from palpitations, tingling of the left side of the head, tinnitus, and sleeplessness. We have never been able to discover any evidence of vaso-motor disturbance, and the temperature, which has been normal, was found to be practically equal in both ears. The case is one of rarity and great interest, and the 'hemiplegic symptoms' might have led a careless observer to place the lesion on the right side of the brain.

The cause of the myelitis is a matter of doubt. Such troubles have arisen from lead poisoning (and be it remembered that our patient is a painter), and they have been observed as a sequel of influenza (from which our patient has suffered). He improved very slightly, if at all, under treatment, and it was demonstrated that large doses of iodide of potassium produced no appreciable amelioration of his condition. I believe the lesion to be myelitis, and I fear the prospects of complete recovery are very slight indeed.

Case 2.—*Transverse myelitis in the upper dorsal region, caused by a round-celled sarcoma originating in the third intervertebral cartilage.* The next case which I have to bring to your notice is one of great interest, and is very full of instruction for all of us; and you will note that although the patient was suffering from a serious and necessarily fatal trouble, his symptoms were most indefinite, and to a large extent subjective. Emile J——, a French commercial traveller, aged 36, was admitted on November 22, 1895. He complained of pain in the back, weakness of the legs, and some difficulty in passing urine. The patient looked ill, pale, and thin. The pain in the back was referred to the scapular region, not very defined, and unaccompanied by any tenderness or prominence of the vertebræ. When asked to walk he did so readily, but with evident feebleness, and there was nothing

in his gait which was characteristic of any recognised systemic disease of the spinal cord or brain.

Sensation of all kinds was perfect, but the legs were cold; the knee-jerks were markedly exaggerated, and ankle-clonus was readily produced on both sides. The skin reflexes, plantar, cremasteric, and hypogastric, were not obtained. It is of interest to state that Dr. Isaac, of Gower Street, who sent the patient to the hospital, stated that both by himself and at the Middlesex Hospital systematic examination had been made four or five weeks previously for evidence of disease of the spinal cord or vertebral column, but with negative results in both instances.

The symptoms from which the patient was suffering had been coming on for ten or twelve months. There was a history of discharge of mucus and blood from the naso-pharynx, but an examination of these parts revealed no disease. He attached considerable importance to a crop of boils which had appeared between the buttocks and the knees a few weeks before his admission, and he said he had got 'much weaker since the boils,' which had disappeared before he was admitted to our hospital.

It should be stated that the difficulty of micturition was, on his admission, more subjective than objective. There was no dribbling or incontinence, and no sign of distension of the bladder. The urine usually contained phosphates, but it was otherwise normal, and was not ammoniacal.

The case was puzzling. The exaggeration of the knee-jerks and the presence of ankle-clonus caused us to search for localised disease in the cord above the lumbar enlargement. The chest moved symmetrically, but he experienced difficulty in changing from the recumbent to the sitting position, and we thought there was want of power in the recti abdominales muscles. When he coughed he complained of pain (not great), but there was a complete absence of any tenderness of the vertebral column.

The power of the arms was good, and there were no sensory symptoms referred to the upper limbs. The left pectoral muscle appeared smaller than the right, and the left side of the face

appeared to move less freely than the right. There was some stiffness of the left shoulder, and this we thought probably accounted for the comparative smallness of the left pectoral muscle. There was no wasting of the dorsal muscles. The symptoms of motor disability, whether of the trunk or lower limbs, were all slight, and in short there was a want of correspondence between the objective symptoms and the subjective symptom of pain between the scapulæ. Pain in this situation is often due to stomach trouble, but there was no direct evidence of disease of the digestive system. It was a case to watch, in the hope that new symptoms might bring new light.

The suggestion was made that it might be a case of disseminated sclerosis, and a note was made on one occasion of slight lateral nystagmus ; but this latter symptom was not to be obtained on subsequent occasions, and in the absence of other evidence of disseminated sclerosis (such as intention tremors and staccato speech) I did not consider this view of the case as tenable.

Again, the patient was a rather emotional Frenchman, and possibly made the most of his troubles, and we considered whether the case was a so-called 'pure neurosis' ; but this view was abandoned because there was an absence of sensory symptoms, such as are common to 'hysteria,' and the patient seemed really ill, and further, he had an unsteady temperature, which fluctuated between 98·4° and 100° during the first five days of his stay in the hospital.

On December 2 (ten days after admission) the patient suddenly lost all power in his legs, and a few days later all sensation had disappeared in the legs, and the ankle-clonus and the excessive knee-jerks disappeared, so that the knee-jerk was only present to a very slight extent on the right side. Incontinence of urine supervened. The temperature on the 10th of December ran up to 104°. For the last few days of his life he was troubled with vomiting. He became comatose, and died on December 11, nineteen days after his admission.

The phenomena of December 2 and the subsequent days made it tolerably certain that we had to deal with a trans-

verse myelitis of the dorsal region, and the post-mortem examination, which was made twelve hours after death, showed the correctness of this diagnosis, and also revealed the cause of the trouble.

The spinal cord was first examined. The neural arches were removed, and the dura mater beneath looked normal. On slitting up the dura mater it was noticed that the vessels on the posterior aspect of the cord were somewhat dilated below the mid-dorsal region. On removing the cord we found that the bodies of the third and fourth dorsal vertebræ were freely movable owing to softening of the third intervertebral disc, and we found at this level a growth not much bigger than an almond, pinkish in colour, projecting into the spinal canal between the bone and the dura mater, and evidently compressing the cord at that point. On making sections of the cord we found that for about an inch there was softening approaching to diffluence where the tumour had compressed the cord, but that above and below this level the cord was apparently healthy. A microscopic examination by Dr. Bradford confirmed the macroscopic appearances, the softened patch giving evidence of acute myelitis, while the cord was elsewhere perfectly healthy.

There was no disease of the brain or of any of the thoracic or abdominal viscera, but on inspecting the cavity of the thorax we found a growth concealing the bodies of the third and fourth dorsal vertebræ, and projecting for about an inch beneath the pleura on the left side. This growth was examined by Mr. Drew, and pronounced to be a round-celled sarcoma.

The post-mortem examination cleared up the case, and showed us why the symptoms were so indefinite. We had not to do with a tumour of the cord or its membranes, but with a tumour originating probably in the third intervertebral disc, and involving the bodies of the third and fourth vertebræ. It extended forwards and backwards, more forwards than backwards. When the patient was admitted to the hospital the backward extension was beginning to exercise pressure upon the cord, and when it ultimately produced myelitis we

diagnosed the effect, but remained ignorant of the cause until the post-mortem examination made the course of the patient's troubles quite clear.

I think I may say that our failure to diagnose the exact cause of the myelitis in this case was quite excusable. He had been examined both before admission and subsequently for disease of the vertebræ, but with a negative result, and it is certainly remarkable that a tumour which, before death, had loosened the connection between the third and fourth vertebræ, should not have produced tenderness of the vertebral spines or deformity of the back. The tumour, by its growth, kept the bodies of the vertebræ in their normal relative position, and had not, as in caries, allowed the bodies of the vertebræ to soften and become merged in each other so as to produce angular curvature. Neither had it projected forward sufficiently to interfere with the function of the thoracic organs. Neither had it pressed upon the nerve-roots so as to give rise to neuralgia and definite referred pains at a distance from the seat of disease.

It is probable that the growth was definitely the cause of some of the early symptoms from which the patient suffered, such as the weakness of the rectus abdominis and the relative smallness of the left pectoralis major. The intercostals, levatores costarum, obliquus externus, and rectus abdominis receive nervous supply from the third, fourth, and fifth dorsal roots, but the nervous supply of the pectoralis major is not, according to Professor Thane, in any degree derived from any root lower than the first dorsal. Every muscle, for its satisfactory working, is largely dependent upon the muscles which maintain the fixity of the parts to which, at one or the other end, it is attached ; and it may well be that a growth, such as was present in this case, by loosening the spinal column in a slight degree, and by interfering with the fibres of some of the muscles nearest to it, might cause much more muscular incapacity than could be inferred by a mere reference to anatomy.

There is one point in connection with this case which demands attention. It is this. It will be noticed that when

first admitted the patient manifested increased knee-jerks and ankle-clonus, or an exaggeration of the deep reflexes ; but some days before death these deep reflexes had almost entirely disappeared, their disappearance being probably coincident with the completion of the destruction by softening of the entire thickness of the spinal cord on a level with the third intervertebral cartilage.

This fact is in accordance with the observations of Dr. Bastian, who has combated the opinion which has been generally held hitherto, and has asserted that so soon as a limited transverse lesion in the dorsal or higher regions of the cord becomes *total* the so-called 'deep reflexes,' which previously were exaggerated, become depressed and abolished.

This condition of the deep reflexes is generally explained by saying that the exaggeration is due to an increase of muscular tonus caused by the cutting off of cerebral control. The reflex movements in the frog are, as is well known, demonstrated far more easily when the animal has been pithed, or the brain removed. In man, the muscular movements which are excited by (*a*) *direct* cerebral or (*b*) reflex (peripheral) stimulation seem to bear, as it were, an inverse ratio to each other. The greater the former, the less the latter, and *vice versâ*. In sclerosis of the pyramidal tracts following a cerebral lesion, or a lesion high up in the cord, the movements in response to cerebral stimulation are lessened or lost, and the reflex movements are exaggerated. In posterior sclerosis reflex movements are lost, but the direct movements are sudden, violent, and impulsive. When lateral sclerosis follows as a consequence of a limited lesion which blocks the pyramidal tracts, the reflex movements may, in the very early hours of the trouble, be entirely lost, which we account for by using the term 'shock.' This is seen in cases of sudden hæmorrhage or embolism affecting the motor tracts of the brain. After this the reflex movements reappear, and reach their full development in a week or ten days, when the degeneration of the pyramidal tracts is complete. The motor cells in the anterior cornu are the points towards which the cerebral stimuli and the peripheral stimuli converge, and when

either one or the other set of stimuli is in abeyance those which remain produce an exaggerated effect. We must, I think, assume that after the cerebral influence has been cut off, and before the degeneration of the pyramidal tracts is complete, stimuli from other points than the brain may travel down the pyramidal tracts, and produce an effect upon the motor cells ; but after the degeneration is complete the motor cells can only be stimulated by peripheral stimuli at their own level, and this stimulation produces an exaggerated effect.

It has been suggested that the increase of the reflex effects is due not only to the cutting off of the cerebral control, but to the non-antagonised influence of the cerebellum. The experiments of Risien Russell and others conclusively show that a removal of one-half of the cerebellum produces an exaggeration of reflex action on the same side, and these and similar experiments lend no support to the theory of cerebellar action as the cause of the exaggeration of the deep reflexes.

In the lower animals a clean and complete division of the spinal cord does not abolish the deep reflexes, which, on the contrary, are exaggerated. It is well known, also, that slow compression of the cord, as in vertebral caries, leads to an exaggeration of the deep reflexes. On the other hand, in cases of fracture of the vertebræ it commonly happens that the deep reflexes are abolished, as has been demonstrated by the careful clinical observations of Mr. Thorburn of Manchester, and others.

It is possible that the *sudden* occlusion of the spinal arteries, either by fracture of the vertebræ or the effect of acute inflammation, may so cut off the blood supply from the central parts of the spinal cord below, as to prevent the manifestation of reflex action, and it is possible that this effect may be more easily produced in man, in whom the spinal cord runs vertically, than in other animals which carry the spinal cord horizontally. The spinal arteries (one anterior and two posterior) are derived from the vertebrals, and the pressure of blood in them must be considerable. These arteries are, it is true, plentifully reinforced by other arterial twigs,

and it is probable that the loss of the blood supply by the arteries running vertically downwards would be soon compensated by those which reach the cord along the nerve-roots, travelling horizontally or, in the lower parts of the cord, nearly vertically upwards. I think it is not possible, especially in man, to neglect the cutting off of a portion of the blood supply as a factor in the production of abolition of the reflexes in cases of fracture or sudden occlusion of the arteries by inflammation.

This interesting point, to which Dr. Bastian has called attention, stands in need of further elucidation.

Case 3.—*Transverse myelitis in the lower dorsal region occurring in a syphilitic subject.* W. D——, aged 38, a stoker, was admitted on August 14, 1896, suffering from paraplegia. He states that about the middle of July he experienced some difficulty in passing urine, and that this difficulty, with some fluctuations, increased, and that he became an in-patient in a surgical ward of this hospital, where he underwent circumcision on account of a swollen prepuce with purulent discharge from beneath it. No stricture was discovered, and on August 6 he was discharged, 'cured.'

The onset of his attack was fairly sudden, and he names August 7 as the day upon which his legs became, as he says, 'shaky,' and a few days later he had lost all power in them. On admission, both legs were completely paralysed, and the acts of defæcation and micturition occurred automatically and unconsciously.

Tactile sensation was almost absent in both legs and upwards to a line midway between the costal margin and the umbilicus. Painful sensations and the appreciation of heat and cold were also deranged, and there was obvious delay in the recognition of tactile impressions.

There was a big blister on the sole of each foot in the arch, and there were blisters behind the head of each fibula and above the sacrum, on spots where counter-irritants had been applied. No counter-irritant had been applied to the soles.

There was a blotchy eruption over both legs, and the patient gave an undoubted history of syphilis which had been contracted ten months before his admission.

The knee-jerks were increased, but there was no ankle-clonus. The plantar (?) and cremasteric reflexes were absent. The abdominal and epigastric reflexes were present.

The urine (which was passed involuntarily) was ammoniacal, and contained pus. There was no marked wasting of the muscles, and none of them gave degenerative reactions.

Since his admission he has very much improved. The sensory symptoms were, as is usual, the first to pass off. Tactile sensation was the earliest to recover, and the other sensory derangements followed. Next he got some control over his rectum, so that incontinence of fæces is no longer present, and finally, he has a considerable return of motor power in the legs.

The knee-jerks are still exaggerated, and there is well-marked ankle-clonus.

The sores upon the soles of the feet are healing well, and showing no tendency to extend either in breadth or depth. The other blebs mentioned healed readily, and, thanks to excellent nursing, there have been no ' bedsores.'

There is less incontinence of urine, but the urine is no longer ammoniacal, and the pus has almost disappeared.

In considering this case we have to decide (*a*) the situation and (*b*) the nature of the lesion. As regards the situation, the facts which guide us are : (1) The complete paralysis of the legs, showing that the anterior crural as well as the lumbo-sacral cord were implicated, which would place the lesion as high as the first or second lumbar segments ; (2) the disappearance of the cremasteric reflex and the persistence of the abdominal reflex, which makes it highly probable that the lesion was situated at the level of the junction of the roots of the twelfth dorsal and first lumbar nerves with the cord, and about the level of the tenth and eleventh dorsal spines.

As regards the nature of the lesion, the points which lead to a diagnosis are : (1) The sudden onset. There had been some premonitory difficulty of micturition, which is common in these cases ; but when the paraplegia declared itself it did so suddenly, so that the patient had no hesitation in fixing the date. This is very characteristic of myelitis. (2) The

patient was undoubtedly syphilitic, and the concurrence of syphilis and myelitis is sufficiently common to make it probable that there is some relation between them. The absence of marked pain in the back, or of neuralgic symptoms referable to the terminations of the sensory nerves which run into the cord at the level of the injury, make it unlikely that we have to do with gumma or pachymeningitis. The attack was too sudden for such an explanation. Syphilis, however, is a great cause of arterial disease, and it seems highly probable that an endarteritis of some of the vessels at this level, leading to their obstruction and consequent faulty nutrition of the cord, may have contributed to the catastrophe. The spinal cord, you will remember, is nourished by one anterior and two posterior spinal arteries, derived from the vertebrals and running nearly the whole length of the cord, and these three arteries are supplemented by others which reach the cord through the intervertebral foramina in its entire length, and travel along the nerve-roots to reinforce the three long arteries derived from the vertebrals.

The sensory symptoms in this case (especially the thermic symptoms and analgesia), combined with the trophic lesions, of which I shall have more to say presently, point to the fact that the parts around the central canal are implicated. These parts are supplied by the anterior spinal artery, and it seems very probable that a blocking of the anterior spinal artery by syphilitic endarteritis at the level of the lesion is the cause of our patient's trouble, or at least an accessory cause. Such a theory will account also for the perfect symmetry of the symptoms. The very marked improvement which has taken place as the result of treatment makes such a theory still more plausible. It is possible also that there may have been some hæmorrhage into the softened cord, which has since undergone absorption.

The almost complete absence of 'girdle sensation' in this case seems to point to the lesion being limited very much to the anterior and central parts, and avoiding the posterior roots, upon irritation of which girdle sensation is supposed to depend.

A very notable feature in the case we have been discussing are the trophic lesions. By a 'trophic lesion' we mean a failure of nutrition. The nutrition of an organ depends upon its power of exercising function. Growth and use are to a large extent mutually dependent. We know that muscles may be increased in size by exercise, and that when not used they waste. The degree of wasting which a muscle may undergo varies immensely. It may be partial or it may be absolute. Lesions of the nervous system which lead to a partial disuse of muscles cause only partial failure of nutrition, while lesions which necessitate a total disuse lead to total wasting, degeneration, and practical abolition.

Our muscles can be made to contract by direct or reflex stimulation, i.e. the stimuli which bring the muscle into use may reach it from the brain or from the periphera. In ordinary hemiplegia the wasting of the paralysed muscles is only partial, because contraction takes place in response to reflex stimulation, a stimulation which is often sufficient to lead to the permanent contracture of the paralysed muscle.

Again, in locomotor ataxy there is seldom more than slight wasting of the muscles, because although reflex stimulation of the muscles may be abolished, direct stimulation is still capable of being exercised.

If a nerve supplying a muscle be divided, a total wasting rapidly follows, because direct and reflex stimulation are alike impossible. If an anterior nerve-root be divided, or if the motor cells of the anterior cornu be destroyed, the muscle undergoes total wasting, because direct and reflex stimulation are alike impossible.

It is common to hear it said that the motor cells in the anterior cornu exercise a 'trophic influence' upon the muscle, but there is no evidence of any trophic influence beyond the fact that all stimuli which reach the muscle, whether direct or reflex, must pass through these cells, and when they are destroyed no stimuli can possibly reach the muscle. Function is the only thing having a 'trophic influence' on organs of which we have any knowledge. A healthy muscle is plump,

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firm, elastic, and is constantly contracting in obedience to stimuli, of many of which we are unconscious. These contractions must influence the blood and lymph currents and regulate nutrition. The muscle which is isolated from all stimuli is flabby, limp, and absolutely inert, yielding to pressure, and having no use for nutriment.

With regard to the blebs and sloughs upon the skin, we must, I think, in looking for a cause have regard to the general circumstances, and not attend too exclusively to individual factors. My belief is that they are due, if I may say so, to an unlucky concurrence of adverse circumstances, any one of which might be powerless to produce them.

Pressure alone, if prolonged, is capable of producing a slough even in a healthy man, as occasional accidents in surgical practice bear witness.

The effects of pressure may be intensified by a variety of circumstances. Thus, in fevers and other prolonged and exhausting diseases accompanied by emaciation with wasting of fat and muscles, the squeezing of the skin between the bones on the one hand, and even an air cushion on the other hand, is sufficient to cause a slough over the sacrum. The pain of prolonged pressure makes us alter the point of pressure ourselves or leads the sick man to ask his nurse to shift his position. If the fever patient is unconscious he will not seek to shift his position, and his danger of bedsore is therefore increased.

A man in a deep drunken slumber will sometimes compress the musculo-spiral nerve between his 'heavy head' and his humerus to such a degree as to interfere with its nutrition and cause paralysis of the muscles supplied by the nerve, but the average man is awakened by the pain long before the compression reaches the dangerous point. If the parts which are compressed are anæsthetic, as in many cases of paraplegia, the danger of sloughing from compression is very great, for the patient not only feels no pain, but, being paralysed, is powerless to shift his position.

When there is anæsthesia without paralysis, as happens in some hysterical cases, the danger of sloughing from pressure

is almost *nil*, because the muscles are plump and serve as protective cushions, and the patient can move freely.

If the skin be irritated the danger of sloughing from pressure is increased, and I would here caution you as to the risk of applying strong counter-irritants, such as blisters, to any part of the back of a fever-patient. The irritation caused by lying in a mess of urine and fæces enormously increases the danger of sloughing, and hence it is that all patients who are unmindful of the calls of nature, whether as the result of paralysis or from the stupor and exhaustion of fevers, require the most watchful care on the part of the nurses and doctors. A sore once formed may be infected by septic organisms, and a rapid extension may result.

It is obvious, therefore, that a patient who is paralysed and anæsthetic must be very vulnerable to the effects of pressure or irritation of the skin. We must remember that muscular action is an important factor in the circulation of the blood, and that in a paralysed limb the return of the venous blood and the flow of lymph are alike deprived of the aid they derive from muscular contraction.

If the muscles underlying the part which is subjected to pressure be really cut off from the motor cells, they lose tone— they no longer serve as elastic cushions to ease the pressure— they sag, as it were, under the weight of the body, and allow the skin to be squeezed to death between the dead weight of bones above and the unyielding bedclothes beneath.

There can be little doubt that vaso-motor irregularities may contribute towards the occurrence of trophic lesions, but our knowledge of these conditions is very slight. The redness which follows an irritation of the skin is, one must suppose, a. vascular reflex. If we tickle the skin we get a rapidly following muscular action. If we slap the skin or make a limited firm pressure upon it there is no involuntary muscular movement, but the pallor which is caused by the pressure is leisurely followed by a blush. In certain conditions of intra-cranial pressure, such as tubercular meningitis, this blush is very pronounced and very enduring, and this exaggerated form of vascular reflex has been called the ' *tâche cerebrale*.'

That gentle tactile impressions made upon the skin should produce reflex muscular acts, while impressions of a more penetrating kind, such as firm pressure, scratching, and the application of heat or chemical irritants, should produce vascular dilatation, is interesting, and seems to point to the fact that the different end-organs in the papillæ and in the deeper layers of the skin may have different central connections.

Although I have lectured on Medical Jurisprudence for many years, it has never fallen to my lot to see an acute case of strychnine poisoning. In these cases, as you are aware, any slight unexpected cutaneous impression will produce a paroxysm of tetanic convulsions, and yet these patients will often ask to be turned over or held, and von Boeck, the writer on the subject in von Ziemssen's 'Encyclopædia of Medicine,' says, with regard to sufferers from strychnine poisoning, that they 'have asked their attendants to rub the skin vigorously, and that this friction did not induce tetanus,' while slight unexpected impressions, such as an accidental kick against the bedstead, produced the most violent convulsions.

It may be, of course, that this difference in effect was due to the mental attitude of the patient—to the absence or presence of cerebral control; but there is a fair amount of evidence to show that slight tactile impressions and firm rubbing produce different physiological effects, the former being mainly reflex muscle-motor, and the latter mainly reflex vaso-motor.

I need not remind you that in the skin there are varieties of nerve 'end-organs,' and that while some, such as the Pacinian bodies, lie deeply in the true skin, others, such as the tactile corpuscles, are more superficial. In cases of paralysis, whether cerebral or spinal, we do not get many very obvious vaso-motor disturbances. There are occasional differences of temperature between paralysed and non-paralysed limbs, and sometimes, as in anterior polio-myelitis, there may be a blue congestion of the paralysed limb, and we have alluded to the *tâche cerebrale*.

The subject is one which is difficult of investigation, but

we must admit the possibility that lesions of the nerve centres may derange the vaso-motor connections, and that such derangements may play a part in the production of skin lesions.

The occurrence of trophic changes in the eye in cases of paralysis of the fifth nerve is very uncertain, and division of the sympathetic causes no trophic change in the rabbit's ear. Anæsthesia alone, and vaso-motor paralysis alone, are unable to cause trophic disturbance ; and I would again repeat what I have already said, that these trophic lesions depend upon a concurrence of many adverse circumstances.

It is clearly our duty to exhaust all the causes which may possibly produce trophic disturbance before we avoid the whole difficulty by calling them 'tropho-neuroses,' and attribute them to the action or want of action of hypothetical 'trophic nerves,' which in the present state of our knowledge is no explanation at all.

But let us return to our patient. When he was admitted there were blebs on the soles of both feet, and behind the heads of both fibulæ, and also over the sacrum. They had appeared in the very early days of his paralysis when the lesion was at its height. After his admission there was no extension of trophic disturbance, and the blebs on the sacrum and behind the heads of the fibulæ soon disappeared. The blebs on the back and behind the heads of the fibulæ followed the application of mustard, but those on the soles of the feet appeared quite spontaneously, and as they were in the hollow of the arch of the foot they could hardly be attributed to pressure. My view being that a small amount of irritation would be sufficient to develop the blebs in the then condition of the patient, I should think of scalding hot foot-warmers before flying to 'trophic nerves.'

But there is one other circumstance which may have acted as a determining factor in the localisation of these blebs.

Dr. Henry Head has shown that our internal organs are functionally related with definite areas of the skin, which fact is accounted for by assuming that the afferent and vaso-motor nerves of the skin area and the like nerves from the related

viscus join the spinal axis at the same level, and come into intimate relationship therein.

In Dr. Head's very able and elaborate papers, published in 'Brain,' and also in the last edition of Quain's 'Anatomy,' you will find diagrams in which these zonal areas on the trunk and similar areas on the limbs are set forth, and accurately referred to certain levels of the spinal axis.

Suppose, therefore, that you find one of these definite areas the seat of subjective pain or demonstrable tenderness, or reddened or occupied by an herpetic eruption, you will have a right to assume one of two things, either (a) there is disease more or less permanent in the related segment of the spinal axis, or (b) there is disease of an internal viscus or organ which is in relation with the identical segment.

If we apply Dr. Head's diagrams to the elucidation of the present case we shall find that the areas upon which the blebs appeared (sacral, solar, and behind the heads of the fibulæ) are related to the sacral segments of the cord, the same segments which are in relation with the afferent and efferent nerves of the bladder.

My view of the case would be as follows : We know that the bladder symptoms preceded the paralysis by a month, and that when he was admitted here there was well-marked vesical catarrh. When, therefore, he became suddenly paralysed and anæsthetic, and was, so to speak, ripe for trophic lesions, the already existing serious condition of the bladder determined the seat of the blebs, which may possibly be considered as analogous to herpes, a 'trophic' disease definitely related to the sensory nervous system.

Yet another point is worthy of mention. So-called trophic troubles are said to be very liable to occur in central diseases of the cord, such as syringomyelia. Or we might put the matter in another way and say that when conduction for pain and temperature are interfered with, trophic troubles are more likely to occur than when tactile sense is mainly affected.

If all the sensory paths be blocked, as was the fact for a short time in the case we have been considering, the danger of trophic changes is greatly enhanced.

Finally, a few words as to the treatment and management of these cases. The third case has proved amenable to treatment, and therefore it will be most profitable to deal with it.

The undoubted history of syphilis led at once to the administration of mercury and iodide of potassium. The mercury has been given by inunction and by the mouth in the form of hydrargyrum c. cretâ, one grain three times daily. And the iodide of potassium has been given in doses running up to twenty grains three times a day. Now and again the administration of these medicines has been intermitted for a few days when the appetite appeared to be flagging, and a simple stomachic tonic has been given instead.

For the rest the treatment has been directed to keeping the patient clean and endeavouring to keep the bladder disinfected.

It is impossible to insist too strongly on the absolute importance of attention to the bladder. If the bladder be permitted to remain foul, the patient is tolerably certain to go from bad to worse. When there is paralysis of the bladder, with constant dribbling of urine, as in this case, the organisms which produce ammoniacal decomposition of the urine travel up the urethra and find lodgment in the bladder, with the result that the contents of the bladder become ammoniacal and irritating, and set up a suppurative catarrh which may travel up the ureter and produce secondary nephritis, and lead almost certainly to a fatal result. The bladder must be washed out daily, the greatest care being taken that the catheter is in an aseptic condition. Carbolised oil (absolute phenol 1, castor oil 7, almond oil 8) should be used as a lubricant, and a wash composed of five grains of quinine (with sufficient dilute sulphuric acid to dissolve it) in a pint of water should be used. If this be not successful in disinfecting the bladder, recourse may be had to an iodoform wash made by stirring up a couple of teaspoonfuls of iodoform emulsion (iodoform five grains, glycerine two drachms) into a pint of tepid water. This seldom fails to be of service. A lotion of boric acid (1 in 20) is also useful. If one lotion does not succeed, another

must be tried, and we must remember that the bladder *must* be kept sweet.

I wonder how often it has happened that a doctor called to a patient, who cannot pass his water, has used a dirty catheter, rendered the bladder foul, and thereby has deprived an incipient case of paraplegia of any chance of recovery.

When the bladder is emptied by catheter previous to washing, some of the urine should be received into a clean vessel, to ascertain whether it is sweet. It is impossible to form any conclusion from the urine collected in a bed-bottle, for these bottles are nearly always foul, and the urine will undergo decomposition in them in a very short time. These patients must be furnished with a urine bottle which lies between the legs, and into the neck of which the penis is inserted. There should always be two bottles in use for every case of paraplegia, one in use and one being cleaned. To clean these bottles it is necessary to rinse them out with dilute acid and then boil them. The bottles should be absolutely aseptic, and should be changed two or three times daily.

It is surprising to see how greatly the patient's general condition improves when the bladder has been thoroughly disinfected.

The prevention of bedsores is sometimes very difficult, but no pains are too great in order to prevent them if possible. Absolute cleanliness is most important. The patient must be scrupulously washed whenever he gets fouled by urine or fæces, and, after washing, the part must be thoroughly dried with a warm soft towel and powdered. If the draw-sheet be damp it must be changed. Some nurses advocate the washing of the sacral region with spirits of wine, which is said to harden the skin. The use of water cushions and of perforated cushions to relieve pressure is often very necessary, and the frequent shifting of the patient so as to alter the points of pressure is useful. The prevention of bedsores is an affair of nursing, and every good nurse feels that a bedsore is a matter which more than any other touches her professional reputation. Nevertheless, you must remember that the doctor is the

responsible person, and my advice is that you *should never visit a paraplegic patient without looking at the back.*

If there be an abrasion the best treatment probably is to dust it with iodoform, and then cover it with boric ointment spread upon folded lint (two or three thicknesses). The patient must lie upon a perforated pillow or a graduated pad.

When the patient's recovery has somewhat advanced, as shown in this case, by recovery of power over the rectum or the return of sensation, it is usually advisable to stimulate the paralysed muscles by means of electricity.

This must be done methodically, and in applying the current you must take care that the patient is not made either cold or wet. Take a faradising battery, with two moderately big sponge-holders moistened with *hot* salt and water. Begin above, and go down the front of the abdomen and legs. I lay stress upon the abdomen because the weakness of the rectus and other abdominal muscles is a contributory cause of the difficulty of micturition, which is so marked a feature in these cases. Use a current of sufficient strength to cause a contraction of the muscles, and make each muscle or group of muscles contract two or three times and no more.

Having attended to the extensors and adductors of the thigh, the tibial and peroneal groups of muscles, proceed to faradise the foot, and I may say that it is most important not to neglect the interossei and other small muscles of the foot, because the contraction of these muscles has the effect of causing a flow of blood to the very extremity of the limb. Having attended to the front of the right leg, wipe it dry with a soft *hot* towel, cover it up to protect it from the cold, and proceed to do the same with the left leg. Then turn the patient over, and faradise the various groups of muscles from the loins to the soles of the feet, taking care when you have finished to leave your patient dry, warm, and comfortable.

The application of the faradising current in these cases is often productive of great good, and the same current applied to the sphincter and bladder will often impart tone to those parts and make an incalculable difference in the patient's comfort.

It will sometimes happen that the first application of electricity does an amount of good which astonishes both doctor and patient. These are the cases in which time has worked an almost complete cure of the central lesion, and the muscles, long disused, were only waiting for a stimulus of sufficient strength to rouse them to their former activity. Such cases are few and far between, and the applications of electricity subsequent to the first are not productive of the same degree of improvement or anything like it. Very often electricity does no good. If it be used too early in the case, while the spinal cord is still the seat of active disease, it may do harm. When, however, a patient, as in this case, has been lying by for months, when his temperature goes to normal and stops there, when he improves in appearance and begins to put on flesh, then is the time when electricity is to do good, and its beneficial results make us recognise that 'There is a tide in the affairs of men, which, taken at the flood, leads on to fortune.'

CHAPTER VIII

TWO CASES OF PHOSPHORUS POISONING [1]

IN my systematic lectures on Medical Jurisprudence I am in the habit of insisting that the study of toxicology is of great scientific value, on account of the fact that the symptoms manifested by the poisoned person flow from a single cause. In the majority of cases which we study in the hospital a number of causes have been at work, and it is often difficult for us to say which out of many may have been most potent in the production of the effects observed. When, however, a healthy person takes a dose of poison and is immediately made ill thereby, we can be in no such doubt, and it is most instructive to observe the numerous and varied symptoms which may arise as the result of a single cause. Poisoning by phosphorus has an especial interest, because in combination it exists plentifully in the body, and it is in itself an interesting fact that a few grains of pure phosphorus should be able to cause the death of the individual, and this without the production of an immediate and severe local lesion such as is produced by the corrosive acids.

It is only in recent years that phosphorus poisoning has become common, and it is evident that until phosphorus itself became a tolerably common article of commerce it was impossible for cases of poisoning by it to be observed and studied. Phosphorus is said to have been discovered by the German alchemist Brandt in 1678, who obtained it by the dry distillation of urine solids ; but it remained a rare chemical curiosity until Scheele succeeded in producing it from bone ash. It was not, however, until phosphorus became a common article of commerce for the manufacture of lucifer matches

[1] Clinical Lecture, U.C.H., 1888.

that cases of poisoning by it began to be recorded. The phosphorus match industry (according to Paton in the article 'Matches' in the 'Encyclopædia Britannica') had its origin in Vienna in 1833, and for a long series of years Austria and the South German states were the principal centres of the new industry. Before this date cases of phosphorus poisoning were very rare, and arose from its injudicious use as an aphrodisiac (Briand and Chaudé). Orfila's knowledge of the subject seems to have been derived solely from experiments on animals ; and Christison (writing in 1836) had never seen a case of phosphorus poisoning in the human subject, though he mentions three cases recorded by continental observers (the earliest being one by Worbe in 1824). The use of phosphorus as a poison has been hitherto more in vogue on the Continent than in this country ; and Briand and Chaudé, writing in 1869, say, 'This form of poisoning increases in an alarming manner,' and they utter the warning that phosphorus paste used for the destruction of vermin may be the cause of poisoning if animals used for food accidentally partake of it. How small has been the amount of phosphorus poisoning observed in this country may be judged of by the fact that in the first edition of Guy's 'Forensic Medicine' (1854) it is not mentioned ; and that Dr. Wilks, in the first edition of his lectures on 'Pathological Anatomy' (1859), does not mention phosphorus as a cause of fatty degeneration of the liver. It was not, indeed, until about the year 1861 that the action of phosphorus in producing fatty degeneration began to be appreciated. Jaundice had been noticed as a symptom of phosphorus poisoning, but its occurrence was regarded as a coincidence, and was attributed to the blocking of the bile duct by the swelling of the mucous membrane of the duodenum, brought about by the irritation of the poison. Fatty degeneration of the liver is a sufficiently common post-mortem appearance, and its occurrence in phosphorus poisoning did not at first excite any special attention. It is now, however, recognised that jaundice and fatty degeneration of the liver and other organs are the chief phenomena of phosphorus poisoning.

Cases of phosphorus poisoning are usually accidental or

suicidal. Its accidental occurrence has been seen in children who have sucked the ends of matches tipped with common phosphorus, or have eaten bread-and-butter smeared with phosphorus paste, which has been prepared for the destruction of vermin. Suicides in this country usually make use of phosphorus paste, a compound composed of fatty matter, flour, and colouring material, and containing about 4 per cent. of phosphorus. On the Continent, however, the heads of lucifer matches have been frequently used for suicidal purposes. It may be well to remind you that the ordinary form of phosphorus which fumes in the air is alone poisonous. Amorphous phosphorus is believed to have no poisonous action.

The cases to which I wish to direct your attention are two in number. The patients were both women, one of whom died, and the other recovered. The first case was one which I saw in consultation with Dr. Hurd Wood, of Leatherhead, and it is by his kind permission that I am enabled to bring the facts before you.

A. L——, aged twenty-three, a young woman living 'under the protection' of a gentleman, in consequence of some annoyance to which she had been subjected in her quasi-connubial arrangements, took at about 4 P.M. on the afternoon of August 30th the greater part of a fourpenny pot of rat paste, and it is a remarkable fact that in doing this she followed the example of her elder sister, who under similar circumstances had destroyed herself a few years previously by taking phosphorus paste. Five hours later—i.e. about 9 P.M. —she vomited, and medical assistance was summoned. It must be borne in mind that she had probably had eight or ten grains of phosphorus (about two grains being a fatal dose) lying in her stomach for five hours, so that ample time had been allowed for the absorption of the poison. Headache and vomiting were the chief symptoms. The first matter vomited, which it was alleged smelt of phosphorus, was thrown away ; and it is to be noticed that none of the subsequent vomit had any smell of phosphorus, nor did it fume, nor was it luminous, which sometimes is the case. On August 31st and September 1st she continued to vomit. On September 2nd

the vomit had the appearance of altered blood. The vomiting was so incessant that it was practically impossible to give either food or medicine by the mouth. On the 2nd, jaundice, a symptom of very grave import, showed itself ; and on the 3rd I saw the patient, about ninety-eight hours after taking the poison. She was in bed, lying on one side, complaining of great pain in the abdomen, which was covered by a linseed poultice. She was quite sensible, but was very unwilling to talk or be disturbed or to attempt to take nourishment. The skin was very sallow. The ocular conjunctivæ were distinctly yellow ; the depth of icteric tint was not very great. The pulse was regular, slightly quickened, and of good force. The heart sounds were normal. Tongue slightly coated ; papillæ at tip and edges rather enlarged. The liver dulness was decidedly increased ; the lower margin was well below the ribs ; and the abdomen over the liver was exquisitely tender, but there was no tenderness elsewhere over the abdomen. There was no diarrhœa, and the stools were said to be natural. The patient complained of very severe pain in the back, and the least vibration or movement of the bed seemed to cause her great agony. There had been severe cramp in the legs. The urine was, it was said, scanty and high coloured ; the bladder was apparently empty. It was not till the following day that Dr. Hurd Wood managed to get a sample of urine for examination, concerning which I will presently speak in detail. As the patient had taken no food since the poisonous dose, and very little during the previous week, I advised that she should have some morphia administered by the mouth, and that after an injection of turpentine she should be fed with peptonised arrowroot and milk per rectum. Owing to the irritable state of the stomach, it was impossible to give turpentine (the recognised antidote) by the mouth ; and I considered it probable that, seeing that ninety-eight hours had elapsed since taking the poison, not much good could have resulted, and probably some harm, by increasing her vomiting and exhaustion. We considered the prognosis to be very bad—almost hopeless. She continued in much the same state till her death, which took place on September 5th. There was no delirium or coma,

the cause of death being attributable to gradual failure of the heart. The patient lay at last almost motionless, and for some hours before death there were lengthy periods during which the radial pulse was not perceptible. The only symptom distinctly referable to the nervous system was cramp in the legs, from which the patient suffered during one night.

The post-mortem examination was not until ninety-three hours after death, because Dr. Hurd Wood and myself hesitated to make it, under the circumstances, until the coroner had given his order. The cause of death was not a matter of doubt, and it was only after the inquest and verdict that the order was given. The examination was made under circumstances of difficulty, as are all necropsies made in private houses, but the main facts were sufficiently well ascertained. The body was still rigid, and was fairly well nourished. The skin was distinctly yellow, the post-mortem staining being strongly marked. The abdomen was distended, and blood-stained fluid exuded from the mouth and nostrils. No luminosity was observed, but it should be added that the day was bright, and it was not possible to absolutely darken the room. There was no smell of phosphorus either before or after opening the body, but those present agreed that it had a peculiar odour. The chief facts observed were the following : 1. Marked fatty degeneration of the liver. It was distinctly not enlarged, but was of about average size. It was yellow in colour, sticky looking, and pitting on pressure. When divided, and looked at from a little distance, it had the appearance of a mass of soft yellow fæces. It broke down easily under the finger. Acini well marked. The microscope confirmed the marked fatty degeneration. The gall bladder was nearly empty, and contained little more than mucus. 2. The heart was perfectly empty, flabby, and flaccid, and in an advanced stage of fatty degeneration. 3. Kidneys soft and flaccid ; pyramids deeply congested ; cortex swollen, of a dirty grey colour, mottled with yellow. A microscopical section kindly made by Mr. Murray showed the epithelium to be full of granular and fatty particles. 4. Spleen not enlarged, very soft, and of dirty plum colour. 5. Lungs deeply congested (hypostatic).

6. Stomach : Mucous membrane swollen, yellowish, and very soft. Not hyperæmic. No congested patch, except at one spot, where there was seen a white particle in the middle of a patch of congestion the size of a pea. This particle we took into a corner of the room, and, producing all the darkness we could, we gradually applied the heat of a spirit lamp, but no phosphorescent fumes were observable. 7. Duodenum yellow and soft ; mucous membrane swollen ; no ulceration or hyperæmia. 8. Colon fairly normal, and containing some masses of absolutely pale fæces. 9. A small ecchymosis was observed on the mesentery, but no ecchymoses were observed elsewhere. 10. Urinary bladder slightly injected ; empty. 11. The vagina contained a good deal of grumous yellowish mucus. A small clot was observed blocking the os uteri. The uterus was deeply blood-stained inside, and contained some clot ; and the ovaries on section showed some Graafian follicles, also containing clots. With the exception of the genital organs, no clots of blood were found anywhere, the blood appearing to be uniformly fluid. We could not ascertain whether or not the patient was menstruating at the time of her decease.

The symptoms and post-mortem appearances which I have shortly laid before you are those of the majority of cases of phosphorus poisoning. In some cases hæmorrhage is a more marked feature, and petechial ecchymoses are more abundant post-mortem. The liver is uniformly fatty, as in this case, but is sometimes mottled with patches of yellow and red. Its size varies : if the patient die early, it may be big ; if, on the other hand, the patient survive some time, it may be in a state of marked atrophy. The kidneys vary also ; they may almost escape, or may be nearly as fatty as the liver. The same may be said of the heart. These variations are to be expected, and depend on the amount of poison absorbed and the duration of the case. Sometimes delirium and coma are a marked feature, which depend probably upon the fatty degeneration of the blood-vessels of the cortex cerebri, and also probably to some extent on the conditions of the kidney, the coma being in a sense 'uræmic.'

Rationale of the Symptoms.—When a poison is absorbed

from the alimentary tract and enters the portal circulation, the liver is necessarily the first organ to suffer from the effects of it, and is usually more markedly affected than any other organ of the body. The effect produced when phosphorus is the poison is that of rapid fatty degeneration. The liver cells are found to be filled with oily particles, and when the fatty change is as marked as in the case under review, it is impossible to believe that the organ is capable to any extent of performing its normal functions. The extent to which the other organs suffer from fatty degeneration depends upon the amount of phosphorus which passes through the liver or is subsequently given off by it into the general circulation.

Why does phosphorus produce fatty degeneration? Here is a field for speculation; but probably our best course pending more exact information is to accept the fact, which is undoubted, and, with regard to the cause of this remarkable property of phosphorus, to say we do not know. This power of producing fatty degeneration is possessed by some other bodies, and notably by antimony and arsenic, but these latter bodies (which, be it observed, belong to the same chemical group as phosphorus) possess the power in a degree very inferior to phosphorus. It is possible that some interference with the process of oxidation in the liver cell may be the cause, and that in the readiness with which phosphorus combines with oxygen may be found the explanation. That this has something to do with it seems the more likely from the fact that it is only phosphorus in its fuming active form that is harmful, and that amorphous phosphorus is not a poison. Further, oil of turpentine, the vapour of which destroys the 'fuming' power of phosphorus, is apparently an antidote to it as a poison. It may be that phosphorus acts primarily upon the blood, and only produces its effect on the liver as a consequence of the blood change. It is certain, however, that in order of time and in severity of effect produced, the liver holds the first place among the organs of the body in phosphorus poisoning. This fatty degeneration ultimately affecting all the tissues of the body accounts for all the symptoms, at least in part. The jaundice is partly caused by the swelling of the

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fatty liver ; the albuminuria by the fatty change in the kidney ; the hæmorrhage so often observed is probably due to a fatty degeneration of the blood-vessels ; and the coma and delirium may be due partly to a fatty change in the nerve cells and the blood-vessels of the cortex cerebri, partly to defective metabolism, the result of the liver change, and partly to 'uræmia' (using the word in its broadest sense), the result of the kidney change. Finally, the weakness is due to fatty degeneration of the heart and voluntary muscles.

The jaundice in these cases does not, I believe, differ from ordinary obstructive jaundice, except that the bile which produces it is the final product of a liver whose power of physiological work is rapidly decreasing. The obstruction to the bile duct is produced partly in the duodenum, where the swollen mucous membrane obstructs the entrance of the common bile duct, and partly, probably, by the swelling of the liver itself causing an obstruction in the smaller ducts. In the above case the bile pigment was plentifully present in the skin and the white of the eye. At the post-mortem examination it was found absent from the fæces in the colon. It is to be remarked, however, that there was no bile pigment in the urine, as shown by the negative result of the nitric acid test. The sample of urine examined (of which I shall say more presently) was obtained on the fifth day of the illness, and probably many hours after the bile-forming power of the liver had ceased. When you have obstruction to the duct of a healthy liver, the formation of bile is continuous, the jaundice gets deeper and deeper in tint, and the bile pigment in the urine increases in amount with the increasing depth of the yellow colour of the skin. In ordinary obstructive jaundice the yellow colour is detectable in the skin after it has disappeared from the urine, and so in this case the yellow colour remained in the skin when not a trace of pigment was to be found in the urine, the reason being that the liver ceased to form bile after the jaundice had been produced. Whether or not there was bile pigment present in the urine in the earlier stages I cannot say, but probably there was. Again, looking at the state of the kidneys, it is not to be wondered

at if the elimination of bile pigment by these gravely affected organs was deficient. It must be borne in mind that in all cases of jaundice the urinary bile pigment appears early, and usually disappears before the skin has cleared.

A few words about the urine ; and, in saying these few words, I must not forget to thank Mr. Ransom, who has very kindly subjected the sample of urine obtained to a careful chemical examination. The only sample of urine examined was obtained on the day before death. This must be borne in mind, and you must not assume that it is characteristic of all stages and all cases of phosphorus poisoning. When this urine was passed the patient was almost moribund, the liver had been changed to a (probably inert) mass of fat and incapable of proper function, and the kidneys were severely damaged. The quantity of urine obtained for examination was less than sufficient to fill an eight-ounce bottle, and of this only about three ounces were available for Mr. Ransom's investigations. It was turbid, with a considerable sediment, and of a golden yellow colour. It was very acid in reaction, and of high specific gravity (1,035). The acidity was also remarkably persistent, and was very manifest even four days after the urine had been passed. It was distinctly albuminous, and after boiling and standing the albumen formed about one-fifth of the bulk. The albumen was caused by the fatty change which the renal epithelium had undergone, a fact which was further indicated by the presence in the sediment of a few granular casts. The high specific gravity was not caused by urea, for, on testing by the hypobromite method, the amount of urea was estimated, first by Mr. Kitchin, the clinical clerk, and afterwards by Mr. Ransom, to be 0·5 per cent. only. Mr. Ransom very thoughtfully suggests in his report that, since the hypobromite method 'decomposes other nitrogenous bodies, this result was probably in excess of the truth, and,' he says, ' I failed to obtain any crystals of nitrate of urea. It is probable, therefore, that urea was almost if not totally absent.' This deficiency of urea has been noticed in other cases of phosphorus poisoning, and also in 'acute yellow atrophy of the liver,' a disease which bears the closest

resemblance to phosphorus poisoning, and of which I shall have more to say presently. The absence of urea is due partly to the inability of the patient to take nitrogenous or other forms of food, and partly also to the incomplete metabolism which results from the disorganisation of the liver. The urine contained sugar, which is a fact that I do not find mentioned in connection with phosphorus poisoning or acute yellow atrophy. It reduced Fehling's copper solution, and also gave a rich Madeira wine colour when boiled with liquor potassæ, and this after the albumen had been removed by filtration. After being fermented with yeast for thirty hours, liquor potassæ and Fehling's solution gave negative results, so that we may feel sure that sugar was really present. Mr. Ransom estimated the amount of sugar to be equal to three per cent. Seeing that the urine was passed when, as I have said, the patient was almost moribund, and when the liver was in an extreme state of fatty degeneration, it seems impossible that the sugar in the urine was due to an increased activity of the liver cells. The more probable explanation seems to me to be that, owing to the disorganisation of the liver, glucose passed from the alimentary tract and through the liver without undergoing any change, or, if the patient was fed upon peptonised arrowroot by the rectum, sugar may have found its way direct into the general circulation through the hæmorrhoidal veins.[1] There was, as I have said, a considerable sediment to the urine, and microscopically this sediment was found to be formed of (1) a *very large* quantity of epithelium, mainly from the vagina ; (2) a few granular casts from the kidney ; and (3) a very copious deposit of acicular crystals, arranged in bundles and occurring in small groups and singly. These were at first supposed to be tyrosin, and a well-known authority on the urine, to whom I showed them, pronounced them to be tyrosin. When dealing with a rare deposit like tyrosin, one with which even the most experienced cannot be very familiar, it is never advisable to

[1] Dr. Hurd Wood informs me that it was found impracticable to administer nutritive enemata, so that the sugar cannot be accounted for by this theory.

trust to form only as a means of diagnosis. Mr. Ransom left a portion of the urine to stand for twenty-four hours with a quarter of its bulk of strong hydrochloric acid, when it was found that the crystals, instead of being dissolved, were even more numerous ; 'moreover, they were accompanied by and merged into larger crystals, which were obviously those of uric acid.' These larger crystals had the colour of uric acid, but the small acicular crystals seen in the urine were very faintly tinged. The crystals were in reality crystals of uric acid, and one of my colleagues informs me that he has seen fine acicular crystals of this kind in diabetic urine. The urine, however, contained tyrosin, which was shown by the following method adopted by Mr. Ransom : 1. The strongly acid urine was filtered to remove uric acid, then neutralised and left to stand under four times its bulk of methylated spirit. A precipitate formed, and tyrosin crystals were obtained both from the precipitate and the supernatant fluid. 2. Crystals of tyrosin were also obtained from the original urine. Crystals of tyrosin were also formed from a decoction of the liver, which was first treated by basic lead acetate, and the filtrate subsequently treated with sulphuretted hydrogen to free it from lead, and evaporated to a syrup. In this way the characteristic crystals were obtained by further evaporation on glass slides, and the syrup gave a pink colour with Millon's reagent. No crystals of leucin were found in the liver or urine. The presence of *sarcolactic acid* was, however, demonstrated, and it is possible that the persistent acidity of the urine may have been due to this body. The condition of this urine affords much food for reflection. The ultimate stage of the metabolism of nitrogenous matter is, in health, urea ; and the chief place where urea is formed is probably the liver. The diminution or absence of urea has been noticed by Parkes in cases in which a great destruction of the liver has taken place as the result of cancer or suppuration, and especially has it been noted in cases of acute yellow atrophy. In place of the urea, we find in these cases bodies which are the result of incomplete metabolism—such as uric acid, tyrosin, and leucin. Whether these bodies are formed in the liver

instead of urea, as the result of an interference with oxidation, or whether, being formed by the spleen and pancreas they merely pass through the inert liver, is an open question, but I confess that in the present case the latter seems to me the more probable explanation. We have seen how grave and widespread are the consequences when a healthy person takes a small quantity of pure phosphorus : vomiting, hæmorrhages, jaundice, headache, pain, cramps, albuminuria, coma, and death in five days, without fever ; and post-mortem evidence of extreme fatty degeneration involving the liver and other glandular and muscular tissues. A point of great interest in phosphorus poisoning is the complete similarity, both clinically and pathologically, to that very rare disease which is described in our text-books as 'Acute yellow atrophy of the liver'— a disease which is characterised by vomiting, hæmorrhages, jaundice, headache, pain, cramps, albuminuria, coma, and death, usually within five days, without fever ; and post-mortem by fatty degeneration of the liver, heart, kidneys, and other organs. It has been sought to distinguish these two diseases in various ways. For example, it has been said that in acute yellow atrophy the liver is small, while in phosphorus poisoning it is big. To this rule, however, there have been found many exceptions, and, in fact, the conditions have at times been reversed. It has been asserted that histologically the livers in the two conditions vary, but now it is admitted that in both the predominant change is fatty degeneration. It was at one time thought that leucin and tyrosin were not found in the urine in phosphorus poisoning ; but this, again, has been shown not to be true, and the case I have narrated is an example. Cases of phosphorus poisoning have been diagnosed as acute yellow atrophy. I believe I am right in stating that a mistake (if mistake it can be called) was made a few years since in this hospital, owing to the patient concealing the fact of having taken phosphorus, and a similar mistake was made some years ago at Netley. In short, I think there can be no doubt that, clinically and pathologically, the two conditions are indistinguishable. There are some other facts which tend to bring these two conditions (if they

be two) together. Acute yellow atrophy is a very rare disease. Thierfelder, who writes the article upon it in Ziemssen's Encyclopædia, and who, with characteristic German thoroughness, has ransacked medical literature for his material, has to found his article on 143 cases only, scattered over a period of forty years. Like phosphorus poisoning, it is a modern disease, and its history is about co-extensive with that of phosphorus poisoning, the first cases having been described by Rokitansky, of Vienna, in 1842, or nine years after the establishment of the lucifer match industry. Whether, as phosphorus poisoning has become more common and better understood, acute yellow atrophy has become less frequent, I do not know, but it is certain that the rarity of the latter condition is at least as great as ever. Acute yellow atrophy, it will be urged, differs from phosphorus poisoning in the fact that it is more common in women than in men. Of Thierfelder's 143 cases, 49 were males and 86 (or just 60 per cent.) were females. In the five years 1876–80 there were in this country (according to the Registrar-General's returns, as quoted by Mr. Wynter Blyth) 46 deaths from phosphorus poisoning. Of these 46, 17 were males and 29 were females, the females constituting 63 per cent. of the total. Of 56 adult cases collected by Falck, 43 were women and 13 men ; and of 71 suicidal cases collected by Otto Schraube, 47 were women and 24 were men. Again, acute yellow atrophy has often occurred in pregnant women. The same may be said of phosphorus poisoning. Thus Hamberg, at the meeting of Swedish physicians in 1881, stated that of 19 fatal cases of phosphorus poisoning collected by him in Sweden, 16 were in women mainly between twenty and thirty years of age ; and of these 16, 15 were pregnant—the phosphorus being presumably taken to produce abortion. Jäderholm has also published tables showing the increase of phosphorus poisoning in Sweden. He has made post-mortem examinations of 15 cases ; of these 15, 3 were men and 12 women ; of the 12 women, 2 were pregnant and 3 had recently aborted. In this connection I may quote Hessler, who says that phosphorus has a great reputation as an emmenagogue and abortifacient.

Thus it appears to be a fact that phosphorus poisoning not only has the closest resemblance to acute yellow atrophy, both clinically and pathologically, but that it still further resembles it in the great proportion of women (and especially pregnant women) who suffer.

Why are women more liable to suffer from phosphorus poisoning than men? In the first place, it may be urged that when women wish to commit suicide they adopt drowning or poison, while men more frequently make use of hanging or firearms. Dr. Ogle has pointed out that women, when bent on suicide, are less careful than men to select painless poisons, nearly 50 per cent. of female suicides by poison in England during the years 1863–82 being effected by strychnia, vermin killer, carbolic acid, and oxalic acid, while 60 per cent. of men employ prussic acid, laudanum, and other comparatively painless poisons. Again, it is evident that accidental poisoning and suicidal poisoning are very likely to be effected by poisons which are at hand, and phosphorus paste is, so to say, a household poison used by women and bought by women for the destruction of rats and beetles, while poisonous matches are, or were, everywhere to be found. Why should pregnant women suffer from phosphorus poisoning more than others? In the first place, it is probable that phosphorus is taken to cause abortion, and I think it will be found that many of the women who have died of phosphorus poisoning were not married, and were persons to whom pregnancy meant disgrace or inconvenience. Of the 47 female cases collected by Otto Schraube (as quoted by Wynter Blyth) 12 were prostitutes. The girl whose case I have been dealing with, and also her sister, who died in the same way, were 'kept women.' A case recorded in *The Lancet* in 1882 by Dr. Capon occurred in a 'kept woman'; and I have heard of a case very recently, the details of which were given me by a well-known practitioner in the south of London, in which the victim was also in the same condition of life. Thus facts seem rather to point to the conclusion that phosphorus poisoning is prone to occur among women of dissolute life, and it is possible that the traditional use of phosphorus as an aphrodisiac may have

some connection with this fact. Among the causes which predispose to acute yellow atrophy are mentioned (in addition to female sex) mental worry, dissolute habits, syphilis, and pregnancy. This rather points to the fact that acute yellow atrophy, like phosphorus poisoning, is prone to occur among women of dissolute habits. Dr. Fagge and other writers on acute yellow atrophy quote Graves's well-known case of three members in the same family, sisters, who died of the disease within eleven months. But in phosphorus poisoning there is also occasionally a family predisposition. I may remind you that, according to the evidence given at the inquest, the sister of my patient had previously committed suicide with phosphorus, and it was whispered—but only whispered—that the mother had met her death in a similar way.

The question will naturally arise, 'Are all or the majority of cases of acute yellow atrophy really cases of phosphorus poisoning?' It is not possible to answer this question, but the conditions being clinically indistinguishable, there can be little doubt that the latter condition has often been mistaken for the former. It must be remembered that suicidal patients are often very reticent, and when really bent on suicide they have every reason for concealing the fact that they have taken poison. If phosphorus has been given with murderous intent, the fact will certainly be concealed. Again, phosphorus may be taken accidentally, and a drunken woman would not be unlikely to take phosphorus paste spread on bread if such had been prepared for the destruction of vermin. Again, it is possible that the flesh of animals, and especially chicken, who have died from eating vermin which have met their death by phosphorus, may be the cause of the symptoms in human beings. But we must not forget that other bodies besides phosphorus are capable of causing fatty degeneration, and among these are arsenic, antimony, chloroform, and the mineral acids. The fact that in acute yellow atrophy the liver is especially affected points to the conclusion that the poison, whatever it may be, is probably absorbed from the alimentary tract. The liver may atrophy rapidly under certain other conditions. Thus, Dr. Murchison, in his classical work

on the liver, records two cases of 'acute atrophy' which occurred in his own practice. One was that of a tailor, aged sixty-six, who was in the Middlesex Hospital for twenty-five days, and in whom, post-mortem, was found an atrophied and fatty liver, with the common bile duct completely blocked by gall stones, with great dilatation of the bile ducts and some purulent deposits both in the liver and kidneys. The patient was febrile except for the last three days of life. This was clearly not a true case of acute yellow atrophy. The other case given by Dr. Murchison occurred in a girl who had been ill for a month. In this case there was acute peritonitis, and she was febrile to the last. This, again, was not an ordinary case of acute yellow atrophy. In short, acute yellow atrophy is a very rare disease indeed, and I think we shall all do well, whenever we feel justified in making such a diagnosis, to be exceedingly careful to investigate the possibilities as to the patient having had phosphorus administered either intentionally or accidentally. Phosphorus poisoning is of great interest as an example of profound constitutional disturbance occurring as the result of the ingestion of a chemical poison and running a fatal course without fever. In these days, when infective microbes are demanding a very large amount of our attention, it is well to be reminded that such things may happen.

The other case I have to mention (and I am enabled to do so through the kindness of Dr. Ringer, under whose care the patient was admitted) need not detain us long. A married woman, aged thirty-eight, took phosphorus paste on bread at 8 P.M. on Sept. 14, and she was admitted to the hospital within an hour. A powerful emetic was at once administered, and she vomited in ten minutes. The breath and vomit both smelt of phosphorus, but there was no luminosity. The stomach was then washed out by the stomach pump, and a drachm of old commercial turpentine was administered at 10 P.M., 11 P.M., 2 A.M., 4 A.M., and 7 A.M., and half a drachm at 9 A.M.—$5\frac{1}{2}$ drachms in all. Vomiting and retching continued, with burning pain in the stomach and great tenderness of abdomen, and especially of the hepatic

region, going through to the back. This persisted through the next day, and at 3 P.M. drachm doses of French turpentine were again ordered. There was diarrhœa, and one of the stools contained altered blood. This patient seemed very sensitive to vibrations, and complained that persons walking about the ward jarred her. The temperature was 100° when admitted, and then became normal. The urine never contained albumen or sugar, but contained a fair percentage of urea. The blood, when examined under the microscope, appeared normal. She gradually recovered, and left the hospital on Sept. 26.[1] There can be little doubt that this patient owes her life to the active treatment pursued. The phosphorus had not been long taken when she was seen, and probably no great amount had been absorbed when the stomach was so promptly emptied of its contents by the emetic and stomach pump. The administration of turpentine was, however, probably conducive to the favourable result. This is regarded as an antidote to phosphorus ; it appears to render it inert ; and we get a clue to its mode of action from the fact that the vapour of turpentine destroys the luminosity of phosphorus.

[1] This patient was readmitted a few weeks after her discharge, and was found to have slight albuminuria. She made a good recovery, and was again discharged.

CHAPTER IX

CHRONIC LEAD POISONING [1]

In Ward 8 you have frequently seen a patient who was admitted to the hospital on September 26, 1895, suffering from obstinate constipation and great muscular weakness. He is a painter, aged 34, and stated on admission that his bowels had not acted for ten days, and he informed us that on three previous occasions (in 1879, 1888, and 1891) he had suffered from obstinate attacks of 'colic' and constipation. As he is a painter by trade, and has a well-marked blue line upon the gums, we have little hesitation in attributing his troubles to lead poisoning. He thinks the present attack was determined by his working, during very hot weather, at painting the vaulted ceiling of a room, all the windows of which were closed.

The attack of colic in September was accompanied by other symptoms from which he has not previously suffered. He stated that for three weeks prior to his admission he had been feeling weak and ill, and three days before his admission he found that, owing to weakness in his arms, he could not lift a cup to his lips.

On admission his complexion was sallow and muddy, and he was much emaciated. He could not extend the wrist, nor, owing to weakness of the deltoid, could he raise his arm. He could not as he lay in bed change from the recumbent to the sitting position without assistance ; he straightened his back with difficulty, he could not stand without help, and was quite unable to walk.

In short, every muscle of the limbs and trunk was in an extreme degree of paresis. All the muscles were soft and

[1] Clinical Lecture, U.C.H., 1895.

flabby, and to a certain degree wasted. I use the term paresis instead of paralysis, because every muscle seemed to be capable of a certain degree of feeble movement, and all of them responded to the faradic current. There was no extreme wasting of any muscles, except perhaps the erector spinæ on the right side. The face rather lacked expression, but no definite paralysis of face or lips or ocular muscles could be detected.

He feels when he is touched, and can tell when he is pricked with a pin, but nevertheless I doubt whether his sense of pain is very acute, because, when the hair of the leg was suddenly pulled he did not flinch to the degree which is usual in a normal subject.

All the nerve-trunks in the upper limbs are exceedingly tender, and it is evident that the nerve-trunks are much more tender than the muscular masses.

When I first saw this patient I was much struck by his voice, which was feeble and toneless, without being hoarse or stridulous. The voice was of a character to excite suspicion of laryngeal paralysis, and on examination with the laryngoscope we found well-marked immobility of the left vocal cord, which was not adducted to the middle line during phonation.

The urine since his admission has been of average quantity but pale, of low specific gravity (fluctuating between 1,014 and 1,008), and up to November 5 contained a trace of albumen, which has not been detected since that date. The urine, kindly examined by Dr. Kenwood on January 9, gave evidences of a slight trace of lead only.

The temperature has been normal except for a few days, when he suffered from slight tonsilitis.

The hæmocytometer on October 1 showed that the red corpuscles were about eighty per cent. of the normal.

For the first week of his admission he suffered from constipation and abdominal pain, but since that time the alimentary tract has afforded no evidence of disease.

In some respects this is not quite a typical case of lead poisoning, although there are no symptoms which have not been caused by lead again and again.

As to the paralysis from lead, you are most of you probably aware that the typical form of lead paralysis is 'wrist drop,' in which the extensors of the fingers and wrists are paralysed on both sides. As a rule the paralysis from lead is limited to the forearms, and to the muscles named. The supinator longus and the intrinsic muscles of the hand usually escape. The paralysis is generally symmetrical, but while both limbs are implicated, it is usual to find that the right arm is affected earlier, and rather more severely, than the left. The affected muscles waste rather rapidly, and in a severe case of long standing give, when tested with electricity, the reaction of degeneration.

With regard to this 'Reaction of Degeneration,' please remember that the important fact to bear in mind is that the muscles do *not* respond, as normal muscles do, to the faradising current, while they respond with undue readiness to the continuous galvanic current.

The non-reaction of the muscle to faradism is said to be due to the degeneration of the intra-muscular nerves. This explanation is probably correct, but it is well to remember that a paralysed muscle which is recovering will sometimes give the reaction of degeneration at a time when it is responding to the stimulus of the will, i.e. it is the seat of voluntary contraction when artificial contraction cannot be evoked by faradisation. This may be due to the fact that the intra-muscular nerves have regenerated on the under-side of the muscle, where the faradic stimulus is not easily applied. Why these muscles should show an *increased* irritability to the galvanic current it is difficult to say, and still more difficult to say why the reaction to the galvanic current shows a *qualitative* as well as a *quantitative* change. This qualitative change consists in the fact that the muscle reacts to anodal opening or anodal closure before it responds to cathodal closure, which is the first reaction to be obtained in health. These qualitative changes are interesting, but their importance from the clinical point of view is as yet not understood, and I want you to cling to the main fact of these reactions of degeneration, which is that *the muscle which does not respond to faradism, responds*

readily (sometimes too readily) to galvanism. The degeneration of the intra-muscular nerves is brought about by extinction of the motor cells in the anterior cornua, so that no stimulus can reach the muscle from the brain, or by a block in the nerve itself due to neuritis, morbid growth, extravasation of blood from bruising, or the actual division of the nerve. So soon as a motor nerve is severed or blocked by the processes named, degeneration begins to extend centrifugally until it reaches the muscle. In mixed nerves which serve both sensory and motor functions, a blockage of any kind occurring in the trunk probably produces a certain amount of centripetal degeneration, as well as centrifugal ; but this latter is far more difficult to demonstrate.

The patient whose case we are considering is not to be regarded as a typical example of lead paralysis. In him the paralysis is of very wide extent, but it resembles other cases of lead paralysis in this, that it began in the forearms, and that the deltoid muscles are among those which are most profoundly affected. Scarcely a muscle in his body has escaped ; all are feeble in an extreme degree, but none appear to be totally paralysed, and all of them, which are accessible, respond to a *strong* faradic current.

The paralysis of one vocal cord is to be regarded as exceptional. Cases of such paralysis from lead are recorded, but I do not recollect having personally observed it before. Naunyn states that in horses which work in lead mills paralysis of the abductors of the vocal cords is common, and that, not unfrequently, this necessitates the performance of tracheotomy. Paralysis of the left vocal cord in an adult is a physical sign which raises a strong suspicion of aortic aneurysm ; but repeated examinations of our patient to this end have always given negative results.

How profound is the change produced in the human body by the continued introduction into it of small quantities of lead ! Lead holds an important position in the Pharmacopœia on account of its styptic qualities, and is frequently administered in cases of internal hæmorrhage. It has a reputation also as a valuable astringent when applied externally,

and the solutions of the salts of lead are probably antiseptic to a considerable degree. This poisonous metal is retained in the Pharmacopœia for its styptic and astringent qualities— qualities which are due to its stimulating action upon the muscular coats of the small arteries and arterioles. It is said to have a similar action upon the muscular coats of the intestines, and to cause in this way the phenomena of 'painter's colic.' Our patient on admission had been constipated for ten days. His intestines, and probably his arterioles, had been the subject of a muscular spasm so sluggish and inert as to be regarded as permanent. When the full styptic effect of lead has been produced the patient is in great danger if its administration is continued. It seems probable that when lead produces a toxic effect the calibre of every arteriole in the body is diminished. If this be the case, can we wonder that the nutrition changes are profound? Absorption from the alimentary tract is diminished, the elimination of waste products by the kidney is seriously checked, and the starved tissues tend to degenerate, so that in many places we find that the connective tissues tend to increase at the expense of gland-cells and nerve-tissue.

Dr. Thomas Oliver, of Newcastle, in his Goulstonian Lectures on 'Lead Poisoning,' gives an excellent series of drawings of the histological changes in lead poisoning. In the kidneys the shedding of the epithelium of the tubules is accompanied by interstitial overgrowth of connective tissue; in the liver cirrhotic changes occur; the fibres of the nerve-trunks are pressed upon by the fibroid overgrowth; and similar changes also occur in all parts of the body. The kidneys are very liable to be affected in lead poisoning. However the poison be introduced, whether by the stomach, by inhalation, or by the skin, the kidneys must perform an important part in eliminating it, and the contraction of the renal vessels seriously damages the organ, and thus its physiological action of eliminating the end products of metabolism is damaged, and we are liable to get gouty symptoms in those who are otherwise predisposed to gout, and not unfrequently uræmic symptoms, inclusive of epileptiform convul-

sions. It is evident that when the kidneys are damaged, the danger caused by the poison circulating in the blood is enormously increased. It is also evident that if a worker in lead acquires a granular interstitial nephritis, the danger he runs from his occupation is increased, and his liability to suffer from recurrent attacks of lead poisoning is very great. The really operative dose of a poison is the balance between the amount absorbed and the amount eliminated. If elimination keeps pace with absorption no symptoms are caused, but if elimination be slower than absorption, or if elimination be checked by disease of the kidneys, then, as we all know, very small doses of such poisons as mercury and opium are liable to produce most untoward results.

The physiological action of lead salts being to cause contraction of the vessels and intestines, it almost necessarily follows that so soon as absorption takes place elimination is checked, and in this fact we find the explanation of the insidious nature of lead poisoning. There seems to be no quantity of a lead salt too small to produce symptoms, *provided it is taken continuously.*

In the patient whose case we are considering there is no history of any attacks of gout, but it is well known that workers in lead are very liable to gout, and that sufferers from gout are peculiarly intolerant of lead. It was Sir Alfred Garrod who first demonstrated in this hospital that the administration of a few grains of lead acetate to a gouty patient almost invariably produced a paroxysm.

This liability of the lead worker to suffer from gout has not been observed in Edinburgh to the same extent as in London, and lately Dr. T. Oliver has shown that the lead workers of Newcastle, and the northern towns generally, enjoy a similar comparative immunity from gout. It used to be thought that the difference in the liability to gout in North and South was to be accounted for by the fact that while the northerners stimulated themselves with non-gouty whisky the southerners used beer, the reputation of which as a gout-producer is considerable. This difference in habits may partly account for the difference in pathological tendencies,

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but that it is not the sole explanation seems certain from the work of Dr. T. Oliver and others. The true explanation of this strange difference is still to be sought.

The symptoms produced by lead are protean, and there is not a single symptom which is constant unless it be the sallow pallid complexion. The anæmia produced by lead is extreme, and the presence in the blood of a very small quantity seems to cause a rapid diminution of both hæmoglobin and red corpuscles.

The lead, after absorption, circulates with the blood, and its effect seems to fall upon different organs in different individuals. We all have our vulnerable points, and these are not the same in any two persons. There seems nothing unreasonable in supposing that any part of the body which has been damaged, and the nutrition of which has been impaired by previous disease or injury, is likely to constitute a vulnerable point. We know, also, that persons have a family tendency towards chest diseases, vascular degenerations, insanity, &c. Did time permit one could bring forward many familiar examples of these propositions. Occasionally it may happen that the parts most used are the ones to suffer most, and this explanation has been offered of the tendency of painters to suffer from wrist-drop. The tendency of different individuals to be differently affected by lead caused Tanquerel des Planches to classify his cases, and to speak of (1) lead colic, (2) lead paralysis, (3) lead arthralgia, and (4) lead encephalopathy. Lead arthralgia, which is not very common, would probably resolve itself nowadays into gouty pains, and pains due to spinal sclerosis and neuritis. Lead encephalopathy (headache, convulsions, tremor, optic neuritis, and atrophy) may be primary, but is more probably, in most cases, secondary to chronic kidney trouble.

What we have to remember is the great fact that the symptoms of chronic lead poisoning are varied and far from uniform, and, in short, that lead may be the unsuspected cause of chronic troubles affecting nutrition generally, or the digestive, renal, or nervous organs.

Many poisons manifest a peculiar affinity for the cells of

certain tissues, which stand in relation to the poison in a position analogous to that of preference shareholders in a trading company. Few, if any, poisons, however, have an *exclusive* affinity for the cells of any tissue, and it is usual to find that after those cells for which the poison has a 'preference' have been supplied other cells in the body begin to get some of the dividend. Lead is more impartial in the bestowal of its injuries than most poisons, and although 'lead colic' is the commonest symptom, we have seen how insidious is the action of lead, and how it may affect every organ in the body. It has been said that 'lead has a peculiar affinity for nerve tissue, but analyses made on the bodies of animals which have succumbed to lead poisoning show that it is universally distributed, and that every part of the body can be made to yield lead.' The distribution of the lead in the body varies considerably.

As lead poisoning is insidious in its onset and very protean in its manifestations, it becomes important to consider what symptoms, if any, are to be regarded as pathognomonic. The blue line which appears on the margin of the gums next the teeth, often spoken of as the lead-line (and which is due to the precipitation of lead sulphide in the tissues of the gums, by the action of the hydrogen sulphide formed by the decomposition of albuminous food particles in the mouth), is the most valuable aid to the certain diagnosis of lead poisoning. Other things being equal, the patient who generates most hydrogen sulphide in his mouth will have the best marked lead-line; while those whose mouths are inapt, either by design or accident, for this decomposition, may suffer from lead poisoning without any lead-line upon the gums. Persons who have no teeth, or those who are blessed with very regular teeth which they keep scrupulously clean and which afford few opportunities for the lodgment of food-particles, may fail to afford the evidence of a lead-line, even though they be saturated with lead. On the other hand, a lead-line once formed may persist, it is said, after recovery has taken place.

Without forgetting the exceptional conditions named, we

may say that a blue line on the gums is a very sure evidence of lead poisoning.

The important point to bear in mind is that the absence of a lead-line does not exclude lead poisoning.

Again, the onset of lead paralysis of typical form (paralysis of extensors of wrist and fingers, without implication of the supinator longus) in persons who have previously suffered from colic is a combination of circumstances which justifies us in regarding the case as one of lead paralysis, even in the absence of a 'lead-line,' and in the further absence of any assignable cause.

Even if there be no lead-line, and if the paralysis be not of a typical kind, we are justified in assuming lead to be the cause of the trouble if the patient's occupation is one which brings him into intimate and daily contact with poisonous salts of lead.

A case of great interest in this connection was under my care in 1894.

The patient, E. S. H——, aged 35, a packer of furniture, was admitted on September 27.

He had previously been admitted twice under Dr. Ringer, on July 5 and August 18, suffering from abdominal pains, constipation, and albuminuria, and on both occasions he left the hospital cured of his abdominal trouble, and with his albuminuria reduced to a 'trace' of albumen.

When admitted under my care he was again suffering from abdominal pain, vomiting, and constipation. His urine was of low specific gravity (1,012), containing one-third albumen, and there were superadded paralytic symptoms, affecting mainly the arms.

On electrical examination we found that the deltoids gave degenerative reactions, and were absolutely powerless, so that the patient could not raise either arm to the level of the shoulder.

The extensors of the wrist and fingers responded only to the strongest faradic currents, while the flexors of elbow, wrist, and fingers, and also the supinators longi and the interossei

muscles responded normally to faradism, but were obviously paretic.

The patient complained of pain in both arms, and the nerve-trunks were everywhere tender, especially in the axillæ. There was some impairment of sensibility to pains, but tactile sensibility appeared normal. The legs were paretic, but there was no actual paralysis. The patient walked fairly well, but feebly, and the reflexes were normal. There was no lead-line, and the teeth were exceptionally clean. There was a distinct history of two attacks of typical gout during the previous year. The patient had been a moderate beer drinker, but he had not the aspect of a drunkard, and he persistently denied that he had ever taken alcohol to excess. He had drunk tea rather to excess. He made a fairly good recovery, and, after a sojourn at Eastbourne, and a few weeks at the Mineral Water Hospital, Bath, he returned to his work.

At the end of June, 1895, he paid his fourth visit to the hospital, but on this occasion his symptoms were mainly abdominal, and there was no marked recrudescence of his paralytic troubles. The urine, however, was still of low specific gravity, and contained a trace of albumen.

Here, then, was a case in which there was no lead-line, and in which there was no obvious risk of lead poisoning in the patient's occupation or surroundings, but which, nevertheless, we have felt justified in regarding as one of lead paralysis from neuritis. The justification of this diagnosis is found in—

1. The form of the paralysis, which is more typical of lead than of any other form of multiple neuritis.

2. The absence of other causes of multiple neuritis. It is true we have never discovered the cause of the lead intoxication in this case ; but of all intoxications which give rise to neuritis, lead is apt to be the most insidious and the most difficult to discover.

3. The history of four attacks of colic, one of which immediately preceded the onset of the neuritis, give a very strong support to our diagnosis, because recurring attacks of colic without evidence of any cause of permanent intestinal

obstruction, and without rise of temperature or distension of the abdomen, are extremely characteristic of lead poisoning.

4. The history of two attacks of gout, and the occurrence of albuminuria tending to become permanent, also lend support to the diagnosis of lead poisoning.

5. Finally, his recovery in hospital on four different occasions, and the immediate recrudescence of his troubles when he returns to his business and domestic surroundings, is a circumstance which points almost conclusively to the fact that his troubles are toxic, and that they are not autotoxic. Therefore the probability that they are caused by lead is very great.

One of the points in connection with lead poisoning, which has been much debated, is the seat of the lesion which produces lead paralysis ; and while some have placed the lesion in the spinal cord, others have contended that the symptoms are compatible with a neuritis.

The fact that the paralyses are generally symmetrical might cause one to infer that the lesions are spinal, but a little reflection will serve to convince us that arguments based upon this ground are untenable. I may remind you that un-doubted anterior polio myelitis—spinal or infantile paralysis— is more often unilateral than bilateral in distribution, and that the undoubted neuritis which occurs from alcoholic, or as the result of diptheritic, poisoning is almost always bilateral and symmetrical; in short, that while some undoubted spinal conditions are unilateral, some undoubted neurites are bilateral. The poison with which we are dealing circulates in the blood, and there is no more difficulty in assuming that corresponding nerve twigs on the two sides of the body suffer than there is in assuming that both anterior cornua at the same level should be attacked. Again, there is no more difficulty in assuming that certain nerve twigs are attacked than there is in assuming that the motor cells of the cervical enlargement at a certain level are selected for attack in preference to those above and below them.

If we admit that certain cells of the body stand in relation to certain poisons in the position of preference shareholders—

which seems to be an undoubted, although an inexplicable fact—we must concede that such preference may be manifested towards cells at the periphery equally with those situated at the centre. If we accept so much, then there is no difficulty in admitting that nerve twigs supplying muscles may suffer in preference to nerve twigs and end organs conveying impressions from the skin.

One must admit that it may be impossible, in certain cases, to say whether the paralysis and wasting of a muscle be due to destruction of a group of cells in the anterior cornu or destruction of or pressure on the axial cylinders of certain nerves, or whether the second condition has followed as a consequence of the first.

The presence of pain in the limb, combined with tenderness of nerve-trunks and muscles, undoubtedly points towards neuritis.

I think, however, that that which should make us regard lead paralysis as due to implications of nerve twigs rather than motor cells is the fact of recovery. A large number of cases of lead paralysis, if the cause be removed, get perfectly well ; and as it is an admitted fact that the regeneration of a motor nerve is common, while the regeneration of motor cells, if it ever take place, is excessively rare, this fact of recovery may almost be regarded as excluding much damage to the anterior cornua.

But it is not reasonable to assume that lead or any other poison has an exclusive preference for any particular organ or area, and one must be ready to admit that lead may inflict a damage upon the spinal cord or brain just as it does upon motor nerves, kidneys, liver, or intestines.

If we come to the conclusion that any case of recurrent abdominal trouble or paralysis is due to lead, then, assuming this conclusion to be correct, the prevention and treatment of the condition is within our grasp if we can succeed in detecting the source of the poison. The insidious and dangerous effects of lead seem to have been known to the Romans, and Vitruvius, the great Roman writer on architecture, mentions the danger of using leaden pipes for the conveyance of water.

It is to be presumed, therefore, that the knowledge that lead-poisoned water will produce colic is very old. Vitruvius was also acquainted with the use of ' red lead ' for a pigment, but I have been unable to discover who first pointed out that painters who used lead paints were liable to colic. From the six-teenth century downwards descriptions of colics which were endemic in certain countries have been given by various physicians. One of the first writers was Francis Citois, a native of Poitou, and physician to Cardinal Richelieu, who described the *colica pictonum*, or the colic of the inhabitants of Poitou. Please note that the word *pictonum* has no relation to *pictor*, a painter. There is no evidence that Citois knew anything of 'painter's colic,' and he attributed the colic of Poitou to the acidity of the local wine. Similar endemic colics had been described in Normandy among the cider drinkers, and in this country an endemic colic was described as occurring among the cider drinkers of Devonshire, first by Musgrave, in 1703, and secondly by Huxham, who in 1739 published a treatise, ' *De Morbo Colico Damnoniorum*.' These writers had no notion as to the real cause of the colic, as a reference to the pathology of Huxham will show. Huxham attributed the colic to the acidity of the cider, and he says :

'By long and frequent drinking of a liquor of this kind, such a quantity of crude, gross tartar is thrown into the blood that it thence becomes very acrid ; and not only the blood, but, from that impure source, all the humours thence secreted. So that, instead of a very soft lubricating mucus, separated by the glands, discovered by Dr. Havers, we have, as it were, a sharp coagulated matter, whence arises great pain in the joints and impotence of their motion. Instead of an exceedingly soft lymph to moisten the nerves, a corrosive ichor ; and hence epileptical attacks. . . . At length, even the very bile, that variously useful balsam of the body, becomes corrupted and quite enervated by the superabundant apple-acid, though in its natural state it was designed to correct acidity.'

I give you this as a very good sample of the pathology of 1739, but you will observe there is no mention or hint of the true cause of the Devonshire colic.

It was not until 1767 that Sir George Baker conclusively proved that the Devonshire colic was caused by lead poisoning in a masterly communication to the Royal College of Physicians, on June 29, entitled an 'Essay concerning the Endemial Colic of Devonshire.' Baker showed—

1. That acid drinks are not necessarily the cause of colic.

2. That the cider drinkers of Devonshire suffered more than the cider drinkers of Gloucester, Hereford, and Worcester.

3. That lead entered largely into the composition of the cider-presses of Devonshire, but not into those of the neighbouring counties.

4. He finally demonstrated that 18 bottles of Devonshire cider taken from his own cellar contained over four grains of metallic lead.

Not only in Poitou and Devonshire did these endemic colics get a footing, but in tropical countries as well. The 'dry belly-ache' of the West Indies, and of some of the Southern States of North America, was thought to be a climatic disease until it was shown to be due to the contamination of rum by lead. Madrid was much troubled with an endemic colic, as were also the sailors in the French navy, but both these 'endemics' have now been shown to be caused by lead poisoning, that in the French navy being attributed to the use of lead in the apparatus for distilling water. I may further remind you that in the old bills of mortality, issued by the parish clerks of London, 'griping in the guts' was a common cause of death, and there can be little doubt that a fair proportion of these were cases of lead poisoning.

I mention these facts in order that we may appreciate the services of Sir George Baker. By showing conclusively that Devonshire colic was really lead colic, he gave us the power of arresting all the so-called 'endemial' colics, and thus saved countless lives, and, what is far more important, prevented the crippling of useful lives.

Now that the symptoms of lead poisoning are well understood, and we are thoroughly alive to the dangerously insidious qualities of the poison, the question of 'lead' is sure to cross

the mind of the physician when confronted with cases in which the suspicion of lead is warranted.

In one of the cases which I have brought before you the suspicion of 'lead' is so strong as to amount to a positive conviction in my own mind ; but we have never been able to demonstrate the source of the lead, and in that important respect our duty towards the patient remains to be completed.

As regards the toxicity of the various preparations of lead, it is admitted that the salts of lead are far more dangerous than the metal. It is, indeed, a question whether metallic lead is to be regarded as a poison.

I remember hearing the late Mr. Hulke make allusion to the case of a gamekeeper who was troubled with that form of dyspepsia which, on account of the accompanying flatulence, the uneducated masses frequently characterise as the 'rising of the lights.' Whenever this gamekeeper was troubled with the 'rising of the lights' he took a charge of shot to, as he said, 'keep 'em down.' This gamekeeper is said never to have suffered from saturnine intoxication ; but we must remember that metallic lead must be liable to be converted into a lead salt by the acids of the stomach or by the acids taken with the food. The doctrine that metallic lead is not poisonous is, in all probability, a false doctrine for practical purposes, and in fact I was informed a few weeks ago, by Dr. George Oliver, of Harrogate, of a patient whose toxic symptoms were caused, he believed, by the practice of putting a shot in his mouth and idly chewing it.

By far the most important manner in which lead gains access to our bodies is by the contamination of drinking-water. The result of such contamination is that lead is taken whenever the water is drunk, or whenever hot infusions made with the water are drunk, or whenever soup or food which has been made with or cooked in the water is consumed. If, therefore, the domestic water supply be contaminated with lead (no matter how slight such contamination may be), some of the dwellers in the house are sure to suffer eventually.

Not all waters dissolve lead. One of the advantages of a hard water containing lime, such as we use in London, is that

it has little or no solvent power upon the leaden water-pipes and fittings. When we get our new supply of soft upland water from the Welsh hills we may have to alter our water fittings, but not till then. The conditions which favour the solution of lead in water are not completely understood, but it may be taken as an approximation to the truth that the purer and softer the water, the greater is its lead-dissolving power. Rain-water has great solvent power on lead : and in houses which are dependent upon rain for their water supply the greatest care must be taken to avoid the use of lead for water-pipes and gutters, storage tanks and service pipes. Waters which are acid in reaction seem to dissolve lead with a most dangerous facility, and the most recent investigations seem to show that the lead poisoning which has occurred in some of the Yorkshire towns during the last few years is caused by the acidity of some of the peaty waters with which they are supplied.

I have no intention of taking up your time by cataloguing all the possible sources of lead poisoning. Suffice it to say, that the lead may be absorbed from the alimentary tract, the respiratory tract, or the skin, and that the economic uses for lead and its salts are very numerous. Acid liquors must not be kept in leaden vessels, or in earthenware vessels finished with a lead-glaze, as we have learnt from the study of cider colics. Where lead poisoning is suspected, you must closely scrutinise the sources and surroundings of all food and drink. Food wrapped in lead—such as tea ; canned or tinned foods, hermetically sealed with a leaden solder ; soda-water syphons with leaden fittings ; tobacco wrapped in lead foil ; snuff adulterated with chromate of lead ; anchovy paste, or lozenges coloured with the same material, are a few among the many recorded instances of accidental lead poisoning by food.

That lead can be absorbed by the skin seems also to be established, and not a few cases of lead poisoning have resulted from the use of hair dyes containing lead, or from the application of pigments containing lead by theatrical artists for the purposes of 'making up.'

Lead poisoning is common to many trades—as the manufacturers of 'white lead,' and other lead pigments, colour-

grinders, painters, pottery-glazers, makers of glazed cards, &c. ; but it seems clearly established that those who are engaged in the manufacture of lead products, such as pigments, are more liable to suffer than those who are engaged in manufacturing articles from the pure metal.

I have said enough to show that lead is almost omnipresent, and that when you have reason to suspect lead poisoning, the source of it often requires diligent search, in which the assistance of a skilled chemist is necessary. One word of caution. A medical friend of my own, practising in the country, was confronted with several scattered cases of lead poisoning, and for a very long time he failed to discover the cause ; but finally he found it, as he thought, in certain packets of an article of consumption which was being rather freely distributed in his district. Lead was actually found, on chemical analysis, in the article contained in these packets, and, armed with this knowledge, my friend, acting as every doctor should do, in the best interests of his patients, gave a warning that the article in question was unfit for consumption. Thereupon the proprietors threatened him with an action for libel ; and, although the action was settled out of Court, it was the source of considerable worry and expense while it was pending.

I take it that the proper course to pursue in such a case would be to impart your suspicions to the sanitary authority confidentially, and allow them to deal with the matter, with the assistance of their analyst.

Finally, I have to speak of the treatment of lead poisoning.

Our first duty is, if possible, to discover the source of the lead and stop its administration, and no more need be said on this head.

But it often happens that it is impossible to change the occupation of the patient, and when that is the case we must adopt such measures as may commend themselves for lessening the risks of such occupation. The chief of these are cleanliness and ventilation. A painter or any other worker in lead should be scrupulously careful to wash his hands before eating, and to thoroughly cleanse his hair, body and mouth every day.

He should always work in washable overalls, and a clean suit should be put on at least once a week. If this be done the worker with lead pigments runs far less risk than if he inhabits for months together the same dirty paint-bedaubed suit of clothes, and is content to eat his meals with paint-bedaubed hands and to wear a mass of dirty hair. A moustache, if it be properly washed and cleaned, is probably a protection to the worker in lead.

Good ventilation is essential, and I would remind you that one of the patients, whose case I have related, attributed his attack to working in an unventilated room in hot weather.

Workers in lead must be well nourished. Dr. Thomas Oliver, of Newcastle, has made experiments which show clearly that saliva, gastric juice and bile, are all capable of dissolving lead, but that if starch be mixed with the lead, less of the lead salt is dissolved by the saliva ; if proteid be mixed with the lead, less of the lead is dissolved by the gastric juice ; and that if fat be mixed with the lead, less of the lead is dissolved by bile.

The conclusion is, then, that saliva, gastric juice and bile have no preference for lead in the presence of those alimentary principles upon which these three digestive fluids are designed to act. The well fed lead-worker runs less risk than the semi-starved, ill-fed worker, and the man who goes to his work with a stomach full of food is less likely to suffer from lead poisoning than he who goes to his daily toil on an empty stomach. Dr. Thomas Oliver is fully supported in this conclusion by the experience of the manufacturers of lead products.

Dr. Oliver is of opinion that all lead-workers should be total abstainers from alcoholic drinks, and this conclusion is supported by the well-known liability of lead-workers to suffer from kidney disease and gout.

As a prophylactic it has been the custom, in some lead works, to provide the employés with a 'lemonade' made with dilute sulphuric acid instead of lemon juice, with the object of converting the lead in the alimentary tract into the comparatively insoluble lead sulphate. Dr. Oliver supports this practice, but it must not be forgotten that lead poisoning has

resulted from taking lead sulphate, and that the constant administration of dilute sulphuric acid is apt to produce constipation, which, in the lead-worker, is very undesirable. It is stated, also, that when this acid drink is provided it is difficult to get the workpeople to take it continuously.

To get rid of the poison from the system and overcome the lead colic, the continued administration of magnesium and sodium sulphate, combined with potassium iodide, has given the best results. It is a good plan also to give the saline aperients mixed with hot water. Enemata may be employed with advantage. Belladonna is also a useful adjunct to the salines.

Lead is eliminated from the system by the bowels and the kidneys. Dr. Dixon Mann is of opinion that when lead is administered medicinally by far the greater part is eliminated by the bowels. Thus he gave two grains of acetate of lead to a patient on five consecutive days. On the fifth day the fæces yielded the equivalent of five grains of lead, and on the sixth day four grains, while one milligramme of lead was the largest amount eliminated by the kidneys on any one day. Whether the lead eliminated by the fæces had simply passed through the alimentary tract is doubtful, but I would remind you that arsenic, antimony and mercury appear to be excreted by the large intestine after absorption from the higher parts of the alimentary tract, and it is possible that the same process may take place in lead poisoning.

In Dr. Oliver's cases the largest amount of lead eliminated by the kidneys in a single day was ·17 grains, and the lowest amount recorded was ·002 grains. The daily eliminations fluctuate in a remarkable way, and occasionally the elimination of uric acid and lead appeared to bear an inverse ratio to each other.

It has long been the practice to administer potassium iodide (gr.ij to gr.v) in cases of lead poisoning, and Dr. Oliver supports this practice ; but it may be stated that Dr. Dixon Mann has failed to find that this drug increases the elimination by the kidney. Iodide of potassium is frequently administered with apparent success in cases of sclerosis and neuritis, no matter from what cause : and, seeing that lead

produces sclerotic changes in a most remarkable way, I think we are justified in continuing to administer potassium iodide in moderate doses.

Warm baths are of undoubted service in lead poisoning. They were in repute more than a century ago, and sufferers from Devonshire colic found benefit from the warm baths of Bath long before Baker showed that this colic was caused by lead. It is probable that soaking in warm water stimulates nutrition and metabolism, and there seems to be no doubt that it is useful in lead poisoning. Sulphur baths have enjoyed a great repute, but whether they are more efficacious than the 'indifferent' water of Bath is doubtful.

For the treatment of the paralysis the most important thing is to keep the limbs protected and warm. Warm baths are very useful, and a man who cannot move a paretic limb in the air may manage to do so when it is immersed in water, which helps to buoy up the limb and diminish the work of the muscles.

Massage and electricity are both useful in the treatment of lead paralysis, but neither of these agents must be employed so long as the nerves and muscles remain tender. If either of these agents be used in the painful stage of the paralysis they are apt to do harm rather than good. One of our patients was subjected to this treatment when the nerves were tender. The right arm and shoulder were daily rubbed by the clinical clerk, while the left arm and shoulder received no such treatment, and it is certain that in this case the left arm (which was not rubbed) seemed to recover power more quickly than the corresponding limb.

While the nerves are tender I think it is a good plan to apply strips of capsicum plaisters along the line of the tender nerves. These serve as a protection, and act also as a mild counter-irritant.

When the patient begins to improve, he is benefited by the administration of tonics, such as liquor strychninæ, combined with mineral acids. The diet should be light and nourishing, and finally the patient should be sent to a convalescent home before returning to his work.

CHAPTER X

FOOD-POISONING [1]

IN commencing these lectures on toxicology I told you that it would be necessary to impose some arbitrary limits on the range of subjects to be treated, and accordingly that we should not deal with those poisons which are the products of living microbes, and which more properly come within the domain of the pathologist. Food-poisoning, however, stands upon the borderland of toxicology and pathology. Food-poisoning is often so sudden in its onset and so rapid in its progress as to give rise to suspicions of foul play, and on that account comes within the range of the medical jurist, albeit that the *vera causa* of the trouble is in many cases not to be distinguished from that which gives rise to a true infective fever. One of the best-known forms of food-poisoning is *sausage-poisoning*, which has been studied in Germany, and has been called botulismus. Let us clear the way by stating that the form of 'sausage-poisoning,' of which I am about to speak, does not include infection with parasites, such as trichinæ or tæniæ, and you will soon perceive that it is something far more serious than an ordinary attack of gastritis from indigestible food. Most of the cases which have been described occurred in the Würtemberg Black Forest, where the incidence of the disease has often resembled that of a limited epidemic attacking whole families or small communities. Up to 1853, four hundred cases had been published, of which one hundred and fifty had been fatal. The sausages which have produced these troubles are of the kind often spoken of in this country as 'German sausages,' that is, sausages which are of large dimensions, and which are 'smoked' and not cooked.

[1] Part of Toxicological Course, U.C.L., 1896.

In the Würtemberg Black Forest it is customary to make such sausages of pig's meat, and to 'cure' them by smoking them in the chimney over a wood fire. It is stated that the sausages which have given rise to poisoning have been made and cured carelessly and in such a way as to encourage putrefactive changes. The danger is said to be greatest when the sausages are too moist and watery in consistence, due to the composition of the sausage or the unsteady drying of it when made. Accordingly, we find that sausages made with liver, brains, or blood, or to which milk has been added, have been particularly blamed ; also that very big sausages have proved dangerous because of the difficulty of drying them in the middle. Further, it is stated that sometimes the accident has arisen because the drying and smoking have not been properly carried out. If the fire go out and the sausages are allowed to freeze in the chimney and to thaw again, it is said that putrefactive changes are very liable to occur.

Let me remind you that 'sausage-poisoning' was well recognised before the knowledge, which we now have, of putrefactive and other organisms and their mode of cultivation was current. We now know that for the cultivation of a large number of these organisms fluid media are necessary, or media in which the proportion of water is relatively large. They flourish in sewage water, in hay-infusions, in the discharge from wounds, and in milk, and the media which are used for their cultivation in bacteriological laboratories consist of sterilised broths or broths mixed with just sufficient gelatin or agar-agar to make them firm at ordinary temperatures. 'German sausages,' to be safe, must be continuously *dried* in antiseptic smoke, and the conditions in which sausages, which have proved dangerous, have been found, are precisely those in which the growth of putrefactive and pathogenic organisms is likely to occur.

Some of the sausages when cut across have been evidently permeated with fungoid growths, and a micrococcus, called 'sarcina botulina,' was many years ago discovered in them. It is noteworthy that in these cases of sausage-poisoning an incubative period, ranging from eighteen to twenty-four hours,

has generally been described, and it is said that the fatal cases have terminated, as a rule, in five or six days. In addition to more or less gastro-intestinal irritation those attacked have suffered from extreme muscular weakness, sore throat, and dysphagia, and various nervous symptoms, such as squint, ptosis, loss of visual accommodation, stupor, and convulsions. The immediate cause of death in most cases has been a gradual failure of the heart.

It is impossible not to be struck with the similarity of these nervous and cardiac troubles with those which so often supervene as a sequela of diphtheria. In diphtheria these nervous symptoms are due to nerve degenerations brought about, according to Dr. Martin, by an acid and an albumose, which are formed as a consequence of the cultivation of the bacillus diphtheriæ on the palate, fauces, or elsewhere. In sausage-poisoning the cause of the phenomena is strictly analogous, the only difference being that the toxic bodies are brewed outside the body, and not in it.

Sausage-poisoning is not common in England, because our habits do not lend themselves to this particular form of food-poisoning, but outbreaks of a disease of a distinctly analogous kind have been observed again and again. Many of these outbreaks have been observed and recorded by Dr. Edward Ballard, F.R.S., one of the most distinguished of the Alumni of University College, to whom the public is indebted for many important discoveries in connection with public health. In all the cases observed the phenomena were those of gastro-intestinal irritation, with febrile and nervous symptoms such as accompany infective fevers, and were preceded by an incubative period. In 1880, at Welbeck, nearly one hundred persons suffered, and four died from eating cold hams which had been stored in a dirty place for the four days preceding. In 1881, at Nottingham, several families were attacked after eating 'cold gravy,' which was made from 'stock' which had been kept in a filthy cellar. At Chester 'tinned sausage' has proved fatal, and at Oldham 'tinned' pigs' tongues produced serious illness in several persons. At Bishop Stortford three families were made ill from eating cold ribs of beef, and at

Whitchurch (Salop) cold brawn and cold roast pork on two separate occasions produced similar results. At Wolverhampton, in 1884, a 'blown tin' of salmon not only proved fatal to two persons, but was found to be most fatal to animals inoculated with it. In 1886 twenty out of twenty-four persons who partook of a wedding breakfast at Carlisle were attacked. The breakfast was cold, and consisted largely of gelatinous dishes, such as jelly and game pie, which had been stored in an ill-ventilated cellar. Twelve persons suffered at Iron Bridge from eating veal pie 'warmed up' on the second day. and at Retford eighty persons in twenty-two families were made ill from eating pork pie and brawn. At Carlisle pork and brawn produced similar results in 1889, and at Portsmouth cold pies proved harmful to nine boys and four adults. Finally, Dr. Ballard seems to have shown that at Middlesbrough, in 1888, an epidemic of pneumonia was largely due to the consumption of half-cooked 'American bacon,' which had been hastily and improperly cured in this country in a factory built over a sewer grating.

In all these cases of Ballard's micro-organisms were found in the food and in the bodies of animals fed or inoculated with the food, and the poisonings were doubtless caused by the ingestion of the bacilli themselves or of the chemical products formed by them. The presence or absence of a well marked incubative period appears to depend upon whether the product of the organism is present in the food in toxic quantities (in which case the incubative period is absent or very short) or whether the bacillus alone is taken and the poison is brewed by it in the body of the infected person.

Ballard points out that in the fourteen instances which he brings together pig meat was the cause of the food-poisoning in nine of them, and the explanation is probably to be found in the fact that not only is pork often eaten cold, but its gelatinous nature lends itself to the cultivation of microbes. Finally, in studying the etiology of food-poisoning, and after arriving at the conclusion that it is due to the fact that the food has accidentally become a medium for the cultivation of pathogenic microbes, we have to inquire how such so-called

accidents arise. It will be observed that food-poisoning has most often arisen in connection with food which has been stored after cooking, and has been eaten cold after an interval of one or more days, and Dr. Ballard was able to show that in all but one of the cases he reported the food had been stored in a foul place.

An interesting case in which the same class of facts was observed was lately brought to my notice by Mr. T. Bullock, of Isleworth. A family bought a leg of pork, and ate it hot on Sunday. None of those who shared this Sunday dinner were made ill, but of those who partook of the cold remains on Tuesday three or four suffered severely from gastro-enteritis. What still remained was given away, and of the three who ate it all were dangerously ill, and two died on the following Sunday. When the facts came to be investigated it was found that the meat after cooking had been stored in a filthy larder under the stairs, communicating, on the one hand, by means of a 'ventilator,' with a dog-kennel, which had 'never' been cleaned, and, on the other, by means of the stairs with the damp basement of an ill-drained house. In the Middlesbrough epidemic it was found that the bacon fat which was removed from the sewer grating was singularly fatal to the animals to which it was given.

It is well known that mussels and other forms of 'shell-fish' are liable under certain circumstances to produce gastro-enteritis of a violent kind, and recent cases seem to show that an important factor in the causation of limited epidemics of 'mussel-poisoning' is the water in which the mussels have grown and been nourished ; and that in not a few instances poisonous mussels have grown in harbours and similar foul places into which sewage has been allowed to flow, and in which there has been more or less stagnation. Not only mussels, but other 'shell-fish,' such as crabs and lobsters, and certain other kinds of fish, such as mackerel, have an evil reputation for being occasionally dangerously unwholesome. It must be remembered, however, that fish of all kinds is very prone to undergo putrefactive changes, and that any evil qualities which it may acquire are not necessarily due to its mode of nourishment,

but to its being stored in unwholesome places on land. The cause of these epidemics is alleged to be the formation of alkaloidal bodies having poisonous properties, and which have received various names, according to their source of origin.

It appears, however, to have been recently established that certain 'shell-fish' which are habitually eaten raw may become the vehicle for the conveyance of the germs of specific disease, such as cholera and enteric fever. This fact appears to have been first pointed out by Johnson Lavis and other practitioners at Naples, where the oysters nourished in the then filthy little harbour of Santa Lucia were frequently the means of conveying the infective poisons of enteric fever and cholera to the consumers of them. In 1893 there were in this country a few cases of cholera, which were investigated by the Local Government Board, and in not a few instances the infection appears to have been traced to oysters grown in 'beds' situated at G—— and C—— ; the official report on the subject concludes with the following words :

'One thing is certain, oysters and shell-fish, both at the mouth of the Humber and at other points along the English coast-line, are at times so grown and stored that they must of necessity be periodically bathed in sewage more or less dilute ; oysters have more than once appeared to serve as the medium for communicating disease, such as enteric fever, to man ; and so long as conditions exist such as those with which the oyster trade of C—— and G—— is shown to be associated— conditions which may at any time involve the risk of the fouling of such shell-fish with the excreta of persons suffering from diseases of the type of cholera and enteric fever—so long will it be impossible to assert that their use as an article of diet is not concerned in the production of diseases of the class in question.'

It will be remembered that in 1895 several cases of enteric fever in this country were traced to the consumption of oysters.

These food-poisonings may be arranged in three classes :

1. Cases in which a diseased state of the animal consumed as food has been the means of causing disease of a similar kind in the consumers. The cases reported in Germany in which

gastro-enteritis closely resembling typhoid has followed the consumption of diseased veal belong to this category.

2. Cases in which the food, without being in itself un-wholesome, is made the vehicle for conveying pathogenic microbes to the consumer. Most of the 'milk epidemics' of enteric or scarlatina and mollusk enteric and cholera belong to this class, but it must be remembered that classes one and two are not always to be easily distinguished.

3. Cases in which food which was wholesome when bought and wholesome when cooked has become infective from being kept in a filthy place. The cases described by Ballard mainly belong to this category, which seems to cover the majority of the cases of food-poisoning.

What has been said with regard to these cases must impress us with the strong analogy existing between many cases of food-poisoning and the specific infective processes which are met with in the various fevers.

The pathology of fever which is now generally accepted is that the body is invaded by microbes which, growing, brew 'toxines' from the blood and tissues, and that these toxines, after absorption, cause the various phenomena of the fever.

Food-poisonings are distinctly analogous. The food may contain microbes which, taken into the body, tardily brew a 'toxine,' which thus produces its effect after a relatively long incubative period ; or the toxine may have been previously brewed in the food in large quantities, and then the toxic effects closely follow the ingestion of the meal.

In the former case, where the attack is slow and insidious, it may be impossible to connect the symptoms with any par-ticular article of diet, but in the latter case the connection between the food and symptoms is obvious. It is clear that the investigation of cases of infective fever and pneumonia cannot now be considered complete without careful inquiry into the food consumed by the various persons attacked.

The chemical nature of the bodies which are the actual cause of the poisonous effect occasionally produced by food has received considerable attention from chemists.

Many of the bodies formed during putrefaction appear to

resemble alkaloids in their general chemical reactions, and Selmi, who was one of the early investigators of these bodies, proposed for them the name of ptomaines, or cadaveric alkaloids. Selmi derived his word from πτῶμα, a corpse, and it has been not unusual to speak of food-poisoning as 'ptomaine'-poisoning. It would seem that the time has come for abandoning this barbarous and unpronounceable word, for not only are poisonous bodies found in other things than corpses, but it is certain that not all the poisonous bodies which result from putrefaction resemble alkaloids. Further, we now know that although found in corpses these bodies are the products of the active life of micro-organisms. It seems better, therefore, in every way, to speak of them as food toxines or putrefaction toxines, and in so doing we call attention to the analogies which exist between food-poisoning and the other infective processes.

For this view I am happy to find that I am able to quote the authority of Dr. Kanthack, who, in his article on the 'Pathology of Infection' in Allbutt's 'System of Medicine' (vol. i. p. 527), says, speaking of the uncertainty which still overhangs the chemistry of bacterial products :

'In the meantime we may sum up that among the specific toxines there have been found—

'(*a*) Ptomaines.

'(*b*) Toxic proteins, toxalbumins.

'(*c*) Nucleo-albumins and albuminoid substances.

'(*d*) Ferment-like bodies (enzymes) secreted by the bacterial cell ; and, knowing as little as we do, it is safest to apply to the bacterial poisons the general term toxines.'

It is well to bear in mind that these 'toxines' may be derived from vegetable bodies (such as ergot of rye and mouldy maize, which have in times past produced epidemics of ergotism and pellagra), and that in all probability they may be volatile and assume a gaseous form. Thus Dixon Mann, in a paper on 'Ptomaines,' published in the 'Medical Chronicle' in 1888, says : 'The peculiar effects—drowsiness, mental depression, and lassitude—which I experienced in the early stage of the operations, when working at the isolation of

pathogenic ptomaines from the organs of patients who had died from infective diseases, incline me to believe that some of these products are volatile.'

Hünefeld found that tissues impregnated with arsenic and allowed to decompose gave off an odour of garlic, and that the arsenic tended to disappear entirely ; and Victor Vaughan also found that at the end of six months he could get no evidence of arsenic from chopped liver which had been kept in a bottle, and which had previously yielded unmistakable evidence of arsenic.

Among the common products of the putrefaction of organic matters are gases such as carbonic acid, hydrogen, marsh gas, and sulphuretted hydrogen, and we know that the latter produces poisonous effects even in small quantities, and, according to Haldane, is the probable cause of the death of sewer men who enter unventilated sewers, and must be regarded as a gaseous toxine resulting from the putrefaction of food refuse such as the contents of kitchen sinks and the abominations known as 'fat traps.' The fact that gaseous toxines undoubtedly exist is one which must not be lost sight of, and is of great practical importance.

It must be borne in mind that food which has become toxic is not necessarily in a repellent condition, and in some instances it has been observed that the toxic qualities of food have lessened when the grosser and more repellent forms of putrefaction have set in. Many persons habitually eat 'game' in a condition which to many of us is sickening and repellent, and others highly appreciate the flavour and odour of 'ripe' cheese—i.e. cheese which is riddled with moulds of various kinds.

It has been stated that the toxines which are the result of anaërobic microbes are especially dangerous, but of this further evidence is necessary. It is the anaërobic fermentation, such as putrefaction under water, which especially gives rise to foul-smelling bodies, but, as has been said, the toxicity of such bodies differs immensely. We are in want of exact information on the whole subject, and doubtless such information will in the course of time be gradually furnished.

The *treatment* of food-poisoning need not detain us, as it has to be conducted on ordinary medical principles, and must necessarily vary with the time which has elapsed between the ingestion of the poison and the commencement of treatment. It must be both eliminative and restorative, but must vary with the symptoms. A certain number of cases end fatally in spite of treatment, just as is the case in the other infective diseases.

It is far more important to consider the measures which must be taken to prevent the occurrence of food-poisoning.

A large proportion of cases have occurred in food eaten raw, and there can be no doubt that the *thorough cooking* of food, by killing the micro-organisms upon which the toxic changes depend, would tend in many instances to prevent the occurrence of the mishaps we have been considering or to lessen the severity of the symptoms.

If the toxines be already formed in large quantities the effect of heat is not always to destroy their toxicity. Some toxines, such as cobra-poison, are said to be very tolerant of high temperatures, and it is certain that food-poisoning has occasionally followed the ingestion of well cooked food, showing that the food toxines are also occasionally tolerant of heat.

In food which is stored after being cooked it is very important to prevent the growth of micro-organisms, or, in other words, to keep it sterile.

I remember once ordering a quart of turtle soup to be sent to an invalid in the country, and I asked the tradesman who supplied it how it was to be kept from 'going bad,' as it was probable that the quantity supplied would be enough for four or five days. His answer was that if it was just raised to the boiling point every day it would keep as long as one chose. This answer was exactly what would have been given by a bacteriologist, and it is noteworthy that many of the sterilising and antiseptic processes which are so frequently employed by scientists and surgeons have been practised by well trained cooks for centuries. Practice and 'rule of thumb' is often perfectly sound, although we may be ignorant of the *rationale* of the process.

In every well conducted kitchen a 'stock pot' (i.e. a pot in which bones and meat are boiled) is kept going for the furnishing of a foundation for soups and gravies, and if its contents are raised to the boiling point every day they will keep continuously wholesome. If, however, the stock pot be neglected, its gelatinous contents are very liable to serve as a cultivating medium for pathogenic microbes. It will be remembered that in more than one of Dr. Ballard's cases of food-poisoning it was the cold gravy which proved to be the toxic material.

It is not likely that we shall abandon the use of cold food, which is both convenient and agreeable, and the danger of eating cold food is not great if certain precautions be taken.

The place where cooked and uncooked food is stored—the larder—must be wholesome, and to this end some care must be used in its construction. The following appear to me to be the points which demand attention in the larder, an apartment which may influence the health of a household to a very great extent.

1. The larder must be *dry*. Both walls and floor should be above suspicion as to dampness. Any mould or mildew growing on the floor or walls is very apt to infect the food. The floor should be of concrete, without seams or joints. The walls should be whitewashed every year, and the shelves (if expense be no object) should be of some non-absorbent material, such as marble, slate, glazed earthenware, or glass. It is better to have a larder above the ground level than below it, because in the latter situation it is very difficult to prevent dampness.

2. A larder must be *cool*. If it have no rooms above it the roof must be so constructed as to keep out the heat of the sun. It is essential that its windows and ventilator should face the north. The temperature which is most favourable for the growth of microbes is one which approaches the blood heat, and, speaking generally, one may say that the higher the temperature the more likely is the 'cultivation' of microbes to go forward. It is important that neither the flue

of the kitchen fire nor that of any other fire should touch the wall of the larder.

3. Good ventilation is essential. The windows should be big, and should be protected on the outside with wire gauze, so as to prevent the access of flies or other insects.

4. On no account must there be a pipe communicating with any underground drain or sewer either inside or, indeed, near to any place where food is stored. The Middlesbrough case shows the possibility of food being contaminated by emanations from a sewer, and also the possibility of epidemic pneumonia resulting therefrom. In short, we must bear in mind that sewer-poisoning may be indirect through the food as well as direct from the sewer itself. When the shelves and floor of a larder are washed they should be wiped dry, and such washing should be carried out preferably in dry weather, so that the drying process may be complete.

We also know how sensitive food is to unwholesome contaminations, and we may well have a doubt as to the sanitary condition of show dairies where a cool splashing fountain plays in the centre, because such a fountain must cause dampness and have a waste pipe, and we must fear that such a waste pipe communicates with a drain. A dairy equally with a larder should be kept cool and dry, and offer no facilities for the ingress of putrefactive products from a sewer.

I would point out that it is not any protection to have pipes trapped, because in every form of trap one must have stagnation, and wherever stagnation occurs there must be putrefaction. A very small quantity of food refuse or milk is enough to set up putrefaction in a trap. If any outlet for water is thought desirable in a larder it should be in the form of an open gutter, which can be thoroughly cleaned and dried, and which should pass directly through the wall on the floor level, the opening in the wall to be closed by a sliding trap-door when the gutter is not being used. No bacteriologist needs to be reminded that a water trap is necessarily a cultivating chamber.

The fact, now only too well established, that food or water

or milk may convey pathogenic microbes to the consumer has led to the advice that all food, milk, water, meat, oysters, &c., should be cooked thoroughly before being consumed, and if this be done it is thought that we shall get no harm from mixing our fæces with our water supply, as we are now practically compelled to do by Act of Parliament.

It is, however, more than doubtful whether we can altogether do without some forms of raw food. Certain it is that our natural appetites impel us to the consumption of many forms of raw food, and that we, most of us, enjoy new milk, raw oysters, fruit, salads, and fermented drinks, which are all in a sense 'alive.' Certain it is that nature intended sucklings to live on raw milk, and we must have misgivings as to the advisability of disallowing this natural nutriment. Our numerous experiments with animal extracts seem to enforce the conclusion that cooked food may be very different in its physiological effect from raw food. The marvellous results which follow upon the administration of thyroid extract prepared by evaporation under a vacuum or by extraction with glycerin are not observed when the cooked gland is given. Most of us also have had experience of the great value of uncooked meat juice, and it is not improbable that the high value of cod-liver oil is due to the fact that it is extracted at a temperature under 180° F.

It is possible that our knowledge on these questions will be more certain at no distant date, but the considerations I have brought forward make it certain that we must pause before we throw over articles of diet which have been used for centuries by the human race.

CHAPTER XI

AUTO-INTOXICATION [1]

A case of insensibility suddenly supervening on extreme muscular exertion

ON February 25 a strongly built man, twenty-one years of age, was brought to University College Hospital in a semi-conscious and irritable condition with the following history. The previous day, February 24, 1894, he had come up to London, a distance of 120 miles, to take part in a cross-country ten mile race, for which he had been in training for some time previously. At 6.30 A.M. on that day he had for breakfast two eggs, a few pieces of toast, and two cups of tea. This was the last time he had anything to drink until after the race. He had lunch in London about midday. This consisted only of a chop and dry toast. The race commenced at 4.30 P.M., and the course, which was partly through plough land, taken as a whole was extremely heavy, owing to recent rain. The patient said that he felt out of sorts before the race began, but he finished well up, doing the ten miles in sixty-six and a half minutes. During the race he did not perspire as much as he ought to have done, and afterwards, when he went to the dressing-room, he felt very thirsty, and drank off about a pint and a half of cold water. After this he became ill, did not recollect dressing himself, and, in fact, remembered nothing more until he found himself in the hospital next evening. His friends state that the race was finished about 5.45 P.M., and after this the patient dressed himself with very little assistance, but he did not seem well ; he appeared 'over run.' Just after leaving the dressing-room with his friends he 'fainted.' They gave him some brandy, but, as he

[1] Clinical Lecture, U.C.H., 1894.

became unconscious and helpless, they carried him to a neighbouring house and laid him down on the floor. Here he lay quite still, with semi-closed eyes, occasionally groaning as though in pain. His breathing was stertorous, and his pupils are said to have been dilated equally and his teeth clenched. He continued thus until 7 P.M., when he began to have convulsions, which appear to have been epileptiform in character. They came on with intervals of one or two minutes, in which the breathing was stertorous and the patient was quite still. A considerable quantity of saliva ran from his mouth, but he did not bite his tongue. At 8 P.M. the convulsions became so violent that it was found necessary to restrain the patient, and at 11 P.M. he was given a hypodermic injection of morphia. This controlled the fits, and he lay quiet during the rest of the night, but was quite unconscious. All next day, February 25, he remained in the same condition, and in the evening was brought up to University College Hospital.

Condition on admission.—The patient lies on the couch in a curled-up position, with his legs flexed on to his abdomen and his arms crossed over his chest. He is in a state of extreme cerebral irritation and resists all attempts to examine him. If his eyelids are opened to look at his eyes he closes them tightly and rolls over on the couch with his face to the wall. He lies quite still, except that occasionally he puts his hand to his head and rolls over on the couch, especially when any attempt is made to examine him. His pupils are equal and semi-dilated ; the conjunctivæ are suffused. He will not say anything in answer to a question, but only opens his eyes and stares vaguely about. He will not put out his tongue. The breath is foul. Pulse regular, full, and bounding. No paralysis can be made out anywhere ; knee-jerks equal on both sides. Except that he at times passes his hand across his forehead he does not seem to suffer any pain. Urine, obtained later, found quite normal in every respect. At 11 P.M., as the patient had had no food for the last twenty-four hours, preparations were made to feed him nasally. The passage of the tube caused such violence on the part of the

patient that the tube was taken out. He then sat up in bed, seemed quite rational, drank about a pint and a half of milk, and took three grains of calomel.

During the night the patient was restless, but quite con-scious, and took his food well. The next day, February 26, he complained of some headache, but was quite sensible and took interest in what was going on. The headache disappeared towards the end of the day. Examination of the patient revealed nothing abnormal. He left the hospital on March 4, no bad symptoms having developed in the interval.

Clinical remarks.—Prolonged insensibility resulting from, or determined by, excessive muscular exertion is sufficiently rare to merit rather special attention. It would, to say the least, be rash to conclude that an insensible and semi-comatose condition betokened any coarse lesion of the brain. There is abundant evidence that such a condition is com-mon without any cerebral lesion whatever. What, indeed, is sleep—that daily recurring attack of insensibility from which, happily, we all suffer—but the physiological prototype of the more intense pathological conditions? Sleep might be light or heavy, sound or disturbed, and accompanied by mutterings, dreams, nightmares, or movements, but it was very seldom that any of these morbid complications of normal sleep were attributable to any definite brain lesions. Sleep was probably brought about by the action of waste products circulating in the blood, and it was said to be generally accom-panied by contraction of the cerebral vessels. We have lately had in the ward a case of so-called 'uræmic coma' and another case of 'diabetic coma,' and I would remind you that in both these cases the cause of the coma was to be looked for in the deranged composition of the blood, and not in any definite lesion of the brain or its membranes. On the other hand, we have now in the ward a patient suffering from pro-found and complete right-sided hemiplegia, in which the lesion in the brain is certainly very extensive, and yet there has never been so much as a cloud over the man's intellect, and he has astonished us all by his singular quickness, accuracy, and volubility. These cases all point to the fact that the connec-

tion between insensibility and brain lesions is by no means constant. The patient under consideration has never manifested any symptoms which warrant us in concluding that there has been any lesion of the brain or its membrane. He has been insensible, he has had fits; but in the absence of any localising symptoms we should be wrong to regard the fits as 'cerebral' or 'meningeal' in origin. It is certain that forty-eight hours after the onset of his trouble a careful examination of the patient failed to reveal anything amiss with any organ of the body. What, then, was the cause of the attack? The answer which is most probably correct is that the patient was poisoned by his waste products. We must remember that ten miles across a heavy country and over obstacles represents a prodigious amount of work, and that this heavy work was concentrated in a very short period of time. If the products of tissue metabolism, as must have been the case, were excessive, and if their eliminations did not keep pace with their formation, then we have a condition distinctly analogous to uræmia or diabetes, and we cannot wonder that symptoms such as insensibility and convulsions should supervene. But, it will be urged, 'this man is a trained athlete, well accustomed to such work, and no similar accident has befallen him on any previous occasion.' This is doubtless true, and it behoves us to review the history which is given by the patient and his friends, and try to discover what were the exceptional circumstances (if any) which led to such an exceptional result. First, we have the statement of the patient that he felt 'out of sorts' when he started for the race. This is a sufficiently indefinite statement, but we shall not be wrong in insisting that the functions of our bodies are not so well performed when we are 'out of sorts' as when we are 'fit.' Next we have the statement that he did not perspire as much as is usual with him in such contests. His statement to me on this point was very definite; he said he 'scarcely perspired at all,' and 'nothing like as much as usual.' If we accept this statement we have a right to assume that, the great cooling mechanism of the body being

relatively inactive while heat production was excessive, there must have been hyperpyrexia and a condition not unlike some forms of heat apoplexy. If there was hyperpyrexia, we have a condition, in addition to the exercise, which certainly would exert an influence over the nature and quantity of the tissue waste. I think there is no evidence whatever that the attack was in any degree attributable to heart failure. The heart has always appeared strong, and in every way normal, and the vigorous struggles of the patient during his unconsciousness negative the idea of cardiac exhaustion. It is possible, however, that the copious draught of water which he took in the dressing tent may have caused a flow of blood into the abdominal vessels and have indirectly produced sufficient cerebral anæmia to determine the sudden onset of unconsciousness. You will have gathered from these remarks that there were three conditions which, so to say, made for unconsciousness— viz. (*a*) waste products circulating in the blood, (*b*) hyperpyrexia, and (*c*) sudden cerebral anæmia. A very important point is the prognosis. Is this man's career as an athlete at an end? Or may we tell him to go on attempting to add to the 140*l*. worth of prizes which he states he has won in the last three years? Is this attack a pure accident, or is it liable to recur? These are questions to which an answer is expected. It would be safest, perhaps (for ourselves), to tell him on no account to run again, but by giving such safe advice we may deprive him of a not inconsiderable income, which he may still be quite competent to earn with safety. Certain it is that the man is organically quite sound, and before advising him to give up using his fine body in manly exercises let us look to his history and see if there be any circumstance which may have an important bearing upon the matter. First, the patient stated that he felt 'out of sorts' when he started for his race, and although all one can say of being 'out of sorts' is that it indicates some derangement in the working of the machinery of the body, it is impossible not to believe that such derangement must have had an important bearing on the *contretemps* for which he was admitted here. I am inclined to attach considerable importance to his diet on the day of the race. At

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6.30 A.M. he had two eggs, two cups of tea, and some toast, and at 11.30 A.M. he had a mutton chop and dry toast, but *nothing to drink.* A cup of tea was put before him at the midday meal, but he did not like it, and put it aside. The amount of food taken on the day of the race seems small in its relation to the man and his work, and it is almost certain that the amount of fluid taken was too small, and I am inclined to attach considerable importance to the fact that, owing to an accidental circumstance, he took no fluid with his midday meal. If a man starts on such a race with too little fluid in him the excretion by the skin and kidneys will be hampered, and his risk of auto-intoxication and hyperpyrexia be increased. In the Turkish bath, when a bather does not sweat the attendants offer him some tepid water, and in the majority of cases the body will bead with perspiration within a few seconds of taking it. If a normal amount of diaphoresis had been started in our patient it is possible that his attack of insensibility might not have occurred. In his work on bodily exercise Lagrange touches upon the subject of auto-intoxication, and he asserts that, although not very common in human beings (unless we include many of the cases of so-called 'sunstroke' occurring in soldiers after a long march), it is far from uncommon in hunted animals, which, he says, are not seldom found dead in their lairs on the day following a prolonged hunt. It is to be regretted that no careful examination of the patient was made in the hours immediately succeeding the race. A record of temperature and an analysis of the first urine secreted might have thrown considerable light on the case, but the circumstances were obviously of a kind to preclude such investigations. After his admission here the highest temperature recorded was 99° F., the urine was high coloured but otherwise normal, and the one other point of note is that the breath was 'foul,' a fact which tends to justify one in ranging the case alongside of 'uræmia' and 'diabetes.'

CHAPTER XII

A CASE OF GOUT [1]

PATIENTS with acute gout are not among the commonest of those admitted to a general hospital, and therefore I deem it advisable to say a few words on the perfectly typical case of this disease which was recently in my wards.

S. P., a man aged forty-three, earning his living by doing 'odd jobs,' was admitted on May 17 with a swollen and painful condition of the right big toe. The swelling, which was of a dusky red colour and extended round the metatarsophalangeal joint and on to the dorsum of the foot, was very tender, and was the seat, as the patient informed us, of continuous pain of a burning, throbbing character. This swelling prevented him from wearing a boot, and made standing at once difficult and painful. Beyond the local condition there was little amiss. The patient was well nourished, plump, and rubicund ; the tongue slightly furred ; the temperature slightly raised (99·2° F.) ; the urine was acid ; specific gravity 1,015, and contained no albumen nor any abnormal sediment beyond a slight deposit of urates. He was kept in bed, placed on a soft diet, and given some saline aperient, and left the hospital quite well at the end of a week. The inflamed joint was painted with collodion, which gave marked and almost immediate relief, but beyond the saline aperient no other drugs were in this case necessary.

The attack had commenced suddenly at three o'clock in the morning four days before he was admitted. He states that he is fond of beer and has drunk it freely all his life, and he admits that the day before the onset of his attack he had drunk very freely of beer. He says he 'cannot remember

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[1] Clinical Lecture, U.C.H., 1894.

how much.' The patient states that he had a similar attack eighteen months ago, and that his father died after an operation for stone at the age of forty-two.

In this case the age, the habits, the previous attack, the sudden onset at 3 A.M., the podagra, the duration, are all typical, and the further fact of his apparent hereditary tendency to gout, as evidenced by the death of his father from stone, is also typical.

The inflammation of the joint was, in all probability, due to a deposit of urate of soda in and around the joint, and it is probable that we might have obtained crystals of uric acid from the blood-serum had we seen him in the earlier days of his attack. An acute attack of gout is always accompanied by an excess of uric acid in the blood, some of which, being deposited in the joints, produces the gouty inflammation. This explanation of a gouty paroxysm, which we owe to Garrod, is universally accepted. Nevertheless, I would ask you not to be too exclusive in your attitude towards the uric acid, but to regard it merely as a tangible and invaluable evidence that the blood is overcharged, and for some reason contains an excess of excremental matters which ought to have been withdrawn from it by the kidneys or other excretory channels.

Gout is due to auto-intoxication owing to a want of balance between the processes of absorption, metabolism, and excretion.

In my lectures on medical jurisprudence I am accustomed to teach that the really operative dose of a poison is the balance between absorption and excretion. It is due to this fact that poisons injected into a vein or hypodermically act, as a rule, far more vigorously than when slowly absorbed through the alimentary mucous membrane. In the first instance the blood suddenly receives a dangerous charge of poison, while in the second absorption is so slow that elimination is able to keep apace with it, and little or no toxic effect results. It is due to the slow absorption and rapid excretion that snake venom seldom produces any toxic effect when introduced into the stomach.

In works on toxicology it is customary to quote the 'fatal dose' of each poison. Such figures are very fallacious, because not only do healthy individuals vary in susceptibility, but the possession of impaired power of elimination owing to the presence of granular contracted kidney increases incalculably the danger caused by the administration of toxic bodies. We all know, for instance, the extreme intolerance for such drugs as opium and mercury which is manifested by the patient with a 'gouty kidney.' These patients have an equal difficulty in eliminating the 'toxines' of specific fevers, and I have on several occasions during the past few years pointed out to you patients in my wards whose tendency to relapse in typhoid and rheumatic fevers seemed to be accounted for by the low specific gravity of the urine, which indicated a feeble power of urinary excretion.

It may help towards an appreciation of gout to shortly consider some other forms of auto-intoxication. In diabetes we have to do with a poison which is the result of deranged metabolism, and so soon as elimination of the morbid products is checked distinct toxic effects are produced. Not only is the diabetic liable to coma, but, equally with the gouty subject, he is liable to a variety of local troubles.

That form of auto-intoxication which we call 'uræmia,' and which is produced by disease of the kidneys, is due in the main to faulty excretion, and not unfrequently constitutes the final phase of gout.

I may remind you of a case of acute auto-intoxication which was admitted under my care last year—the case of a young athlete who became insensible and slightly convulsed at the close of a ten mile race across country—due, as we thought, to the sudden overcharging of the blood with waste products. (See page 205.)

The ordinary 'bilious attack,' with its headache, malaise, vomiting, &c., such as is common in schoolboys who have overgorged, is due to absorption being greatly in excess of excretion.

These cases of auto-intoxication are due, then, to excessive absorption, deranged metabolism, and defective excretion.

Normally the three processes bear a due proportion to each other, the balance of power is maintained, and 'health' is the result. George Herbert's dictum that 'man is all symmetry' is only true sometimes. I was once informed by a person of good education who had undergone 'two months' hard labour' that he never knew till then the bliss of being in perfect health—a bliss attributable to the fact that his intake was certainly not greater than (probably slightly below) his output. There can be no doubt that a fair proportion of those who become gouty are endowed with exceptional powers of primary digestion and a keen appreciation of the pleasures of the table. It is the maintenance and indulgence of these powers and pleasures after the completion of the full development of the body which often determines the occurrence of gout, an occurrence which is hastened by a forced abstinence from physical work or any impaired power of excretion by the kidneys. In these cases the gout is due to a relatively excessive intake.

It has fallen to my lot to see not a few very athletic men with enormous muscular development who have been sorely tried when they have been called upon to exchange the bat for the pen and the sliding seat for an office stool. Their symptoms have been obscurely gouty and accompanied by marked hypochondria. Enormous muscles inadequately worked seem to be a doubtful blessing. The symptoms in these cases are presumably due to a deficient output in the form of muscular work and its accompanying respiratory products.

The marvellous power of primary digestion which is often possessed by the diabetic must have the effect of putting a severe strain upon the liver, and whether the faulty metabolism of the diabetic may not in part be due to the want of balance between gastro-intestinal and hepatic function is at least a question. The great good obtained in some of these cases by administering opium by the mouth, which must partly stop and delay primary digestion, is an interesting fact.

The man with a feeble power of primary digestion, to whom all the pleasures of the table mean stomach ache, and who

manages to live on a surprisingly small quantity of the simplest food, not unfrequently outlives many of the jovial companions of his youth, who succumb to some form of visceral disease shortly after they have passed their prime.

There is probably no absolute measure by which we can gauge the assimilative and excretory power of any individual. Men are as unequal in this as in all other respects. The gouty are, however, distinctly divisible into two classes, the plethoric and the anæmic and emaciated. In the former class the appetite is good, the assimilative power great, and the attacks of gout are acute and often precipitated (as in the case I have brought before you) by dietetic excess. The health between the attacks is often robust. In the latter class adequate nutrition is impossible, there are generally distinct evidences of chronic kidney trouble, and it is probably the chronic difficulty of excretion which determines the malnutrition. Workers in lead afford typical examples of this latter class. Lead is a styptic causing contraction of the blood-vessels and anæmia. It checks excretion from the bowel by bringing about colic and constipation, and very rapidly induces (as has been pointed out by Oliver, of Newcastle) a fibroid change in the kidneys and liver. It is to Garrod that we owe a knowledge of the relation of lead-poisoning to gout. He it was who showed that the administration of lead salts to gouty subjects almost invariably determined a gouty paroxysm. This paroxysm is almost certainly due to the arrest of all excretory functions by the physiological action of the lead salts. Although we have at the opposite ends of the scale cases of gout which in the one case are due to excessive assimilation and in the other to defective excretion, we must not forget that we have all grades between, and, undoubtedly, the defective excretion is the fact of most practical value when we are called upon to treat cases of gout.

In treating cases of gout our efforts must be directed towards the maintenance of a tolerably even balance between assimilation and excretion. Undoubtedly much can be done by dietetic means, but I am inclined to think that hitherto too much attention has been given to the uric acid factor in gout,

and, instead of regarding it merely as a certain and most valuable evidence of a loss of assimilative and excretory balance, attempts have been made to check the formation of uric acid in the system by an over-nice and sometimes fanciful regulation of the diet.

Physiologists have not yet given us any acceptable theory of the genesis of uric acid, and one who has thought deeply on the subject and has enjoyed much practical experience of gout has lately warned us that he has been unable to accept any theory. He has abandoned theories in despair, and says 'that way madness lies.'

It seems certain that it is not an intermediate product between proteid food on the one hand and urea on the other, and there is no evidence that uric acid can be changed to urea by increased oxidation within the body. The fact that uric acid constitutes the chief urinary constituent alike of the hot-blooded and rapidly breathing bird and the cold-blooded and torpid snake makes any such possibility very unlikely.

The fact that all snakes are carnivorous and birds largely graminivorous, and the further fact that uric acid calculi are frequently met with among teetotal Mohammedans and the vegetable feeders of India, makes it very unlikely that we can control the deposition of uric acid in the tissues by merely withholding certain articles of diet.

Gouty persons are usually depressed and very often come to us asking for 'tonics,' and saying they are a 'peg too low' and want 'keeping up.' To accede to their wishes is a great mistake, and is analogous to heaping coals on a dimly burning fire instead of raking out the bars and getting rid of the waste products which prevent free combustion. Gouty patients are often told to eat poultry instead of 'butcher's meat,' but this cannot be defended on purely chemical grounds, for analysis shows there is very little difference indeed in the chemical composition of 'white meat' and 'red meat.' As the picking of the bones of a bird is a longer process than the gorging of slabs of solid meat it is probable that the poultry eater gets less flesh than the consumer of butcher's meat, and

this may be a good thing for him. If, however, the patient translate 'poultry' into 'boned quails stuffed with foie gras and served in aspic,' or similar dainties, he may effectually defeat his doctor's intentions.

Gouty persons are often warned against sugar, and are told on no account to touch fresh fruit such as peaches, grapes, and strawberries ; and, although the restrictions cannot be upheld on any chemical theory having reference to uric acid, there seems little doubt that gouty persons are not tolerant of sugar and sweet fruits.

It is clear that the gouty man must 'eat to live,' and must avoid pampering his stomach and taking more food than he requires. If he assimilate more than his excretory organs can satisfactorily deal with his auto-intoxication commences. If his urinary organs be overtaxed it is no wonder that the most insoluble of the urinary constituents (uric acid) should be one of the first things to be left behind. I would remind you that birds and snakes, in which uric acid is the chief urinary excrement, pass a urine which is semi-solid. This is necessitated, so to say, by the comparative insolubility of uric acid, which requires some fifteen hundred times its weight of water for solution, and which is insoluble in alcohol and most acids. It is no wonder that uric acid should be left behind when a competition arises among the candidates for urinary excretion. The greater the amount of material which has to be excreted by the kidneys the greater the likelihood that uric acid will be left behind among other things. It is manifestly important to cut off the pampering extras from the gouty man's diet. Among these extras are the majority of sweet things and fruits, both dried and fresh. If these things were taken as a real part of the meal for the purpose of satisfying a legitimate appetite the gouty person would soon accommodate himself to his diet, just as do those patients who undergo the 'grape cure' in Switzerland ; but when, after satisfying the appetite with soup, fish, entrée, joint, sweet, savoury, he continues to sit at the table and amuse himself with 'dessert according to season,' which he consumes without appetite, he necessarily overtaxes his excretory organs and increases his risk of gouty troubles.

The very bad reputation which strawberries have with the gouty is due to the fact that they are an extra tacked on to a diet already too liberal, and that, being cool and soft, any quantity almost can be eaten *without appetite*. Those articles of diet which demand little or no appetite on the part of those who consume them are very dangerous for the gouty. Sugar is essentially a diet for the young and rapidly developing, who have a natural craving for it, a craving which usually disappears as life advances. By freezing syrups so that the coolness masks the sweetness sugar is taken by the adult without appetite. The punch *à la Romaine* in the middle of dinner and the concentrated mixture of cream and syrup (called ice cream) at the end of dinner are the means of putting a mass of very strong and concentrated food into the stomach of a man who probably has already eaten more than he requires.

If a gouty man restricts himself to plain, simple food, and rigidly avoids all extras, such as dessert, his own appetite is probably his best guide in the selection of food. If he wishes to indulge occasionally in fruit he should take it as a substitute for pudding, or eat it at breakfast in lieu of something else.

Legitimate appetite and the craving for special articles of food is an astonishing fact of probably the first importance, notwithstanding that writers on dietetics are nearly silent on the subject. It is always a dangerous thing to make very sudden and arbitrary changes in the diet of those whose habits are formed, and in doing so I think no wise physician should neglect to attend to the clearly expressed likes and dislikes of his patient. In this connection I may mention the case of a singularly hale and hearty gentleman of eighty-seven years, whose dark hair, perfect set of teeth, and boy's appetite were the wonder of his friends and acquaintances. When this gentleman was recovering from influenza and pneumonia his doctor, finding something in his urine which reduced ' Fehling,' promptly cut off all the sugar from his diet. But the ' old boy ' was fond of his pudding, and when the pudding was placed upon the table he always clamoured (and successfully) to be served. Thereupon the family entered into a saintly con-

spiracy in their father's interest to eat no pudding, in order that eighty-seven might not be led into temptation. The result of this was that the patient was detected in the pastry-cook's shop wolfing up tarts like a schoolboy. This gentleman is now in his ninety-second year, still has a craving for sweet things, and is practically sound in body, very active, and clear in intellect. Undoubtedly in this case the natural cravings for sweets have done him no harm.

Another instance of irresistible appetite has come under my notice in the case of a naval officer who had been many months at sea in a ship in which the fresh meat and tinned vegetables ran out. The men were put upon a daily allowance of lime-juice, but my friend had neglected to take his daily dose. This officer was the pink of good manners and a perfect stranger to the vice of gluttony ; nevertheless, when the ship stopped at an obscure port and he took a walk on shore his eye caught sight, in the window of a general store, of a bottle of 'piccalilli' bearing the label of a well-known firm. The sight of the bottle produced a craving, and in a few minutes he had bought the pickles and consumed a large part of them by the aid of a button-hook. This is another instance of the right guiding of the appetite.

Why is it that the whole human race when it has reached a certain degree of civilisation has taken to fermented drinks ? Why does the whole world use allyl in some form or another ? What is it that has prompted us to cultivate plants which contain an alkaloid which chemists call theine, caffeine, theobromine, &c., according to its source of origin ? Finally, what is it that enables my dog to select the 'agropyrum caninum' with absolute certainty and without any previous instruction in the matter ? If we cannot answer these questions, let us remember to be sure of our grounds before we decide that our patients are not to be allowed reasonable scope in the selection of food.

In the selection of the gouty man's drink we have to take care that he gets as little food as possible in the guise of beverages. Thirst is the cry of the blood for water, and for the purpose of slaking thirst water alone is necessary. Unfortunately,

water is not popular with those who have become accustomed to the various alcoholic beverages which the human race has used for a good many thousand years. The alcohol which the gouty man takes with his meals may be of use to him by delaying the digestive process in the stomach and putting a slight check upon assimilation. That alcohol is a food there can be no doubt, but, unlike most other foods, it seems incapable of being stored in the body, so that any excess is rapidly excreted by the lungs and kidneys. Uric acid is insoluble in alcohol, and the man who takes an excess of alcohol with his excess of food is more likely to suffer from deposits of uric acid than he who takes excess of food only.

Port wine and strong beer are the types of drinks which undoubtedly favour the occurrence of gout, because they are not only rich in alcohol but in extractive matters or ' body ' also. The rich alcoholic drinks are really foods in disguise, for they contain, in addition to the alcoholic food which cannot be stored in the body, a large amount of carbohydrate which *is* stored in the body, as the brewer's drayman, fattest and placidest of beings, bears witness. The gouty man's drink must be the thinnest which he can be got to take, and it is now very much the fashion to order a small quantity of distilled drink drowned in mineral waters as a substitute for wine. The effervescing wines, in which there is a large amount of natural and added syrups, are very bad for the gouty, and when they have been made ' dry ' by the addition of constipating astringents they are not much better. All syrups and liqueurs must be avoided by the gouty, and it is probable that syrups bring quicker trouble to the gouty man than the raw sugar, which has to be dissolved (more or less slowly) in the stomach.

All the so-called teetotal drinks which are made with syrups are probably as bad from the point of view of gout as beer.

The greatest mistake, however, which the adult can make when he discards alcohol is to fly to milk.

When ' he asked water, and she gave him milk,' we must remember that she was contemplating murder, and we must

not forget that milk is one of the strongest foods, if not the strongest, which is within the reach of man. Milk has no right to be regarded as a beverage, and the gouty adult who abandons alcoholic drinks and takes to milk *as a beverage* jumps, I believe, 'out of the frying pan into the fire.' I am not arguing against the use of a milk diet, which is valuable enough in some cases of disease, nor do I object to the use of milk in cookery, but a word of warning needs to be given to those adults who employ milk instead of water for slaking thirst, or, in other words, use milk as a beverage, or, to repeat the expression I have used earlier, take a strong and insidious food *without appetite*.

It has fallen to my lot to see several worthy persons in whom this great dietetic error had produced undoubted symptoms of gout, including deposits of urate of soda. The man whose blood is habitually overcharged gets nervous, gloomy, and apprehensive, almost to the verge of melancholia, and I have seen this condition produced not only in those who are gluttonous and wine-bibulous, but also in those who for conscience' sake have forsworn the pleasures of the table and who have fallen into the error of using milk as a beverage. Milk is a marvellous *food* for children, and upon it they will grow and develop in a way which is simply astounding. Those whose growth and development are at an end cannot digest and assimilate milk as the child can, and the adult who drinks milk instead of water for the purpose of washing down a mixed diet commits a very dangerous dietetic blunder.

You will have gathered that the great dietetic rule for the gouty is to live on simple food, and to be ever on his guard against all those forms of food which we habitually take without appetite. It is doubtful if any rule of life for the gouty can be better than Abernethy's, which was 'to live on sixpence a day and earn it.' Plain food to satisfy appetite, with plenty of exercise in the *fresh air*, at once checks assimilation and spurs excretion, and these are the indications for treating the gouty.

As regards the treatment of the gouty, next in importance to the diet is to keep the bowels rather freely open. In this

way not only is the nutritive matter kept for a shorter time in contact with the alimentary tract, but it is also probable that the large intestine serves very considerably as a channel for the elimination of waste products. In cases of poisoning with antimony, arsenic, and mercury it is certain that much of the poison is eliminated by the large intestine, and it is also probable that in the cases of auto-intoxication which we have been considering it serves a similar purpose. It is always undesirable to check the occasional diarrhœa which is common in gouty subjects, and equally undesirable to check occasional hæmorrhage from piles. For plethoric subjects the best purgatives are saline aperients, or aperient mineral waters, which they take well. For anæmic subjects it is better to give an aloetic pill with the meals.

When patients go abroad to ' take the waters ' not only are their habits of life completely altered, but the copious draughts of warm saline waters have the effect of washing the waste product out of the blood. When a patient has been through a London season, rising late, eating and drinking a great deal too much, and spending his time in overcrowded rooms, it is no wonder that he improves when he rises at six, drinks a pint or so of hot saline water before breakfast, lives sparely and carefully, and spends his whole day in exercise in the open air. Life at these baths is very pleasant, and the *régime* which is necessary is fashionable. Of late the fashion of drinking hot water at home has been advocated, but if a man prolongs this treatment he is apt to become anæmic, flabby, and washed out. The waters which are most in vogue for the gouty are Carlsbad (purgative), Homburg and Kissingen (saline), Contrexéville and Vichy (alkaline), Buxton, Bath, Baden, and Gastein (warm and indifferent).

For the gouty paroxysm, in addition to a saline purgative, colchicum may be given, a drug which has retained its popularity with the gouty for many years, although how it acts towards this end is by no means certain.

I am accustomed to prescribe something like the following :

℞ Sodii sulphatis,
 Magnesii sulphatis, āā ʒss ;
 Sodii carbonatis,
 Sodii chloridi, āā gr. v ;
 Potassii iodidi, gr. i ;
 Vini colchici seminum, ♏xv ;
 Aq. ad ℥ i. Fiat haustus.

Sig.—To be taken every four hours in a claret glass of hot water.

It is generally admitted that gout may be inherited ; and there is no difficulty, on physiological grounds, in accepting the fact that a man may resemble his parents internally as well as externally. Just as deficiency, redundancy, or peculiarity of external features may run in families, so may similar qualities of internal organs be inherited. We know that certain families have good appetites and fine digestions, while others, to use a phrase of the late Sir Andrew Clark, may inherit 'renal inadequacy' with a consequent tendency to gouty manifestations.

On the other hand, it must not be forgotten that 'inheritance' may not be of a true physiological kind, but may depend upon an education in luxury and indolence or the inheritance of a large income and a well stored cellar.

CHAPTER XIII

A CASE OF HÆMATURIA FOLLOWING INFLUENZA [1]

THE chief symptom from which this patient has been suffering is the presence of blood in the urine.

The urine was blood-coloured and highly albuminous, while under the microscope we found a large number of blood corpuscles. We were therefore enabled to pronounce the case to be one of *hæmaturia*, and not *hæmoglobinuria*, in which the colouring matter of the blood finds its way into the urine without the corpuscles.

Having satisfied ourselves that the urine contains blood, the next question to be answered in such a case is as to the source of the blood. From what part of the urinary tract does the blood come—kidney, ureter, bladder, or urethra ?

If the urethra be the source of blood only the first portion of the urine passed contains blood ; if the bladder be the source of the blood the last portions of the urine passed are most liable to contain blood. This is a very good practical rule, and one which you will find most useful if you are careful not to become the slave of it. In medicine it might almost be said there are no absolute rules for diagnosis, and anything like a ' rule of thumb ' must ever be employed with a wary eye for exceptions.

In this case the blood was equally distributed throughout the urine, and was in large quantity. These facts alone seemed to indicate that in all probability the blood came from the kidneys.

For the further elucidation of the case we must leave the one great objective fact of blood in the urine, and have recourse to the history of the patient, and such subjective facts as he may submit to us.

[1] Clinical Lecture, U.C.H., 1893.

The patient is a well made, temperate young man of 20, earning good wages as a jeweller, and in his family history there is no evidence of any hereditary taint. His occupation is sedentary (making filigree ornaments), but is carried on in a well ventilated workshop.

On June 21, 1893, the patient was seized with influenza, and it appears that the symptom so common in that disease of pain in the back and loins was rather unduly pronounced. There seems also to have been some tendency to vomit. He improved up to July 2, when the pains and sickness recommenced with greater violence, and on the following day the pain in the back and loins was sufficient, as the patient says, to 'double him up.'

He was admitted to the hospital on July 6. The expression which the patient uses to describe his pains, that 'it doubles him up,' gives us a clue to the fact that the pain is paroxysmal, and he further tells us that he suffers no pain in the intervals between the paroxysms. When asked to indicate the spot which is chiefly affected by the pains he puts his hand over the right loin and flank. Severe paroxysmal pain referred to the abdominal region is most commonly caused by intestinal, hepatic, or renal colic, and in the case we are discussing it certainly is due to the latter cause.

We have next to consider what is amiss with the right kidney that it should become a source of hæmorrhage, and whether the blood comes from the right kidney only or from both. That the blood is present in the urine in considerable quantity, and that the amount has shown no tendency to fluctuate, is rather in favour of its coming from both kidneys, and the fact that the symptoms of renal colic are referred to one side only must not be taken as conclusive evidence that the lesion is unilateral.

When admitted the patient had a persistent, moderately high temperature, which fluctuated from a little below to a little above 100° F. Notwithstanding the slight pyrexia and the hæmaturia, it was not possible to regard the case as one of acute Bright's disease or acute tubular nephritis, and for three reasons—(1) that the amount of urine passed was not far

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short of normal ; (2) that there is a complete absence of œdema, so that the finger firmly pressed upon the sacrum fails to make the slightest impression ; and (3) that on examination of the urine with the microscope we fail to see any renal epithelium, whether in the form of casts or otherwise.

The evidence of any local condition in the pelvis or body of the kidney adequate for the production of such marked hæmaturia is not very great. There is no swelling in either flank or loins, and palpation of the kidney has failed to detect any enlargement or tumour, or, except on one occasion, tenderness.

Pyelitis, whether tubercular or calculous, is usually accompanied by more or less tenderness on palpation ; and if the pelvis of the kidney contains a stone of any kind not only is there more or less tenderness on palpation, but a persistent pain, which is usually referred to the loins.

Again, an irritant in the pelvis of the kidney or in any part of the urinary tract usually leads to frequency of micturition, a symptom which, in the case we are discussing, was rather remarkable for its absence.

We have no evidence that the symptoms of renal colic were ever present before the appearance of blood in the urine, and that being the case one is inclined to attribute them to the passage of small clots from the pelvis of the kidney along the ureter.

On microscopic examination of the urine we have once or twice found crystals of uric acid ; and, although this may warrant us in suspecting that there may be a so-called sabulous deposit of uric acid in one or both kidneys, it is well to bear in mind that we have no very strong evidence of such a condition, and no evidence whatever of any calculus in any part of the urinary tract. We have not been able to make out that any stone has been passed into the bladder or from the bladder. The symptoms of stone in the bladder have been, and still are, absent.

If there be a sabulous or sandy deposit in the pelvis of one kidney, say the right, and if there be some general cause (such as influenza) which may determine an engorgement of, and

hæmorrhage from, the kidneys, then it is reasonable to suppose that a kidney which is already in a pathological condition would suffer more than its fellow, and it is conceivable that blood finding its way into the pelvis of the kidney would be more likely to coagulate if the pelvis had been previously roughened by uric acid. These facts are brought forward in defence, as it were, of the position previously taken up—that the hæmorrhage is probably from both kidneys, in spite of the unilateral symptoms, and that the accident, so to say, of a little uric acid in the pelvis of one kidney would be sufficient to account for the unilateral symptom of renal colic.

It is more than probable that a fine young man like the patient, whose natural destiny should have been some muscular exercise in the fresh air, but who is fated to sit on a bench making filigree in Clerkenwell, should develop what may be spoken of as the uric acid diathesis. This so-called diathesis is, broadly speaking, due to the imperfect metabolism of the nitrogenous food and nitrogenous waste-products, and it not only makes its appearance in those who over indulge in the pleasures of eating and drinking, but also in those who follow sedentary occupations, and who look pale, thin, and under-fed. It may be that our patient will develop a renal calculus in the future, but one must guard against attaching too much importance to the appearance of a few crystals of uric acid, which may be present from causes which are transient and temporary.

We now come to what is, perhaps, the most interesting part of the case, viz., the occurrence of the hæmaturia during an attack of influenza.

Of all the acute infective diseases influenza is, perhaps, the least definite in its character. In its simplest form it consists of pyrexia, with the addition of malaise and certain aches and pains, but it seems to have the power of producing congestions and inflammation of any organ of the body. When an epidemic wave of influenza has passed over London the Registrar-General's returns have shown that the mortality from all classes of disease has been increased, and this may be taken as evidence of the power of influenza to find out the

weak spots of the individuals which it attacks. Pneumonia
and bronchitis are the most common complications of influenza,
but instances of digestive troubles, diarrhœa, vascular dilata-
tion, ear troubles, brain troubles, and spinal cord troubles
have come within the experience of most who have seen much
of the disease.

Kidney troubles seem to be among the least frequent of
the manifestations of influenza, but the case under considera-
tion is at least the third which has come within my own ex-
perience.

The first case was that of a young lady who was a member
of a household which was smitten by influenza in the first
quarter of 1892. The symptoms at first were not particularly
alarming, but hæmaturia showed itself, and this was followed
by a suppression of urine, which led to a fatal result. This
young lady had never suffered from pronounced kidney disease,
but on making inquiries to ascertain whether there had been
any manifestations of unsoundness of the urinary organs
previous to the fatal attack, we found that she had been in the
habit of rising once or twice during the night for purposes of
micturition. There was no post-mortem examination, but the
frequency of micturition and a rather pronounced family
tendency to tubercle gave support to the supposition that
there was a chronic kidney trouble sufficient to constitute a
vulnerability of that organ in the event of the invasion of the
body by an infective disorder.

The second case was that of a gentleman between 40 and
50 years of age, who, towards the close of an attack of influ-
enza, was alarmed by the onset of hæmaturia, and who took
the somewhat imprudent course of travelling eighty miles by
train in order to get advice. The patient completely recovered
from his hæmaturia, but the impression left upon me was that
previous to the onset of the influenza the kidneys had been
overtaxed by the jovial habits of their owner, who I suspected
had been endowed by nature with a somewhat keen apprecia-
tion of the pleasures of the table.

The third patient, the one before us, has suffered from
hæmaturia during an attack of influenza, and in him, as in the

other two, we have reason to suspect a vulnerability of the kidneys because of the crystals of uric acid which have been discovered in the urine.

These cases illustrate what is very common in medicine, viz. the plurality of causes. When we ask, 'What is the cause of our patient's hæmaturia?' we seem compelled to reply that the predisposition to hæmaturia was due to the uric acid diathesis, but that the actual occurrence of hæmaturia was determined by the attack of influenza.

A very troublesome symptom when the patient was admitted was vomiting, and concerning this symptom a few words may profitably be said. Vomiting is a common occurrence during attacks of renal colic, and it is also an equally common symptom of 'uræmia.' An attack of renal colic may be regarded, for all practical purposes, as a local disorder ; but 'uræmia' is a grave general condition—a blood state—dependent upon the inability of the kidneys to perform their proper function of excreting the waste products of the body. The vomiting of renal colic is a purely reflex phenomenon due to the irritation of the pelvis of the kidney and ureter, and may be compared to the vomiting which follows the tickling of the fauces. Uræmic vomiting, however, must be regarded as due to an inability of the stomach to 'deal' with materials put into it, whether by digestion or absorption. The body being unable to excrete its waste material with freedom, it is evident that the absorption of new material will but increase its difficulties, and that the rejection by vomiting of material put into the stomach is, under the circumstances, highly desirable in the best interests of the patient.

Now, the vomiting of the patient whose condition we are considering was not limited to the periods covered by the fits of paroxysmal pain, but occurred independently of these ; and, in fact, the patient rejected by vomiting nearly everything which was given him. This very important fact affords evidence that we are dealing with a general condition, and not merely a local trouble in the pelvis of one kidney. It supports the theory that both kidneys are engorged with blood, and consequently hampered as to their physiological functions, and

that the occurrence of pain on one side only is, so to say, due to an accidental circumstance.

This discussion as to whether the hæmaturia is due to a local or general condition, and whether the patient has both kidneys dangerously engorged or is merely passing a calculus from the pelvis of one kidney, is of great practical importance, and must largely influence the treatment to be adopted and our forecast of the result. If treatment is to be reasonable and scientific we must have before the mind's eye a picture of the pathological condition with which we have to cope. In the case before us I think we must picture two engorged kidneys seriously hampered in their action, and not merely a little stone in the pelvis of an otherwise healthy kidney.

The patient was in a critical condition because of the danger of uræmic poisoning. The loss of blood, although considerable, was not sufficient to cause us any anxiety ; and attacks of renal colic, although severe and hard for the patient to bear, are so rarely accompanied by rupture of the ureter or any similar accident that they can scarcely be said to have increased the gravity of the prognosis.

Having discussed the case, and having arrived at the views which seem most reasonable, we are in a position to consider the treatment. The chief danger being that the engorgement of the kidneys may increase, and that the patient may die of uræmic poisoning, we must adopt such measures as may lessen the engorgement, and be careful to do nothing which may overtax the kidney.

Clearly the worst thing we could do would be to check the hæmorrhage. The loss of blood is not sufficient to endanger life, the patient's pulse is fairly good, and his condition does not indicate a dangerous degree of weakness. The engorged kidneys are bleeding, and we ought to be thankful for it, for there can be no surer road to disengorgement.

The worst thing to do would be to give astringents internally to check the hæmorrhage ; and about the most dangerous combination in such a case as the one before us would be the very common one of *Lead* and *Opium*. Lead would increase the tendency to the deposit of uric acid ; and the eliminating

power of the kidney is, in its present condition, so doubtful that comparatively small doses of opium might produce untoward results.

In consequence of the very doubtful capacity for excretion possessed by the kidneys in their present condition, I would recommend no narcotic drug for the relief of the pain until other less dangerous measures having a reputation as anodynes have failed in their effect.

To help to disengorge the kidneys we may endeavour to produce 'a determination of blood' to some other part of the body, and to this end nothing is more efficacious than counter-irritation. In selecting our counter-irritant, however, some caution is necessary. We must not employ any counter-irritant which is likely to irritate the kidneys, and therefore the use of cantharides or turpentine is not permissible. Again, while we want a counter-irritant which will act quickly and with vigour, we must be careful not to raise blisters, for a raw surface, especially over the loins, is a thing to be avoided at all times, but most particularly when the kidneys are inactive and in cases, like the one under discussion, in which œdema might make its appearance at any minute. The best counter-irritant in such a case seemed to me to be 'dry cupping' over the loins followed by hot fomentations. This was employed, and you saw how in a very few minutes we had succeeded in making the whole of the lumbar region of the back look red and swollen. We had ocular demonstration that our mode of counter-irritation was effectual, and there can be no doubt that 'dry cupping' (the application of cupping glasses without the withdrawal of blood) is a most valuable mode of treatment, and one which, just now, is a little too much out of fashion.

When I was a student we had upon the staff of this hospital 'a cupper,' whose duty it was to make the round of the wards after the visits by the physicians and surgeons and apply cupping glasses (with or without the subsequent application of the lancet) to those patients for whom this method of treatment had been prescribed. The rapidity and effectiveness of 'cupping,' as practised by the late Mr. Betts, can hardly be conceived by those who never saw that professor of the art operate

on the loins with three or four glasses and a spirit lamp, and convert the concavity of the lumbar region into a big red protuberance in scarcely more time than it takes me to tell you of it.

The next point in the treatment is to make sure that the bowels are kept freely, but not excessively, moved. The bowels in cases where the free action of the kidneys is interfered with become an important channel for the elimination of waste. To this end our patient has had a daily dose of saline aperient, which has produced the desired effect.

Finally, it is desirable to give the kidneys all the rest possible. Food and exercise both tend to throw work upon the kidney. The patient, therefore, must be kept absolutely in bed, so as to reduce the external and internal work of the body to a minimum, and thus bring the need for food to a minimum also.

The regulation of the diet is all-important. In this case nature has taken the law into her own hands, for the patient vomited almost everything which was given to him. The patient vomited because his kidneys were engorged, and I pointed out that so soon as the kidneys were disengorged the vomiting would cease, but no sooner. The vomiting was not sufficient to cause any alarm, and our patient's condition was not such as to raise in us any serious apprehensions of a dangerous degree of inanition. If a man be allowed water *ad lib.* for the purpose of quenching his thirst he is able to stand the deprivation of food for a long time ; and the public exhibitions of 'fasting men,' of which we have had several of late years, have taught us that the human body may be maintained for several weeks without the administration of any visible food other than water. Now, our patient's pulse was good, and, even supposing that he should continue to reject his nourishment for several days by constantly repeated vomiting, we had to remember that the vomiting was a physiological consequence of the inefficiency of the kidneys, and that until the efficiency of the kidneys should be restored the proper nourishment of the patient was impossible. No attempt was made to give nutritive enemata or peptonised food, nor was

any drug given with the intention of arresting the vomiting by acting on the stomach. The patient was placed upon a spoon diet from which the nitrogenous elements were removed as far as practicable. Farinaceous food, grapes, stewed fruits, bread and butter, weak tea and a little milk were allowed, together with as much water as the patient felt a need for. As for the times of giving food and the amount given, the regulation was left to the patient's inclinations, for in such a case as this the appetite is the best guide.

These measures—absolute rest, dry cupping, slight purgation, and a restricted diet—proved adequate. The temperature fell to normal ; the urine showed less and less blood until, finally, not a trace of albumen was found in it ; the pains in the loins ceased ; the vomiting stopped ; the appetite returned, and, finally, our patient left the hospital about a fortnight after his admission, in a state of practical health, for the purpose of recruiting his strength at one of our convalescent homes before returning to his jeweller's bench in London.

CHAPTER XIV

THE RELATION OF SLIGHT DEGREES OF ALBUMINURIA TO LIFE ASSURANCE [1]

WHEN your secretary did me the honour to suggest that I should open a discussion upon some subject of interest to the members of this society, I at once selected one which seemed to me to be of great importance and much in need of discussion. It is a subject which is not only of interest to medical officers, but is one, I fancy, upon which the minds of managers and actuaries are apt to dwell; and I think I should not be wrong in stating that there are many managers who are under the impression that there is a growing tendency among medical officers to keep away business on the ground of albuminuria—a ground which does not always seem to them to be adequate. Although I feel greatly the honour of being allowed to address you, I must crave your indulgence, and ask you to discriminate between a paper which is voluntarily offered and one which is furnished, on rather short notice, in response to a request. Your secretary's request has been made at a period of the year when my time is very much occupied in a variety of ways, and I have been quite unable to find leisure to look up the literature of the subject and to make myself acquainted with the opinions of all the authorities who have written upon it. You must excuse my shortcomings in this respect, and I must take comfort to myself in the thought that in the discussion which is to follow other members will make good my deficiencies. This paper has been written, not with the idea that I shall teach any of our members very much, but most certainly in the expectation that I shall learn a great deal from others, and I

[1] Communicated to the Life Assurance Medical Officers' Association, 1894.

hope I am not wrong in regarding our association as a co-operative society in which the experience of each is for the benefit of all.

Let me first define what I mean by albuminuria. I would define it as the formation of a precipitate in an acid urine when it is boiled, and which remains persistent after the addition of a few drops of additional acid. This is the commonest test for albumen, one which has stood the test of time, one with which we are all intimately acquainted, and one concerning which we may fairly claim to have an adequate knowledge of the clinical import. As far as our knowledge goes, the test I have mentioned is more free from sources of fallacy than any other, and reacts with albumen and albumen only.

Of late years several other tests for albumen have received attention, and, although they are very delicate and very convenient, they precipitate peptones, albumoses, and alkaloidal bodies in addition to albumen, and I would humbly submit that our knowledge of the clinical import of these bodies in the urine is, as yet, too imperfect to enable us to base upon their presence a definite line of action in relation to the insurability or otherwise of the lives of persons whose urine gives evidence of their presence. If a medical examiner hastily rejects or loads the life of a person whose urine becomes cloudy on the addition of potassio-mercuric-iodide or picric acid it may well happen that he may keep away 'good business' from the office. If a cloud appears when these tests are used it should always be a rule to confirm the presence of albumen by the older and commoner test before making any recommendation as to the acceptance or otherwise of the life. It is necessary to say this because students and young men fresh from the schools are often rather prone to put their trust in the last new thing, and to discard the older and better understood processes, and it may well be that candidates have been reported as suffering from albuminuria when, in fact, they have been suffering from nothing of the kind.

When we are confronted with slight albuminuria we have

to determine whether or no the life is insurable. The discovery of slight and unsuspected albuminuria should, I think, make us look carefully at the candidate a second time, to be sure that we have not overlooked some of the usual concomitants of such a condition. A trifling hypertrophy of the heart, slight pallor, deficient body weight, slight increase in the tension of the pulse, ever so slight a puffiness round the ankles, a tongue slightly furred or slightly tremulous, a florid complexion or any other evidence of intemperance ; or any slight evidence of an old and perhaps forgotten syphilis at once assume an importance when joined with slight albuminuria which otherwise they would not possess.

Then, again, we probably all know the candidate who impresses us unfavourably without our being able to say very definitely why. Examiners for life assurance become like practised horse dealers. We cast an eye over the candidate and we get an instant impression from his physique and demeanour that he is likely to turn out, on examination, to be either 'sound' or otherwise. It is never satisfactory to recommend the *rejection* of a life without being able to put upon paper such reasons for the rejection as may seem adequate to the lay members of the 'board.' We must have examined not a few unsatisfactory and unwholesome-looking subjects that impress us as 'bad lives,' and yet it has been impossible to find adequate reasons for rejection. In a few of such cases slight albuminuria has solved my doubts, and I have recommended rejection on the grounds of slight albuminuria and general appearance. Sometimes a candidate is unable to furnish a sample of urine at the time of examination ; and if the examination has otherwise proved in all respects satisfactory we give him the benefit of the doubt and accept the life without examining the urine. In the case of candidates whose general appearance gives an adverse or doubtful impression I always postpone my recommendation until the urine has been examined. The discovery of slight albuminuria is thus of great importance, because it necessarily tinges all the facts of the case, including the family history, and gives

an importance to trifles which otherwise we might feel inclined to neglect.

If, on finding a trace of albumen in the urine, a second examination of the candidate fails to reveal any other adverse fact, what is to be done ? Clearly the case *must* be postponed for a second or a third examination of the urine, and I think it is important that the second examination should not be too soon after the first. An interval of at least a week, or better still a fortnight, should be allowed in order to give ample time for the subsidence of any temporary disturbance which may have caused the albuminuria. This second or third examination of urine tells us whether or no the albuminuria is *temporary* or *permanent.*

If the albuminuria is *slight* and *temporary*, and if the candidate is otherwise sound, looks strong and hearty, with no suspicion of intemperance and with no flaws in the family history, I should feel inclined to recommend him as an average risk, but, of course, I should rate him up for trifles which, under other circumstances, I might feel inclined to neglect.

If the albuminuria is *slight* and *permanent*, what is to be done ? This is a question which can hardly be answered categorically. Some lives (perhaps the majority) will undoubtedly have to be rejected, and I think we shall all agree that, if accepted, an extra premium will have to be charged.

We have all of us certainly had experience of patients who have lived for many years with albumen constantly in the urine. I have in my mind the case of a lady, aged seventy-six, whose urine has certainly been albuminous for twenty years. Dr. Francis Hawkins has communicated to the Clinical Society two remarkable cases in which life had been prolonged to extreme age in spite of persistent albuminuria of very many years' duration, and doubtless the members of this society will be able to tell of many more such cases. On the other hand, Dr. Munn, of New York, who is quoted by Dr. Hilton Fagge, has reported on sixty-nine lives rejected in the space of four years by a New York

office on account of albuminuria. Within the four years he tells us that four of the cases had died, and that the others were evidently deteriorating. Such an observation as this, apart from details, is not of much service, and I believe the question of the rejection or loading of cases of slight permanent albuminuria is one which must be left to the individual judgment of the examiner. The eligibility or otherwise of a candidate for life assurance depends on a variety of circumstances, and, while on the one hand none of these can be neglected, I think it would be unwise to allow any one fact to outweigh all the others, and act, as it were, automatically as an absolute bar.

We shall all of us probably agree that a candidate whose urine repeatedly shows the presence of a distinct amount of albumen is to be accepted, if at all, only with a considerable amount of loading.

Since it has become the custom to examine the urine of patients as a matter of routine we have come to a knowledge of the fact not only that albuminuria is much more common than was previously suspected, but that albumen may be temporarily present in the urine of those who afford no reliable evidence of kidney disease as the result of various causes, such as cold bathing, excessive exercise, injudicious diet, sexual excess, &c. Whether or no the increased frequency with which albuminuria is reported is entirely due to the routine searching for it is doubtful. That it is mainly due to that cause there can be no doubt. If we could be sure that it was entirely due to that cause our business colleagues might say with some justice : 'We made good profits in the days before the institution of such a routine. We are content with the profits we have made, and we object to have business kept away by such routine.' The calculations of actuaries are made, it must be remembered, on 'average lives,' and if everybody who passed the door of the office came in and effected an insurance no medical examination would be necessary.

In these days of competition insurance offices form no exception to the rule, and it is quite natural and quite proper that

managers of offices should jealously watch any action on the part of their medical officers and medical advisers which may appear to them to have the effect of keeping away 'good business.' A man who presents himself for insurance is a 'bird in the hand,' and if he be outwardly sound and healthy-looking our business colleagues naturally feel disappointed if such a life be rejected, and scarcely less disappointed if it be 'postponed' (with the risk that he may go to a competing office) because the urine shows a cloud of albumen when it is acidified and boiled.

Some offices are inviting bad lives by doing away with their medical watch dogs, and are holding out the inducement of 'no medical examination.' This is a very hazardous policy, for it is idle to shut our eyes to the fact that minor degrees of fraud are not uncommon among candidates for insurance. False statements, doubtless, are not infrequently made from ignorance or carelessness, but equally, undoubtedly, they are often made intentionally. When a man insures for the sake of making provision for his wife and children he is tolerably sure to think twice before he makes any untrue statement which may invalidate the policy ; but the man who is insuring for the protection of a money-lender is less likely to be so scrupulous, even if we set aside the fact that these persons often belong to a class which, to put it mildly, is little troubled by scruples. Those who are driven to their wits' end to raise money do not hesitate to become mildly fraudulent, and it is well to remember an old proverb to the effect that 'it is difficult for an empty purse to stand upright.'

It must by no means be forgotten that the increasing frequency of albuminuria among candidates for life assurance may be due to causes of far more serious import than the mere fact of the establishment of a routine among the examiners of lives. We must not forget the fact that the expectation of life in males after twenty years of age was, according to Dr. Ogle, less in the decade 1871–80 than in the period 1838–54, which was used for Dr. Farr's life table. The great majority of those who insure their lives are males over twenty, and the fact that the expectation of life in this

particular section of the public is tending to decrease is one which insurance offices cannot afford to neglect. This fact is brought out very forcibly in the annual reports of the Registrar-General, who gives the death rates at twelve age periods for five successive decades between 1841 and 1890. It will answer my purpose and make the matter less complicated if I give the results for the first and last decades only. It will be seen that the decrease of the death rate in males, which has been so remarkable during the last half-century, is most marked in the first three decades, after which there is a considerable increase up to the age of seventy-five :

ENGLAND.

Death Rates at Twelve Age Periods.—Males.

—	0-	5-	10-	15-	20-	25-
1841–50 .	71·2	9·2	5·1	7·1	9·5	9·9
1881–90 .	58·7	5·3	3·0	4·4	5·8	7·9

—	35-	45-	55-	65-	75-	85-
1841–50 .	12·9	18·2	31·8	67·5	148·3	312·3
1881–90 .	12·5	19·7	34·0	71·3	146·5	305·8

It is not unimportant to notice that this increase in the death rate of males in the prime of life is far more marked in urban than in rural places, for I suppose one may take it for granted that the urban population furnishes more candidates for insurance than the rural population. Let us take the age period of forty-five to fifty-five, and see how the death rate for this period varied in different localities for the year 1890 (the returns for which I happen to have by me). The death rate of males between forty-five and fifty-five in the whole of England was, in 1890, 19·2 ; in London, 22·8 ; in Lancashire, 27·1 ; in the West Riding of Yorkshire, 22·9 ; in Wiltshire, 14·1 ; in Dorsetshire, 12·0 ; in Huntingdon, 9·4. These are facts which cannot be neglected, because they show an increasing mortality among the class from which candidates for life assurance are largely drawn.

Again, let me point out that the death rate from diseases

of the urinary system is tending steadily to increase. The annual death rates per 1,000,000 persons living from diseases of the urinary system for the six quinquennial periods intervening between 1861 and 1890 were as follows : 246·2, 286·8, 326·4, 374·8, 422·2, 446·6. Since 1858 the death rate from urinary diseases has doubled. A part of this increase in the Registrar-General's returns of the death rate from urinary diseases may be accounted for by increased knowledge and change in nomenclature, but I take it that it is not all due to these causes, and we should be very wrong to neglect so startling a fact. Clearly, then, 'albuminuria' is a matter which demands very careful consideration by examiners for life assurance and also by managers and actuaries.

Seeing that we have always to be on our guard against mild forms of fraud, it is necessary to be quite sure that the urine we examine is the urine of the candidate for insurance. The urine must always be passed in the presence of the medical officer, and I may as well state that I am practically certain that on two occasions an attempt has been made to deceive me. One person, I believe, brought a small quantity of urine in a test-tube which he surreptitiously tilted into the urine vessel (he was a chemist), while another handed me a sample of urine which was cold and thick, and which I have no doubt he brought in a six-ounce bottle concealed in his pocket. Both were rejected, and it was ascertained that the latter had been previously rejected by another office for albuminuria.

The urine of women is far more difficult to form an opinion upon than the urine of men, because there is a periodic cause which may give us slight albuminuria, not to mention so frequent a trouble as leucorrhœa. Again, it is not possible to remain in the room while a lady is furnishing a sample of urine, and thus the fraud of urinary personation, if I may call it so, is more easy for a woman than a man.

It is to be regretted that we have no means of getting the after-histories of persons rejected for albuminuria. This seems to me to be necessary before we can come to any very decided opinion on the subject. I am, however, unable to suggest any method by which such after-histories can be obtained.

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CHAPTER XV

A CASE OF CONGENITAL HEART DISEASE [1]

THE patient whose case we have to consider is a child (William F.) aged four years. He is suffering, and has suffered from birth, from 'cyanosis'; and a glance is sufficient to tell us that he has what used to be called 'the blue disease.' He was admitted the day before Christmas (December 24, 1895), and this is the third time within a year that he has been in this hospital. In the early spring he was admitted to the children's ward, and while there he developed *measles* and was discharged. After his measles he suffered from *whooping cough*, and recovered from it without difficulty. On May 20 he was admitted a second time into the hospital under my care. At this time he was dangerously ill with high fever, the temperature 104° F., the pulse 160 and over, and the respirations 52. This febrile attack lasted some three weeks and gradually subsided. There was a cloud of albumen in the urine during its height, there was some enlargement of the spleen, no throat trouble, some slight bronchial catarrh, no pneumonia, no diarrhœa, no characteristic eruptions. It seemed to run a definite course, and the child made a good and complete recovery. That it was an acute fever due to an infective process one could not doubt, but as the materials for a more exact diagnosis are wanting we will call it 'simple continued fever.' The patient made a good recovery and was sent to a convalescent home. He has been in fairly good health from the time of his discharge, in June, until his third admission here on December 24, when he was suffering from urgent dyspnœa, and within a few days of

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[1] Clinical Lecture, U.C.H., 1896.

his admission developed typical chicken pox, from which he has completely recovered.

I have dwelt upon these facts because it is of interest to note that this child of four, with serious congenital heart disease, has passed through four severe 'acute specifics' in a twelvemonth, and has recovered from them quite as readily as a normal child. The parents and all the brothers and sisters of the patient are reported as alive and healthy.

The patient's trouble is 'breathlessness.' While lying in bed or sitting quietly in a chair the child is quite comfortable, and laughs and plays with his toys in a state of evident enjoyment. Any attempt at exertion brings on extreme shortness of breath, accompanied by marked lividity of the skin. These attacks of breathlessness are at times alarming, and more than once have caused us to think that death was imminent.

The child is fairly well developed and nourished. It has a slight 'pigeon breast,' but beyond this there is nothing which calls for remark excepting that *the fingers and toes are excessively clubbed*, and the long diameter of the nails is far more convex than usual.

This clubbing of the fingers and toes and the lividity of the lips, ears, fingers, and toes, combined with the statement that the child has always been blue from birth, and has always been liable to attacks of dyspnœa, make us certain that we have to do with a condition of congenital heart disease.

When we proceed to make a methodical examination of the organs of circulation we notice on *inspection* that the beat of the heart is nowhere visible, and that, in spite of the cyanosis, there is no distension of any venous trunk either in the neck or elsewhere.

On palpation we find the apex-beat of the heart is faintly perceptible in the fourth, fifth, or sixth interspaces between its normal situation and the nipple line. No thrill is anywhere to be felt, and no trace of œdema is anywhere to be detected throughout the body.

On percussion we find that the cardiac dulness extends from the left edge of the sternum to a parallel line slightly

outside the left nipple, while its upper level is at the upper edge of the fourth rib. So far, therefore, the physical signs are those of slight enlargement of the left side of the heart.

On auscultation we hear all over the cardiac area a loud, blowing, systolic murmur, which reaches a maximum of loudness and harshness at the junction of the second left costal cartilage with the sternum. Under ordinary circumstances we might feel absolutely certain that the lesion in the case before us consisted in stenosis of the pulmonary artery. It is, indeed, the only diagnosis which we are warranted in making, and, judged by the statistical records of lesions observed in similar cases, it is probably the correct diagnosis. But when one is dealing with a condition which is certainly congenital it must always be borne in mind that the malformations of the heart brought about by faults of development are so varied and so bizarre that even with the organ before one it is often difficult to say which cavities and blood-vessels merit the names which are used in relation to the normal heart and vessels.

When we find, as in the present instance, that the physical signs point to a lesion of the right side of the heart, this may be taken as affording in itself strong presumptive evidence that the lesion was formed while the patient was still *in utero*.

These lesions may be caused by defects of development or by definite endocarditis occurring *in utero*. Endocarditis occurring after birth almost invariably attacks the left side, but when it occurs before birth it almost invariably attacks the right side. This is a fact which has to be accepted, but the explanations hitherto offered have not been altogether satisfactory. Before we can pursue this subject further it will be well to glance at a few facts connected with the fœtal circulation, which the accompanying figures may help us to comprehend more readily.

In the fœtus you will remember :

1. That the blood of the superior vena cava—true venous blood from the head and upper limbs of the fœtus—passes (as in the adult) through the right auricle to the right ventricle and the pulmonary artery. The lungs not being in action, very little, if any, enters the branches of the pulmonary artery, but the whole, or nearly the whole, is conducted to

the descending aorta through the ductus arteriosus or ductus Botalli.

2. The blood of the inferior vena cava—placental blood, some of which has passed through the liver of the fœtus—enters the right auricle, and is conducted by the Eustachian valve directly through the foramen ovale into the left auricle, and thence, as in the adult, to the left ventricle and aorta.

You will see, therefore, that the right ventricle of the heart, as in adult life, is filled with venous blood, while the placental blood, which may be regarded as quasi-arterial, passes direct through the foramen ovale to the left side of tne heart, as in the adult.

Further, it is evident that the work of the two fœtal ventricles cannot be far from equal, a proposition which is supported by the fact that the thickness of the ventricular walls at birth is equal, or nearly so. The liability of the right cavities of the fœtal heart to suffer from endocarditis is not to be accounted for by the character of the blood supplied to them, nor by the fact that, being more worked than the left cavities, they are more liable to disease. Unless the pressure exerted by the two halves of the ventricular portion of the heart were equal, or nearly so, it is difficult to believe that the dividing septum, which grows gradually between the right and left ventricles, could undergo its regular development.

So far, then, the physical signs in our patient point to 'pulmonary obstruction,' which is the lesion most frequently found in those patients with congenital heart disease who have succeeded in living for any considerable time.

One would expect that an obstruction of the pulmonary artery would lead to great hypertrophy and dilatation of the right ventricle, with damming back of the venous blood, general œdema, and obvious distention of the systemic veins throughout the body. But in the patient before us there is no venous engorgement or œdema, and no evidence whatever that the right ventricle is enlarged. There is, however, some evidence of enlargement of the left side, but we must remember that we may be quite wrong, because anomalous conditions are not to be judged by ordinary rules.

You will bear in mind that in the fœtus the blood coming to the right auricle has two courses open to it, (1) through the tricuspid orifice to the ventricle and pulmonary artery, and (2) through the foramen ovale to the left auricle, and, there being two channels of escape from the right side of the heart, we shall find that the redundancy of one compensates for deficiencies of the other. If our assumption in the case before us be correct—that there is a pulmonary stenosis—it is evident, from the absence of all signs of venous engorgement, that the blood finds another channel of escape, and the probability is that the foramen ovale is widely open. If we could be sure that the endocarditis, which presumably has partially closed this child's pulmonary artery, occurred in the later weeks of intra-uterine life, after the completion of the septum ventriculorum, we might feel perfectly sure that the foramen ovale is patent ; but as we cannot be sure that the foramen ovale is patent, and as the endocarditis may have occurred at an earlier period, before the closing of the septum ventriculorum, we must admit as an alternative that this septum may be deficient. A third and by no means improbable suggestion is that the blood from the right side of the heart passes partly through the foramen ovale, partly through the defective septum ventriculorum, and partly through the stenosed pulmonary artery. A reference to the diagrams will show clearly how a stoppage of the normal channels in fœtal life leads inevitably to the permanency of the fœtal channels.

Fig. 4 is intended to bring before you the normal circulation of the fœtus shortly before birth, with the patent foramen ovale, the ductus arteriosus, and as yet undeveloped branches for the lungs.

Fig. 5 shows the circulation shortly after birth, with developed branches for the lungs, closed ductus, and closed foramen.

Fig. 6 shows the effect of complete occlusion of the pulmonary artery, entailing a patent foramen (or septum ventriculorum, or both). In this case the branches for the lungs are supplied through the aorta, and the ductus remains patent. A very few cases have been recorded in which a fatal endo-

carditis has blocked the tricuspid orifice, a lesion which entails
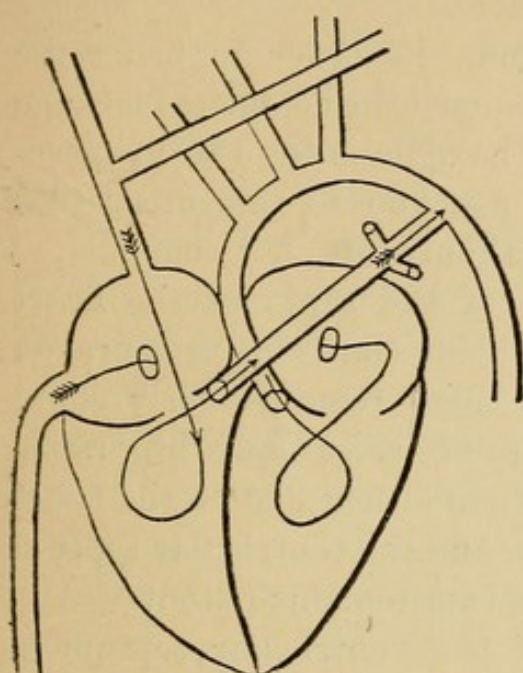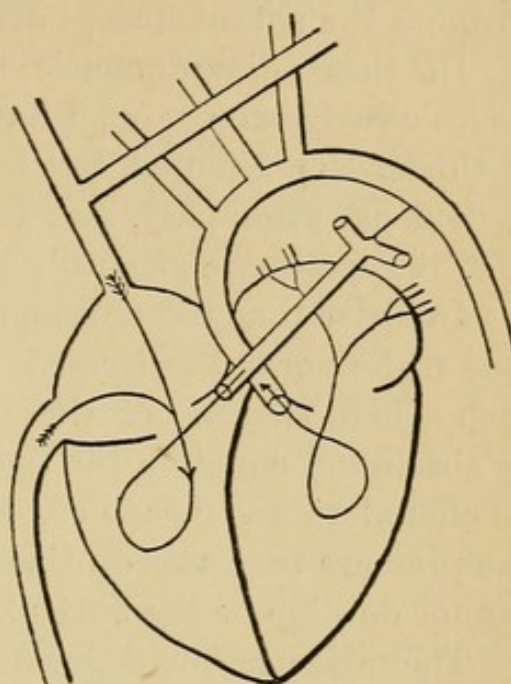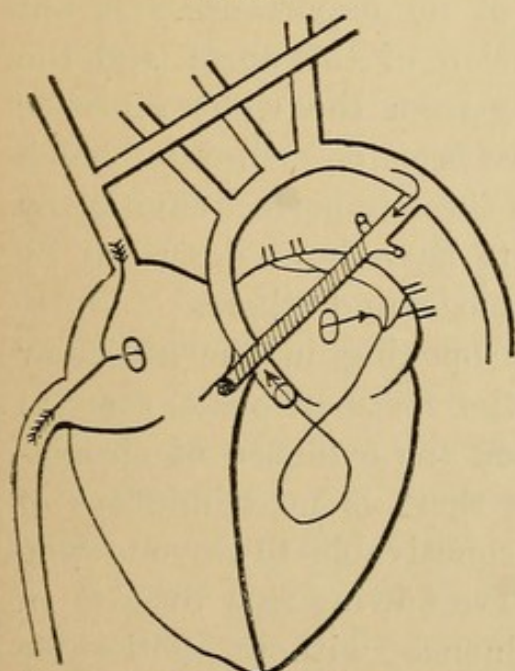patency of the foramen and ductus.

FIG. 4.

Diagram of the normal fœtal circulation
through the heart just before birth.
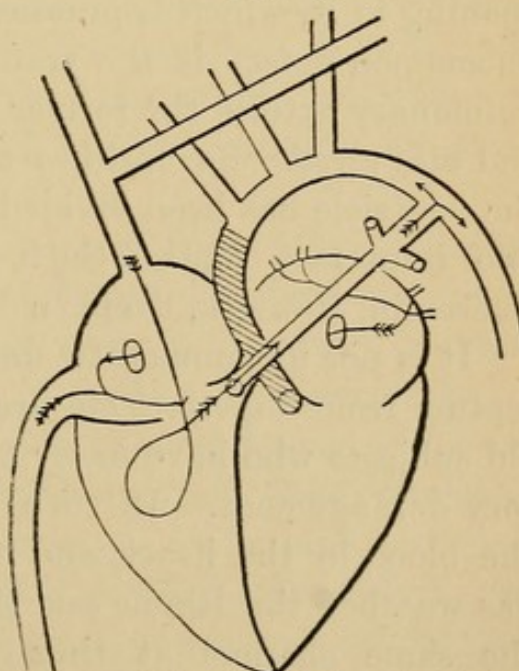
FIG. 5.

Normal condition after birth. The fora-
men ovale and the ductus arteriosus are
both closed.

FIG. 6.

Abnormal obstruction of the pulmonary
artery.

FIG. 7.

Abnormal blocking of the arch of the
aorta.

Fig. 7 shows the effect of obstruction on the left side of
the fœtal heart. We have supposed a complete blockage of

the aorta. In this case the blood, being unable to escape from the left side, travels through the patent foramen (or septum, or both) to the right side, and the aorta is supplied through the patent ductus arteriosus.

In these diagrammatic schemes, in order to bring the issues clearly before you, I have assumed the complete blockage of this or that channel, but this I have done for the purposes of demonstration only, and I will ask you to remember that such blockages are generally partial only, and not complete.

In order that the development of the septa of the heart may make normal progress it is evident that the pressure on each side of the line of the septa must remain nearly equal or absolutely equal on the two sides of it. If any hindrance be offered to the escape of blood from either side of the heart the pressure increases on that side, and the ventricular septum cannot develop or the auricular septum remains patent.

The absence or deficiency of the ventricular septum is shown in none of these diagrams, although it is a very common defect The septum may be entirely wanting, so that there is practically only one ventricle, or there may be only a small opening in it, which is practically of no importance. A not uncommon defect is the transposition of the aorta and the pulmonary artery, the former rising from the right ventricle and *vice versâ*. Several hearts have been described in which the ventricle has been divided into three imperfect cavities by two imperfect septa. Such hearts have been described by Théremin, Peacock, Stephen Mackenzie, and others.

It is not uncommon to find an opening in the auricular septum from imperfect closure of the foramen ovale, even in old subjects who have never afforded any evidence of circulatory derangement. In such cases there is no admixture of the blood in the heart, and it is conceivable to anyone who has watched the Rhone and the Arve flowing side by side in the same channel at their confluence without noticeable admixture that there may be very little admixture of blood even with considerable ventricular deficiency, provided the muscular strength of the cavities on either side of the defect is tolerably equal, and the normal outlets from each ventricle

are open. Thus it has happened that considerable defects have been found post-mortem where there has been no appreciable cyanosis during life.

As to the cause of the cyanosis, I think there can be no doubt that it is due to imperfect aëration of the blood, as asserted by Lees.

In the normal adult heart no blood is driven into the systemic arteries that has not been thoroughly aërated.

If the pulmonary artery or the aorta be completely blocked, then the aëration of the blood which is driven into the systemic vessels cannot be more than half the normal amount. When the aëration of the blood is hindered, as in cases of dilated right heart with emphysema and bronchitis, we get cyanosis, but the cyanosis is in these cases largely due to an increase of the blood-pressure in the veins, leading to their over-distention, and it is probable that this over-distention is the chief cause of the evident blueness of the complexion. I would, in passing, remind you that cyanosis may be caused by the administration of certain bodies which are now much used as medicines, such as aniline, antipyrin, exalgine, and antifebrin. The cause of the cyanosis produced by some of these antipyretic coal-tar products is uncertain. There may be some paralysis of the hæmoglobin analogous to what takes place in poisoning with carbon monoxide, but there is usually no marked dyspnœa.

In cyanosis from congenital heart disease there is always more or less dyspnœa, and, although this symptom may not be noticeable when the patient is at rest, it generally becomes urgent when any extra exertion is made. Dyspnœa may, of course, occur without venous obstruction, as we see every day in cases of ordinary anæmia. Sufferers from this congenital trouble are obliged to lead very tranquil, sedentary lives.

It is to be noticed that in the case which forms the basis of our lecture to-day the temperature, when not febrile, has been normal. Body temperature, we must remember, depends on many factors, of which oxidation is only one, and even though this factor of oxidation may be below the normal, this may be compensated by diminished loss of heat. Cases of

congenital cyanosis have been recorded in which the body temperature was habitually below the normal.

The temperature of our patient, apart from the definite attacks of acute specifics which have been mentioned, is suspiciously unsteady, and repeatedly has run up to 100° F. and over. One cannot but fear that tuberculosis may be the cause of this, although we may have been unable to find any more definite evidence of this condition. Tuberculosis has frequently been recorded in conjunction with congenital cyanosis, and there is no justification for the statement which has been made that these conditions of the heart are antagonistic to tuberculosis. This child has another symptom which is very commonly but not universally present in congenital heart trouble—viz., well marked clubbing of the fingers and toes. The second toe on each foot is shaped like a clove, the end of the toe being irregularly globular. The finger nails (and to a less extent the toe nails) are very convex in their long diameter.

This clubbing of the fingers and toes is often said to be due to venous congestion, which thus makes itself evident at the extremities, but we must remember that it is not observed in persons who have swollen legs, the result of tortuous varicose veins, nor is it seen in cases of bronchitis and emphysema with dilated right heart and obvious general venous engorgement.

In phthisis we get the curving of the finger nails, but it is not common to see clubbing in cases of even advanced phthisis. It is very often seen, however, is cases of empyema, but is not one of the recorded symptoms of ordinary serous pleurisy. In empyema it may appear very rapidly after suppuration has commenced, and it is said to disappear when the thoracic abscess has been drained and cured. The clubbing seen in empyema does not appear to have any very definite relation to the amount of dyspnœa caused by the embarrassment of the lung.

I am very much in doubt whether the clubbing is due (entirely, at least) to venous engorgement. In this child there

is no evidence of venous engorgement, but the clubbing is very marked.

It is right, however, to mention that Dr. J. W. Ogle recorded ('Pathological Society's Transactions,' vol. x. p. 103) a case of clubbing of fingers, with cyanosis of the tips and great hypertrophy of the nails, on the right side only, due to the pressure of a large subclavian aneurism on the axillary vein, thereby causing an 'exalted nutrition' of the finger tips.

Mr. Canton also ('Pathological Society's Transactions,' vol. xvii. p. 428) recorded the case of a negro who had clubbing of the fingers of the right hand only, which is also attributed to the pressure of a subclavian aneurism on the axillary vein.

Dr. Lees, who recorded a case of transposition of aorta and pulmonary artery, with a deficient septum ventriculorum, which had marked cyanosis of the head and upper limbs without a trace of clubbing, expresses his opinion that clubbing is due to venous congestion, and its absence in the case which he records ('Pathological Society's Transactions,' vol. xxxi. p. 62) is attributed to the fact that no obstruction to the return of blood from the systemic veins was present. Dr. Lees's patient was only seven months old at death, and it was not until the fifth month of life that a systolic murmur at the left base was developed.

I am of opinion that venous obstruction cannot be regarded as the sole factor in the production of clubbed digits. The true explanation remains to be found.

As regards the *treatment* of congenital heart disease, not much need be said. The condition is essentially incurable, but many of the patients manage to live on for considerable periods. One case at least died at over sixty years of age, and a case recorded by Dr. Stephen Mackenzie died at the age of thirty-nine, after leading a fairly active and useful life.

It need hardly be said that febrile diseases are usually dangerous to such patients, and that any pulmonary disorder which interferes with the physiological action of the lungs greatly increases the discomfort and liability to attacks of dyspnœa. During his recent attack of chicken pox our little

patient was intensely cyanotic and breathless, and appeared to be much relieved by inhalations of oxygen.

The arrangement for a tranquil life, the avoidance of all excitement and exertion, and the protection as far as possible from intermittent maladies are the lines of treatment to be ordered, but beyond this one can say very little.

CHAPTER XVI

THE MANAGEMENT OF CHRONIC HEART DISEASE [1]

IN dealing with sufferers from chronic heart disease we must not allow the fact that the lesions of the valves due to endocarditis are certainly permanent, to take too strong a hold of our minds. Whether the valvular lesions be permanent or otherwise, there is nothing more certain than the fluctuations which take place in the condition of sufferers from chronic heart disease. There are many such who may be spoken of as among the permanent clients of this and of every hospital. They are admitted in a state of great suffering and danger, during their stay in hospital they improve, and recover sufficiently to go out and pursue their occupations for a time ; but after a few months we are tolerably sure to see them back again, and so these sufferers spend their lives in a painful endeavour to fight the battle of life with damaged organs.

Let us take the common case of a sufferer from mitral disease with thickening and deformity of the valves permitting regurgitation through the mitral orifice with each systole of the ventricle. With this condition of the mitral there is often some thickening of the aortic valves as well, which offers obstruction, perhaps slight, to the outflow of the blood into the aorta.

The suffering of the patients in this condition necessarily depends upon the amount of valvular disease and the amount of work which they have to do. There are many patients of this class engaged in quiet pursuits who go on for years, and manage to do a large amount of useful work. As long as they are not hurried, and are able to 'take their time,' they do very well ; but a little over-exertion or any failure of general

[1] Clinical Lecture, U.C.H., 1893.

health is a severe trial to them, and often brings their life
into jeopardy.

In these cases there is always an accentuation of the
second sound over the pulmonary cartilage, indicating increased
tension in the pulmonary artery. This is the key to the
danger, as indicating that the encumbered left ventricle is
throwing extra strain upon the lungs and pulmonary
artery. The failure of the left ventricle to completely propel
its contents in the proper direction necessarily leads to an
engorgement of the lung and increased pressure on the right
side of the heart and the venous system generally.

We must never fail to regard the heart and lungs as one
organ, and to remember that the lungs are in reality placed
between the two halves of the heart. We must bear in mind
that grave damage to the circulatory part of this compound
organ must affect the pulmonary part, and *vice versâ*.

I want especially to say a few words on the hygienic
management of patients with chronic heart disease, and for
this purpose I will select, out of several cases which have
lately been in my wards, one which, in particular, has given
us proof of the good results of such management.

W. G., aged 41, bricklayer, admitted June 1, 1892. Had
scarlet fever fourteen years ago. Works hard, and is a good
deal exposed to weather ; drinks moderately, and has had
syphilis. About four years ago he first noticed that his legs
swelled, and that he was very short of breath on exertion.
At this time he was an out-patient of the Middlesex Hospital.
Two years ago he was a patient at the Royal Free Hospital
for the same condition, so that when here he was paying his
third visit to a hospital in four years for shortness of breath
and swelled legs.

The patient, on admission, complained of swelling of the
legs and abdomen, shortness of breath, and a difficulty of
passing water, the urine only amounting to half a pint in the
twenty-four hours. This condition of things began a month
previously, and had got steadily worse.

The physical examination of the patient revealed the
following facts : marked œdema of the legs, scrotum, and

trunk, with slight indications of fluid in the peritoneal cavity. The lungs were big, slightly emphysematous, and overlapped the heart, so that the size of the heart could not with certainty be made out. There was a loud mitral murmur. The heart sounds were somewhat feeble. The pulse rather hard. The urine was scanty (less than a pint in 24 hours), high coloured, sp. gr. 1,022, and contained a considerable trace of albumen, but no casts. The tongue was flabby and slightly furred.

Here, then, we have an ordinary typical case of chronic heart disease, with slight emphysema of the lungs, and, probably, very slight chronic kidney trouble as well. This is the third attack, and what we have to ask is, Why do these attacks recur? Why, if they be due to heart disease, do they get practically well? When one gets a complete circle of symptoms, as in this case, it is often difficult to say what part of the circle is most important, but I should look to the varying conditions of the left ventricle as most likely to afford an explanation of the trouble. We have, probably, some slight thickening of the mitral valve, caused by endocarditis, occurring as a complication of scarlet fever fourteen years ago. This condition does not seem sufficient to cause him any annoyance under ordinary circumstances. If, however, from any cause, the left ventricle dilates, then the mitral incompetency becomes serious, the tension in the pulmonary artery rises, the right side of the heart dilates, and the venous system throughout the body becomes engorged.

The left ventricle will dilate (1) from weakening of the cardiac muscle, so that it becomes incapable of withstanding its normal amount of internal pressure. This might arise from anæmia, the result of insufficient food or debauchery, from fatty degeneration, or from the weakening effect of high body temperature, the result of a febrile attack such, for example, as influenza.

(2) The ventricle may dilate in consequence of over-exertion and excessive effort in the course of his ordinary occupation, or as the result of some accidental occurrence necessitating effort.

(3) The ventricle may dilate in consequence of obstruction to the outflow of blood, either by a narrowing of the aorta or by a rise in the tension of the systemic arterial system generally, an occurrence which is very liable to take place in those who have a gouty condition of the kidneys.

As our main object in the treatment of such cases is to get the dilated ventricle to resume its normal dimensions, it becomes very necessary to bear in mind the possible causes which may determine its dilatation.

When we look at a patient such as the one with which we are dealing we have to remember that the outward condition is the evidence of the inward condition. The encumbered circulation gives us the œdema of the lower limbs and trunk. This œdema, although most marked in the dependent parts, is due to a central cause, and as a consequence every organ in the body is more or less congested—lungs, stomach, and intestines, liver, kidneys, and brain. As a result of this every organ performs its duty imperfectly. There are difficulties of primary digestion, metabolism, defæcation, urination, and at night when the head is somewhat lowered in position the patient wanders instead of enjoying sound sleep. Evidence of this inefficient action on the part of the kidneys is afforded by the urine, which is scanty and slightly albuminous. In the state of the kidneys we have evidence of the vicious circle in which a patient may move. The condition of the heart causes an engorgement of the kidney, the engorgement of the kidney and the faulty purging of the blood of the products of nitrogenous metabolism increases arterial tension, and this in its turn increases the tendency of the left ventricle to dilate. Do not let us forget that, in the general venous engorgement, the heart does not escape, and that, its nutrition being interfered with, the tendency to dilate increases equally with the active causes of dilatation.

There is room for doubt in this case as to whether the condition of the urine is or is not entirely secondary to the heart disease. The main reason for leading me to believe that there is a chronic kidney change independent of the heart

trouble is the fact that the dropsy is rather out of proportion, if one may say so, to the other symptoms ; and this
belief that the kidneys are the seat of independent trouble is
rather strengthened by the history of scarlet fever as a very
probable cause of chronic kidney mischief, and by the fact
that when the patient left the hospital there was still a slight
trace of albumen in the urine. If, however, there be a
chronic induration of the kidney apart from the heart trouble
it must be very slight.

In the treatment of such a case and in the endeavour to
bring about a diminution in the size of the left ventricle,
clearly the first indication *is rest* as absolute as possible. The
circumstances of the patient must be such that the heart has
only the internal work of the body to grapple with, and to
this end he must be put to bed and ordered to remain there. I
think it is most important that the patient should remain in
bed during the whole 24 hours, for if he be allowed to get up
and make any effort which materially quickens the action of
the heart he may undo in ten minutes the good which may
have been gained during the previous hours of rest. Nothing
is so damaging to a weakened heart as any muscular act
accompanied by 'effort,' i.e. an act which necessitates the fixation of the thorax and the suspension of the respiratory act.
When we see a man strain (whether it be at a massive act,
such as lifting a sack of flour, or a trivial act such as cracking
a walnut with the fingers), and watch the face get purple and
the veins stand up upon the forehead, we cannot fail to be impressed with the strain which is put upon the organs of circulation by the act. No person with chronic heart disease,
especially if he be under medical treatment, is likely to attempt
any massive acts involving strain, but I must remind you
that among minor acts which often necessitate effort is the act
of defæcation, and it becomes very important to protect the
patient from the evils of straining at stool and to keep the
bowels moderately relaxed by the aid of gentle purgatives.

If the heart is to have the greatest amount of rest attainable the patient must be kept comfortably warm and be fed
upon a sufficiency of food, while every precaution is taken

against giving any excess. If the temperature of the body be not artificially maintained in cold weather more food must be taken, and the more food that is taken the more work the heart has to do. The digestive and assimilative powers are both lowered, and it becomes necessary to give food in the most digestible form. The patient must be put upon a strict spoon diet, but not too fluid, and it may be advisable to use peptonised food for a time.

In such cases as these it is sometimes difficult in private practice to prevent injudicious friends from stuffing the patient with turtle soup, wines, and various delicacies, in the mistaken belief that he can be 'fed up.' No greater mistake can be made, for not only is the patient placed in circumstances which necessitate a minimum of food, but the engorged viscera are barely capable of dealing with that minimum. Nothing is so prone to produce palpitation of the heart, even in health, as the administration of an excess of food. If the food lie undigested in the stomach it may produce irritation and reflex palpitation, or if it decompose and undergo fermentation it may produce flatus, which will not only set up reflex palpitation, but cause direct encumbrance of the heart as well by distention of the stomach. Or if it be digested and absorbed, and if elimination of the waste by the kidneys do not keep pace with absorption, we get the blood overcharged, increased tension in the systemic arteries, and palpitation of the heart as a consequence. I am inclined to think that, on the whole, excess of food does most harm if it be absorbed, and that the midnight palpitation of gouty subjects is, broadly speaking, due to the fact that eliminative processes do not keep pace with absorption.

I think that as a rule the best guide for the administration of food is the patient's appetite, and that if there be, as often is the case, a purple furred tongue and a disagreeable taste in the mouth it is unwise to push the feeding. While the patient is in bed endeavouring to get the physiological rest for his heart he must have nothing but spoon food, which should be given in small quantities and at frequent intervals. The bowels must receive careful attention, and if the urine be

scanty and albuminous a rather free flux from the bowel will do nothing but good. Anything like violent purgation must be avoided, but it must also be ever present in the mind that constipation is sufficient *per se* to produce functional disturbance of the heart, and that in the treatment of chronic heart disease the care of the bowels is all-important.

But to resume the history of the case with which we are dealing. He was admitted with cardiac dropsy, a mitral murmur, and scanty albuminous urine on June 1, and he was put to bed and placed on a spoon diet. In addition to this, his prescription paper will show you that he had one dose, and one only, of purgative medicine, and not a single dose of any cardiac tonic or any other drug.

What was the result? Perhaps this is best answered by a reference to the urine chart, which shows that on the first day of admission the amount passed was 17 oz., and the next day only 20 oz. After this the quantity gradually increased until, some ten days after admission, it amounted to 168 oz.

As the quantity rose the specific gravity fell, and with the increase of the flow of urine the dropsy gradually disappeared.

The urinary flow having reached a maximum, gradually declined again, until the physiological quantity of 48 oz. in the twenty-four hours was reached. All the other symptoms improved, and on June 22, on which day the patient was discharged, *the mitral murmur was no longer audible, the only sign of dropsy was a slight pitting on firm pressure over the sacrum, and the urine was normal in amount and specific gravity, and contained only a very faint trace of albumen.*

This patient was practically cured ; and if his condition of life had been such as to allow of his making health his main object in this world one would have been justified in predicting still further improvement in the course of time. As it is, one must, I fear, predict that sooner or later he will reappear here or elsewhere with a recrudescence of his chronic trouble.

The case is, as regards symptoms and physical signs, a very ordinary one, and yet I have thought it advisable to bring it very specially to your notice as being worthy of more than ordinary attention.

Why did the man, who was admitted suffering from a considerable suppression of urine, manifest such copious diuresis after being a few days in bed ? The partial suppression of urine was due to the venous engorgement of the kidneys, the result of his dilated left ventricle, and the regurgitation through the mitral orifice, causing increased tension in the veins throughout the body, inclusive of those of the kidneys.

When the heart was put to rest, and had only the internal work of the body to do, the strain on the left ventricle diminished, the blood was driven forwards into the capillaries with hourly increasing force, and driven backwards into the auricle with a force which hourly diminished. Thus the arterial tension was maintained while the venous tension diminished, and the kidneys were soon able to perform their functions in a normal manner.

But, you will say, the function of the kidneys became more than normal ; it was excessive, for the diuresis was extreme. This is true, and the cause for it is to be found in the large amount of anasarca—the large quantity of watery fluid which had to be removed from the body.

Dropsical fluids vary much in specific gravity ; those which are caused by inflammatory processes, as in pleurisy and peritonitis, have a specific gravity of 1018 or higher (Halliburton), while non-inflammatory transudations have a specific gravity of 1015 or lower (Halliburton), and of these latter the fluid of œdema in cardiac dropsy has the lowest specific gravity (1010). With enough fluid exuded into his cellular tissue to cause marked œdema of the legs and trunk it is not to be wondered at that when the kidneys became sufficiently disengorged to perform their functions that the diuresis became excessive.

Now, in cases like the one with which we are dealing we commonly follow a routine ; and when we are confronted with a case of cardiac dropsy we prescribe, almost automatically, one of the common cardiac tonics and diuretics, such as digitalis, or strophanthus, or citrate of caffeine. If either of these drugs or any other drug having a reputation as a diuretic had been given we should, perhaps, have been quite ready to ascribe all the improvement which took place to the drug which had been

administered, and we should have said that, certainly, it was a powerful and valuable remedy. But the only cardiac tonic and diuretic which this patient had was rest—rest which was afforded by his bed and his diet. In clinical therapeutics it is always impossible to get rid of the *post hoc* fallacy. Of all therapeutic agents rest is probably the most important. This patient's organs were unable to functionise normally, because they were being over-pressed ; we gave them rest, and their function was resumed.

Tonics and stimulants are therapeutic agents which are given with the object of, so to say, spurring an organ to make increased efforts, but we must remember that ' action and re-action are equal and contrary,' and that after stimulation there is the reaction of depression. It is sometimes very desirable and very useful to give stimulants in order to enable a patient to tide over a crisis ; but you must remember that, as the spurs alone will not keep a horse going, unless there be intervals for rest and nourishment, so cardiac tonics are of no permanent use unless it be found possible to give the organ rest as well.

It very often happens that a few doses of digitalis will do great good by enabling a heart to systolise with sufficient energy to commence grappling with its difficulties ; but it is my invariable custom, unless the patient's condition is one of urgency and danger, to delay the administration of cardiac tonics until a few days' rest have enabled us to see whether or no the organ has sufficient muscular power to overcome the obstacles which are hindering it, without artificial help. If, as in the present case, the heart recovers with rest alone, I am sure it is a great gain for the patient. No one is more conscious than I of the great use of drugs in cases of heart disease, but I am sure that it is well to do without them if such a course be possible.

A few words may be said as to the general advice which should be given to sufferers from heart disease.

There be many such who can scarcely be said to need such advice, their condition being one of chronic dyspnœa, which makes exertion or excess of any kind an impossibility. There

are, however, many persons with damaged hearts who are either quite ignorant of the fact or who suffer so little inconvenience that they might, if properly managed, remain practically unconscious of their trouble.

Take the common case of a young man with a mitral systolic murmur. He is brought to me, possibly, because he is 'out of sorts,' and in the course of examination the mitral murmur is discovered. Do not on any account tell such a patient bluntly that he has 'heart disease.' Your patient does not understand pathology, and the word 'heart disease' is in his mind associated with sudden death and coroners' inquests. The shock of hearing such intelligence may do him definite harm, and certainly the after moral effects are likely to be serious. You must convey to him the idea that his circulatory organs have been a little overstrained, and you must impress upon him that if he be careful of himself he will probably practically recover.

The main indication in all these cases is to regulate the pace of living by the condition of the damaged organ.

Everything which involves hurry or effort is to be avoided. Moderate exercise does nothing but good, but such patients ought never to work themselves into a state of 'breathlessness' or anything approaching it, and I think that, for such, all games and exercises involving competition are to be absolutely disallowed. Gentle rowing will do good, but anything in the shape of boat racing is most harmful. Such patients must never play 'matches' of any kind, because when one is associated with others in games and exercises one is in honour bound to make every effort, and put forth one's whole strength, and it must be remembered that a single serious muscular effort may so strain a damaged heart that the patient, instead of being practically unconscious of his condition, may find himself permanently short of breath.

For sufferers such as these competitions of all kinds, both mental and physical, should be disallowed.

The great danger which is run by a sufferer from mitral disease is from cold. The lungs are permanently congested, which makes them very susceptible to the effects of cold,

and an attack of bronchitis is sure to overstrain the right side of the heart by increasing the already slight difficulties of the pulmonary circulation. A sufferer from mitral disease may be unconscious of his trouble, but after 'catching cold' he may come to us complaining of puffiness about the feet and breathlessness, which shows conclusively that the right side of his heart has been overstrained.

Sufferers from mitral disease seldom die suddenly. Their progress downhill is gradual, and lung complications are generally very active in bringing about the fatal result. Although sudden death is not common in mitral disease, the tenure of life is very uncertain, because the patients have little power of resisting common ailments. 'Colds' are very apt, in these cases, to 'attack the lungs,' and it goes hard with them when they fall victims to influenza or similar troubles, which usually cause small inconvenience to the physically sound. I am accustomed to regard sufferers from mitral disease as ineligible for life assurance.

The wealthy sufferer from chronic heart disease should live in a place where fresh air and gentle exercise may be got without climbing hills, and he should live upon the ground floor of the house in order that the fatigue of going upstairs may be avoided.

No two cases of chronic heart disease are precisely alike, for it is evident that with the possibility of obstruction or regurgitation, or both, at each of four orifices, combined with many different conditions of the lungs, the permutations and commutations become almost infinite.

Perhaps the most incapacitating form of chronic heart disease is mitral obstruction, for not only does it produce serious engorgement of the lung, but there is a failure to supply the left ventricle with its proper quantity of blood, and the systemic arteries are starved, while the pulmonary vessels are gorged. Sufferers from this disease are always weakly and breathless.

Simple aortic regurgitation, however, although liable to terminate suddenly, often incapacitates the patient singularly little. We have two extreme cases in the wards now, one of

whom has followed the occupation of a horse cleaner and the other that of a tinplate worker, both laborious employments involving much muscular exertion, up to the time of admission. I can also call to mind the case of a young man suffering from aortic incompetence who used to come occasionally to my out-patient room, and who earned his living as an acrobat upon the stage. The left ventricle readily hypertrophies, and in this way compensates for the regurgitation, and so long as the mitral valve remains fairly competent the patients do not complain of breathlessness, there is no œdema, and they look hard and healthy in the face. These patients are, as you are aware, in danger of sudden death, and their lives are absolutely uninsurable.

CHAPTER XVII [1]

TREATMENT AND MANAGEMENT OF PULMONARY TUBERCULOSIS

BEFORE coming to details of treatment and management, and in order that any recommendations I may make to that end may appear reasonable and justifiable, it is necessary to give a comprehensive glance at certain points in the ætiology and pathology of tuberculosis, concerning which there is now a very general agreement.

The labours of many statisticians during the past half-century have shown conclusively that the death rate from phthisis bears a very general direct proportion to the degree of concentration of population. It is essentially a disease of cities, and amongst dwellers in cities and large towns it affects especially those who are engaged in sedentary indoor employments, such as clerks, shop assistants, and compositors. Where many persons are congregated under a common roof, with insufficient cubic space, phthisis is prone to be very rife, especially if the other hygienic conditions are defective. We accordingly find that the death rate from this cause in barracks and prisons formerly reached a very high figure. Now, however, that greater attention is paid to cubic space and ventilation the death rate from phthisis among soldiers and prisoners is very much lessened. It is important to bear in mind that in comparison with overcrowding, and especially overcrowding *indoors* and under a common roof, all the other ætiological factors of phthisis hold a very subordinate position. It is a disease of all climates, and its occurrence does not seem to be much influenced by latitude. In warm climates, where city populations bask *out of doors* in the sunshine, there is, generally speaking, less phthisis than in cities which have a rough and

[1] Clinical Lecture, U.C.H., 1894.

variable climate, like Vienna, whose inhabitants suffer more from phthisis than those of any other city in the world. In Vienna the population is largely devoted to indoor industries, and the inhabitants live in large tenement houses which are warmed by stoves. The variations in temperature are often sudden and extreme, and few who have been there in winter will forget the cutting severity of the wind. There are certain islands in Europe which are said to enjoy an immunity from phthisis which is more or less complete, and it is interesting to note that these islands—Iceland and the Faroe Islands—have cold and severe climates. It is also interesting to note that among European cities the northern cities—Christiania, Copenhagen, Stockholm, Königsberg—have a slightly lower death rate from phthisis than some of the cities lying in warmer latitudes. Other things being equal, it would seem that elevation above the sea-level lessens the tendency to phthisis. The plains are, as a rule, more crowded than the mountain tops, and there can be no doubt that the immunity of mountaineers is due not only to their well-developed chests and athletic lives, but also to the sparse population of the regions which they inhabit.

Having arrived at the conclusion that the main factor in the causation of tuberculosis is overcrowding, the great discovery of Koch comes, as it were, to give additional point to our conclusion.

Before Koch's discovery medical opinion was becoming more and more firm as to the infective nature of pulmonary tuberculosis, and the discovery of a *contagium vivum* in the form of the tubercle bacillus converts a theory into a fact, and gives us a solid basis both for treatment and prevention.

While one must not forget the possibility of the conveyance of tuberculosis by tuberculous meat or milk (uncooked), we shall all readily admit that the usual channel of conveyance is the air, and that tuberculosis, although a chronic trouble with a doubtful incubative period, must, for practical purposes, be classed with *air-borne* contagia, such as diphtheria, and the other infective fevers which are conveyed through the air. Although it is possible that the air may be infected by the

coughing and sneezing of tuberculous patients, still the most probable source of infection seems to be from tuberculous sputa, which, becoming dried, are raised in the form of dust.

It stands to reason that the danger of being infected in this way is much greater indoors than out of doors. Tuberculous sputa falling out of doors probably quickly succumb to the saprophytic organisms which abound everywhere. When, on the other hand, they fall indoors, they dry up, and, not being kept moist, putrefaction is delayed, they do not succumb to saprophytes, but, being persistent spore-bearing organisms, they remain ready to commence growing so soon as they fall upon a congenial soil. A volume of air which is enclosed in a room cannot, in spite of all our efforts, be renewed with anything approaching the completeness and frequency that a similar volume is renewed when not so enclosed. If we assume that a given volume of air contains a certain number of infective particles, it follows that such particles are the more likely to infect the greater the difficulty of renewing the air. Hence it follows that our danger of infection from all air-borne contagia is much greater indoors than out of doors. Given the number of infective particles in any volume of air, the danger of infection might be stated mathematically. There seems, however, good reason to believe that overcrowding in rooms and houses intensifies the danger of infection from air-borne contagia apart from mere mathematical considerations. It is evident that if our danger of infection is due to the particles of dried sputum raised in dust, the stirring the air and raising the dust, which is much greater in a crowded room than an empty one, is, in itself, a source of danger. Again, it appears probable that air which is heavy with carbonic acid and watery vapour has a greater power of carrying floating particles than air which is pure and dry, and thus we are able to adduce two physical reasons which may account for the danger of overcrowding apart from the mathematical considerations based upon a knowledge of the cubic contents of the room and the number of sources of infection. It is probable that there are other causes besides physical ones which enhance the danger of overcrowding, and it may well be that

the difficulties experienced in the proper aëration of the blood may increase the liability of the infective particles to 'take root' in the individual who inhales them. All these considerations show how important it is that sputa should be received into vessels, and be systematically burnt or chemically disinfected.

It must never be forgotten that the liability of the infective particle to 'take root' and grow varies with the individual. Some are very susceptible to phthisis, others very insusceptible. Tuberculosis is so common that one would suppose that in a great city like London there must be large numbers of tubercle bacilli floating in the air, and yet comparatively few of us become definitely infected. It must be supposed that a large number of those who get infected practically recover without suffering seriously, i.e. they throw off the infection. We know how common it is in the post-mortem room to find lungs adherent at the apex, or calcified or caseous glands, in subjects who have never been regarded as phthisical, and we may justly conclude that a fair proportion of these appearances are the records of a successful fight with the bacillus tuberculosus.

It is difficult to say precisely what it is that causes the excessive vulnerability to phthisis in certain individuals, and why the bodies of some should prove a more congenial soil for the growth of tubercle bacilli than the bodies of others. Few of us doubt that a tendency to tuberculosis is distinctly heritable, and we know that the families in which such liability exists contain a large number of individuals who are feeble, 'lax-fibred,' prone to anæmia, and very liable to catarrh. Not infrequently they have a characteristic figure, with a long, narrow chest, and it may well be that with chests of such a shape the expulsion of bacilli which may gain access to the lung is no easy matter.

Now, our main subject, the prevention and cure of pulmonary tuberculosis, may perhaps be best handled by considering what we can do to protect persons having a liability to phthisis from their probable fate.

In the first place we must seek by fresh air and exercise to 'develop the chest,' and see that the expansion and con-

traction of the lungs are both improved by exercise. Walking, running, climbing, or anything which calls for full inspirations will do this. Lagrange has pointed out that during exercise our need of deep inspirations is largely in proportion to the muscular mass which we put in action, and he therefore holds that the exercise of the legs in running is the best form of exercise to develop the chest. There are some who hold, as I hinted just now, that the immunity or comparative immunity from phthisis enjoyed by dwellers in mountainous districts is due not so much to peculiarities of climate as to the well-developed chest of the mountaineers, whose deep and forcible respiratory movements give but a small chance for the permanent lodgment of bacilli.

While endeavouring to 'develop the chest' we must take care that it is not overdone ; for if, by excessive exercise, we allow the subject to get 'blown,' we may induce emphysema, which may give us enormous girth of chest with very feeble contractile power in the lung. Subjects who are prone to phthisis should carefully guard against over-training and exercises which induce breathlessness. The exercise should be carefully graduated, and the amount slowly and cautiously increased.

Such persons should be careful to avoid, if possible, 'catching cold.' This is a difficult matter, for the proneness to catarrh probably constitutes one of the chief causes of the liability to tuberculosis. Catarrhal secretions from mucous membranes obviously form a nidus for the growth and increase of bacilli ; and there can be no doubt that a most important part in the prophylaxis of consumption in all its forms is to keep the mucous membranes clean and free from catarrh. To this end there must be strict attention to diet, rigid temperance in the use of alcohol, and the avoidance of all sources of mechanical and chemical irritation, such as dusty atmospheres and (especially if there be a liability to laryngeal catarrh) tobacco smoking in all forms, inclusive of cigarette smoking.

Directly a patient gets a chronic cough, whether it be laryngeal, pharyngeal, or bronchial, there can be no doubt that his liability to become infected with tuberculosis is appreciably

increased. The caution, therefore, to *maintain clean mucous membranes* is most important.

Persons who have a family tendency towards pulmonary tuberculosis should, if possible, live in the pure air of the country, and should select a profession or occupation which involves as little confinement indoors as possible. So long as they be free from actual lung disease, and provided they be warmly clothed and have a sufficiency of good wholesome simple food, I do not think it is necessary to ' coddle ' them too much. I am rather an advocate for sending them to tolerably bracing places, and rather prefer the east or south-east coasts of this country to the more soft and warm climates of the south and south-west. The life of a farmer or a sailor is the ideal life for these persons. I have seen, I think, some few lives saved by being taken from a close office and sent to work in the fields ; and I can recall one case, the son of a mother who was the subject of pronounced tuberculosis, in whom the enlarged and shotty glands completely disappeared during two years' schooling at Ramsgate, and who became quite strong when he emigrated to Manitoba, where the extreme severity of the Canadian winter produced no ill effect.

If circumstances render it impossible to select a completely outdoor life, we must do the best we can. Only the other day I saw the son of a small builder, who had never shown any manifestations of phthisis until he was moved from the carpenter's shop to the office, whither he had been attracted by the mistaken notion that the desk is a nobler field for energy than the bench of the handicraftsman. By my advice he left the office to enjoy the muscular exercise and the comparatively pure air of the carpenter's shop.

These delicate subjects should carefully avoid all over-crowded places, and especially in the evening, when the danger of catching cold on changing the stuffy atmosphere of the theatre or meeting-room for the chilly night air is very great indeed.

Now, when these persons develop the first signs of phthisis we commonly send them away to some health resort, and it is well that you should thoroughly understand the object with

which they are thus sent into temporary exile. The main object is that they should breathe as much pure air as possible.

Notwithstanding that cold cannot be regarded as in any sense a cause of tuberculosis, it is nevertheless a great source of discomfort and danger to those who, having become tubercular, are the subjects of that chronic pneumonia which is caused by the growth of bacilli in the lung, and which we usually speak of as phthisis. Persons with chronic lung disease are as a rule very intolerant of cold, and any undue chilling of the surface of the body is apt to produce a congestion of the lung with an extension of bronchitic and pneumonic troubles. The Registrar-General's returns show with never-failing regularity that with a fall of temperature there is invariably an increase of deaths from respiratory disease—phthisis, bronchitis, and pneumonia. The true meaning of this is, I take it, not that cold is a great cause of respiratory disease, but that it is a great cause of death in those who are the victims of respiratory disease.

Since we are accustomed to send wealthy patients suffering from incipient phthisis to the sunny south to pass the winter months, I will, without going into details as to the relative merits of this or that place, make a few general observations which may be of help to you in giving advice on this matter.

Now, remember that our object in sending the patient away is that he may breathe pure air, and that the amount of exercise which he will get in the pure air will very largely depend upon the amount of fine weather and sunshine.

But we must bear in mind that in the winter the hours for outdoor exercise extend from about 9 A.M. to 4 P.M., so that in the 24 hours there are about 7 hours in which the patient may be out and 17 hours when practically he *must* be indoors. He will breathe the air of his house for twice as many hours as he breathes the open air. Therefore it follows that the selection of the patient's house or rooms is quite as important as the selection of the locality in which the house is situated. I may say at once that in my opinion there can be no worse place for a sufferer from chronic phthisis than a monster hotel built tier upon tier, with overcrowded public rooms, in which *poitrinaires* are often herded together in a manner which is most

undesirable. I believe that the aspect of the patient's rooms is a matter of prime importance, and one which is too often neglected. The bedroom should face the south-east, in order that the early visit of the sun may tempt him from his bed, enable him to dress in the warmth, and to admit the fresh air by opening the window more frequently than otherwise would be the case.

South-east is admittedly the ideal aspect for a house, but I would say that for patients visiting the Riviera a south-east aspect is almost essential, because of the virulence of the dry, cold, north-west wind, or mistral, which is very trying for invalids. If the windows of the rooms occupied by the patient look to the south-east he gets the whole benefit of the morning sun with absolute protection from the mistral, which, when it blows with violence, is far more keen and cutting than any of our east winds here.

A word of caution must be given as to allowing real invalids to participate in the gay social life which is rather too common in health resorts which have become fashionable. When last I was in the Riviera I remember encountering a young American who was suffering from laryngeal phthisis, and who, instead of basking in the sunshine, spent the greater part of his days in the rooms at Monte Carlo, and of course was getting far more harm than good from his expensive journey to Europe. I think it is most important to lay stress on the fact that, for those who are suffering from incipient phthisis, all participation in crowded entertainments of every sort and kind should be absolutely disallowed.

I am inclined to think that in this country there are few spots better suited for the residence of patients in the early stages of phthisis than our own dry bracing sunny south downs, always provided that there be sufficient shelter from the north. A good well-built warm cottage facing south-east or south, with a wide verandah, and situated on the slope of a chalk down, is an almost ideal dwelling for the phthisical.

There is no greater practical mistake than to send patients to distant health resorts if they are unable to bear the expense of obeying one's orders comfortably. To send persons away

who are obliged to calculate the expense very narrowly ; to send them from 'home comforts' to spend the winter in some cheap 'pension,' of which overcrowding and underfeeding are the main characteristics, is to court failure.

I am not going to discuss the merits of different health resorts or dwell upon the distinguishing characteristics of the sea-shore and the mountain tops. The main object which the doctor has in view is to enable the patient to breathe pure air. Some health resorts are being killed by their own popularity, which has increased the price of building land, and has led to the building of houses in too close proximity to each other. In a health resort every house should be isolated and stand in a garden of its own. It is well to bear in mind the difference between a holiday resort and a health resort. As a rule we shall find that in proportion as a place grows in popularity with 'trippers' and holiday excursionists it becomes undesirable as a residence for real chronic invalids.

So far my advice has been to encourage persons who have a constitutional tendency to tuberculosis, or who may afford actual evidence of the disease in its early stages, to select callings and professions and to live in places which will give them a fair chance of throwing it off, and afford as few opportunities as possible for their favourable 'soil' being frequently re-seeded by the bacilli which are likely to be present in the air of houses or places to a degree bearing some proportion to the density of population.

But, it will be asked, can we do nothing to help the individual except by attending to his surroundings ? Has the discovery of the bacillus tuberculosus given us no clue to some method of destroying the microbe *in situ* ?

The answer to this question is very doubtful, and mainly for the reason that we are unable to get away from that stumbling-block of scientific therapeutics known as the *post hoc* fallacy. If, when a patient with phthisis is removed from the fœtid atmosphere of some wretched hovel, where he has been half starved and overworked, to the comparative purity of our hospital ward, where light, air, good food, and absolute repose are afforded him, we administer to him some remedy

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which has acquired a reputation for the 'cure' of consumption, and if, in a few days' time, his cough be easier, or his temperature lower, his appetite improved, or his weight increased, to which of many circumstances are we to attribute his improvement? Assuredly we have very slender grounds for attributing it to the drug. To a certainty the *patient* would attribute his improvement to the drug, for with patients, as a rule, *post hoc* is *propter hoc*, and belief in drugs is absolute.

When a drug rapidly produces some easily appreciated change in the bodily condition we manage to escape from the *post hoc* fallacy. That purgatives cause diarrhœa, that anti-pyretics lower febrile temperatures, that morphia causes sleep and contraction of the pupils, that pilocarpine and atropine influence the secretion of sweat are facts which nobody can doubt. When, however, we are told that the cessation of vomiting is due to the administration of ipecacuanha in doses so small as to elude every known test we may accept the statement as a matter of faith, but we must admit that it is absolutely incapable of experimental proof.

In the same way the cause of the improvement of chronic invalids whose condition is known to be for ever fluctuating is almost incapable of demonstration, although one is glad to be able to say that the improvement of myxœdema by the administration of thyroid extracts is apparently an exception to the rule.

It follows, therefore, that we must receive all statements as to the effect of drugs on such a disease as pulmonary tuberculosis with the utmost caution and reserve.

In discussing the treatment of phthisis by drugs I shall limit myself entirely to a consideration of those measures which have had for their object the destruction of the bacilli at the seat of their formation.

The use of inhalations of various kinds had been recommended and largely employed before the discovery of the bacillus, and since its discovery this line of treatment has had a fair trial. In many cases inhalations may have alleviated the patient's condition by somewhat lessening cough and secretions, but it cannot be claimed for any of them that they

have had any effect in arresting the progress of the disease. When we consider how very dilute must be any antiseptic vapour which is capable of being inhaled into the lungs, when we consider that there is always residual air in the lung, and when we consider still further that only a small minority of the bacilli lie on the free surface of any cavity in the lung so as to be within reach of any antiseptic vapour which may be inhaled, we cannot be surprised at the failure of this mode of treatment to arrest the disease.

Koch's efforts to kill the bacilli by the injection of 'Tuberculin'—the product afforded by the pure cultivation of the bacilli—will be long remembered as one of the most interesting episodes in the history of therapeutics. Although the treatment was not destined to fulfil the sanguine expectations of the originator, the work which led to its promulgation was of the highest scientific value. The rapidity with which the profession throughout the world came to the conclusion that the dangers of employing Tuberculin were considerable, while the advantages were doubtful, was also creditable to the scientific methods of the nineteenth century.

The administration of antiseptics by the mouth has not been productive of any decided benefit. The salts of mercury and the tar products have been used without decided effect. Among the latter one must mention creosote, which is just now going 'out of fashion,' after having been very much in vogue.

It seems to me that if any useful result is to be obtained by the employment of antiseptics in pulmonary tuberculosis it is important to choose one which is eliminated by the lung —something which is absorbed from the stomach, mingles with the blood streams, and is eliminated by the lungs.

The bodies containing the active principle 'allyl,' such as garlic and onions, seem to fulfil these indications, and seem worthy of a trial. The most potent of these bodies appears to be garlic, for it is well known that the smell which garlic imparts to the breath is most persistent, and is produced by very small quantities. The allyl compounds such as allylic alcohol and oil of mustard were shown by Koch to be strongly

antiseptic, and to have a remarkable power of inhibiting the growth of the bacillus anthracis. In this respect they rivalled the salts of mercury. If it be possible to hinder the growth of the bacillus tuberculosus in the lung, it seems at least reasonable to try the effect of such a body as garlic. But, it will be said, if garlic is capable of hindering the growth of bacilli tuberculosi, we ought to find that phthisis is less prevalent among garlic-eating populations than elsewhere. Facts are wanting to decide this point either one way or the other, but even should it be shown now or hereafter that there is no correspondence between the habit of eating garlic and a small death rate from phthisis it would not, to my mind, be a very strong argument against giving garlic a trial, for one could only suppose that such a method of treatment would be useful as a help when other circumstances were favourable. It could not reasonably be expected to be of any use in the face of overcrowding, starvation, dirt, and squalor such as are common enough in many of the cities of southern Europe.

Now, I think I may safely say that I have found garlic of distinct service in cases of dilated bronchi with fœtid expectoration. In two such cases in which the expectoration was purulent and horribly fœtid we administered garlic in the food until the odour of garlic was permanently present in the breath. In both cases the disgusting fœtor of the expectoration was replaced by an odour not fœtid, but pungent, and reminding one in some degree of the odour of syringa. The change in the odour of the sputa was the same in both cases. The fœtor disappeared, and was replaced by an odour which was not that of garlic, but distinctly *sui generis.*

Mr. Ionides, who was my house physician when one of these cases was in hospital, treated the sputa with ether, and manufactured in this way a very pungent 'scent,' in which the syringa quality was distinctly present. In these cases the expectoration lessened, and the condition of the patient was distinctly improved.

Dr. Sidney Martin tells me that he also has given garlic to patients suffering from dilated bronchi and fœtid expectoration, and with distinctly good results.

Then we have had two cases in the wards of boys who were apparently suffering from basic phthisis, and who made no improvement until they were placed upon garlic, when a rapid and complete recovery took place.

In private practice I can recall the case of a gentleman suffering from fibroid phthisis who improved most remarkably and gained weight very rapidly while he was taking garlic, which he did most persistently.

Another case was that of a girl with aphonia, who had very characteristic pyriform swelling of the aryteno-epiglottic folds, so that neither I nor Dr. Adams, of Buckhurst Hill, who brought the patient to me, had the slightest hesitation in making a diagnosis of tubercular laryngitis, and giving the most guarded prognosis. The patient among other measures was ordered to eat garlic. When next she came to see me, a month or so later, the laryngeal condition had completely subsided. The result was so remarkable that one is inclined to doubt the accuracy of one's diagnosis, but nevertheless the result justifies one in trying the remedy again in similar cases.

I have usually given the garlic in the food. A ' clove of garlic ' has been chopped up and mixed with the beef tea, and only very few patients have been unable to take it in this way. The evidence that the patient is under the influence of garlic is the persistent smell of it in the breath, a smell which is equally present when the garlic has been administered in the form of an extract enclosed in gelatine capsules.

Garlic, I need not say, is no new remedy. It formerly held a place in the London Pharmacopeia, but the extant British Pharmacopeia does not recognise it. I see that Dr. Brunton states in his work on pharmacology that garlic is still in the Pharmacopeia of the United States. It is mentioned by Pereira and by most of the previous writers on Materia Medica. It was used by Hippocrates. Among the properties ascribed to garlic by old writers is that of a stimulating expectorant, so that its employment in lung diseases is no new idea. Its acrid taste and the disagreeable smell which it gives to the breath have naturally militated against its becoming a

very popular remedy. I have found, however, no difficulty in persuading patients to take it. Administered with food, it has never caused any serious gastric disturbance, the only trouble in its administration being a natural repugnance to the flavour and odour on the part of some patients.

The essential oil of garlic is said to contain a sulphide of allyl, and in this connection I would remind you that other sulphides, such as sulphuretted hydrogen, sulphur waters, the sulphides of calcium and potassium, have enjoyed a certain reputation in tuberculosis and 'scrofulous' diseases.

Among other remedies which I think I have found serviceable in phthisis are the so-called 'alteratives,' such as arsenic or potassium iodide in small doses and the sulphides above alluded to.

Counter-irritation by means of iodine or stimulating liniments, well rubbed in until the skin of the chest is thoroughly reddened, often have the effect of lessening the cough and expectoration.

Garlic is not only given off by the breath, but by the skin also, and I have been for some years in the habit of administering it to those cases, especially common in children, in which a cadaveric odour of the skin develops after attacks of acute specific disease. In such cases warm baths and soap are of no use, but I have found that if garlic be administered until the skin reeks of it the cadaveric odour is got rid of, and is no longer noticeable when the smell of the garlic disappears.

I have thus brought to your notice a body which, on theoretical grounds, seems worthy of a trial, and which from practical experience I feel inclined to continue to administer to sufferers from phthisis in its early stages.

The following very interesting letter and history was most courteously sent to me by Dr. Skelton, of Bristol, and seems to me to be worthy of reproduction without abridgement :

May 24, 1894.

'Dear Sir,—Seeing your paper on the treatment of pulmonary tuberculosis in the "Clinical Journal" of January 31,

in which you advocated the use of garlic, I happened to be able to give it an immediate trial. Mr. P., my patient, was at that time expectorating very offensive sputa to a large amount after a slight attack of hæmorrhage. He has been under my care for some years, having come here from Derby (he is an employé of the Midland Railway Co. in a responsible position in the head office here) for the benefit of his health, as he had been subject to hæmoptysis. He has had several returns of the hæmorrhage, sometimes to a very profuse extent. The blood comes from what apparently is a large cavity or dilated bronchus about the middle of the left lung.

'Between the attacks, which are generally annual, he gets fairly strong again, though I think he is gradually losing flesh. He is a most intelligent man, and of a cheerful disposition, making the best of everything. When he started taking the garlic I told him to keep an account of his condition from day to day. This I enclose, and it, I think, tells its own story very graphically.

<div align="right">'I remain, yours faithfully,</div>

<div align="right">'H. Skelton.'</div>

Diary written by the patient himself.

January 3rd, 1894.—Slight attack of hæmorrhage.

6th.—Discharge cleared.

7th.—Last injection of ergotine.

8th to 20th.—Increase in discharge to nearly half a pint per day, very offensive to taste and smell. Inhaling turpentine and creosote.

21st.—Commenced taking creosote, mixture and pills.

22nd to 31st.—Gradual decrease in discharge to about a quarter of a pint per day, but still offensive. Slowly increasing in strength.

February 1st, 1894.—Commenced eating garlic. One piece at tea time (5 P.M.).

2nd.—Garlic three times. Twice raw and once cooked. Marked decrease in discharge.

3rd.—Garlic three times. Further decrease in discharge. Considerable increase in strength

4th.—Garlic twice.   Further decrease in discharge.

5th.—Garlic three times.   Discharge reduced to *my* normal quantity, i.e. only a few expectorations during the day.

6th and 7th.—Garlic three times daily.   Result as above.

8th to 10th.—Garlic twice daily.   Result as above.

11th to 17th.—Garlic twice daily.   Gaining strength.

18th to 24th.—Garlic once daily.   Gaining strength.

24th to March 3rd.—Garlic once daily.   Discharge so slight that I have not required the use of an 'invalid's mug.' This I have not been able to dispense with during all my illness, viz. five and a half years.   Strength still increasing.

March 3rd to 10th.—Influenza cold.   Continued with the garlic.   No increase in discharge.

10th to 17th.—Still suffering from cold, damp weather. Continued taking garlic ; increase in discharge towards end of week.

17th to 31st.—Cold better and discharge reduced.

April 1st to 30th.—Continued the garlic, small piece each day.   Discharge slight all the month.

Garlic.—*February 1st*, 1894.—Purchased 1 oz., cost 1½*d.* This I ate in five days, having (about) one section of a bulb at each meal, viz. breakfast, dinner, and tea.

Once I tried it cooked, but found it then had little flavour ; all the other I took raw.   Relished it best with bread and mild cheese, or bread and butter.

After eating the first piece the discharge from the lung, from being of a pale green colour and very offensive, became almost white in colour and without any objectionable taste or smell.   On the second day it tasted and gave an odour of garlic.

Urine gave a strong odour of garlic.

As soon as I commenced eating it my appetite increased, digestion improved considerably, and I noticed a marked daily increase in strength.   Had much better sleep, and perspiration at night entirely ceased.

CHAPTER XVIII

A CASE OF TUMOUR OF THE LUNG [1]

A MAN aged twenty was admitted to University College Hospital on December 12, 1894. He stated that on October 8 of the same year he noticed, on getting up to go to his work in the morning, that he was very short of breath and felt cold all over. He had been quite well previously, but the symptoms were so severe that he took to his bed at once and sent for medical advice. He remained in bed, till the end of the month, when, feeling better, he got up and went about for a fortnight, but did not resume his occupation. One morning about the middle of November, while out walking, he was suddenly seized with a severe aching pain in the middle of his back, which caused him to double up. He immediately returned home and took to his bed, stopping there till he entered the hospital. Since the middle of October he had suffered from pain in the left side, which was worse on coughing. For a week—a month before the date of his admission—he expectorated sputum stained with blood. He had been treated during his illness with poultices, blisters, &c. He had had influenza three years before, but no other illnesses. He said that he had always been delicate. There was nothing noteworthy in the family history. On his admission it was found that he had a slight cough, but no expectoration; he had some dyspnœa on exertion; there was no cyanosis; the respirations were 40 per minute. On examining the chest it was noticed to be poorly covered, long, and narrow, and that the intercostal spaces were better marked on the right side than on the left, which side hardly moved at all on respiration. Below the third rib on the left side

[1] Clinical Lecture, U.C.H., 1895.

there were absolute dulness on percussion and loss of vocal fremitus, of breath sounds and of voice sounds. In the second left intercostal space there was distant bronchial breathing with ægophony, the percussion note being impaired very much. In the first left intercostal space the percussion note and breath sounds were normal. Behind there were on the left side, below the spine of the scapula, very diminished vocal fremitus and dulness on percussion, with faint bronchial breathing and very weak voice sounds. In the left supra-spinous fossa the percussion note was impaired and the breath sounds very weak, but bronchial in character. The physical signs over the right side of the chest were normal, except that the heart could be seen beating over the second, third, and fourth spaces on the right side internally to the right nipple line, with the heart's apex beat in the fourth space midway between the right nipple line and the right border of the sternum. The impulse was somewhat heaving in character. On listening at the apex a well marked systolic murmur was heard, which was only conducted inwards as far as the middle of the sternum. There was nothing abnormal in the urine ; appetite fairly good, bowels regular. There was no œdema anywhere, and there were no dilated veins and no enlarged glands. As the patient had all the signs of an extensive effusion into the left pleural cavity, an attempt was made on the day after his admission to aspirate the chest in the sixth space in the mid-axillary line. An ordinary bottle aspirator was used with a moderate-sized needle, but as nothing but a little blood was drawn off the needle was again inserted on the same day, this time just internally to the angle of the left scapula, with the same result. The blood drawn off contained nothing but corpuscles. The temperature was 100·2° F. on his admission, and varied between that point and 98° for the next few days, and after that kept below 100° for some days. Soon after admission he was noticed to have a slight prominence of the chest wall in the left mammary region, which gradually became more marked, and on comparing a tracing of the chest wall taken on January 12, 1895, with one taken a month previously, a con-

siderable difference could be noticed in the size of the left side of the chest, especially in the anterior part. His general condition altered very little during this time ; the temperature chart from December 23 to 30 showed an evening rise to nearly 101°, with a morning fall to normal, but from the latter date till his death his temperature did not rise above 100°, and after January 1 or 2 it never rose above normal. His pulse during the whole time he was in the hospital was rapid, on an average 120 to the minute, with the respiration 30 per minute. On January 3 an exploring needle was again introduced (this time into the left side of the chest in front), but with a negative result. On the 15th some œdema of the chest wall in the left mammary region was noticed, which gradually increased. About this time, also, some dilated veins were noticed in the left axilla, and both external jugular veins were found to be distended ; there was also some distention of the veins of the forehead. From the 15th till the time of his death the patient passed very small quantities of urine, which was of high specific gravity and contained a trace of albumen on two occasions, but at other times was normal. On the 29th some œdema of the lower part of the back on the left side was noticed, that on the front of the chest being more marked ; there was also some on the left side of the face. He now could only lie on the left side, being unable to breathe in any other position. On February 2 a small, rounded, almost fluctuating swelling was found just over the anterior surface of the head of the right humerus ; it was painless, and the skin over it showed no signs of inflammation. On puncturing the tumour a little blood escaped, which on microscopical examination was found to contain cells of a sarcomatous nature. The dulness on percussion, loss of vocal fremitus, &c. now extended over to the right as far as a line drawn downwards from the middle of the right clavicle to the right nipple, and then to the costal margin, the heart's apex beat being just to the left of the right nipple in the fourth space. The signs were limited to the left side behind. From this time the patient became rather rapidly worse, the

œdema increasing and the dyspnœa being more marked ; he, however, apparently did not suffer any pain, and slept well. The growth at the head of the humerus increased slowly in size, and another small tumour was found on February 10 just below the left scapula attached to a rib. He became delirious on the evening of the 18th, and died the next morning.

At the post-mortem examination the diaphragm on the left side was found to be very much depressed by, and the whole of the left side of the thoracic cavity filled with, an extremely soft growth ; it was dark brown in colour and enclosed in a capsule formed apparently of thickened pleura, which was firmly attached to the chest wall every-where. At the upper and anterior part of the tumour, and continuous with it, was found a layer of collapsed lung about half an inch thick, seven inches long, and four inches wide at its upper part and narrower below. The heart and pericardium were displaced to the right, as was also the right lung. The two former were healthy, but the right lung contained a small growth about the size of a marble on its posterior surface. The liver contained a few small growths about the size of peas scattered through its substance. All the other organs were healthy. There were no enlarged glands, except one in the anterior mediastinum. Microscopic examination of the tumour showed it to be a round-celled sarcoma, which was rapidly degenerating.

Remarks.—The great interest of this case consists in the impossibility of diagnosis in the early stages. The history was that of an acute onset, and the physical signs on admission were those of effusion into the left pleura. Even after the failure of aspiration, which necessarily aroused one's suspicion as to the true cause of the trouble, it was hard to believe that such failure was not due to some mechanical condition—such as profuse and œdematous adhesions—which prevented the flow of the fluid from the pleura. The physical signs were uniform in character, and there were no patches of dulness and resonance or of diminished and increased vocal fremitus, such as are common in growths in the lung. The

necropsy, which revealed a soft pultaceous tumour uniformly distending the visceral pleura, fully accounted for the physical signs during life. There was a history of very slight hæmoptysis, but this was never repeated, and merely gave rise to the suspicion that there was tubercle as well as fluid. When, towards the end, there appeared marked œdema of the chest and distention of the veins on the left side of the face and chest, *and this without any rise of temperature*, the diagnosis became almost certain, and with the appearance of the soft tumours on the neck of the humerus and one of the ribs one felt quite sure as to what would be found postmortem. It is interesting to note that the weight of the patient, which was 6 st. 11 lb. on admission, fell to 6 st. $7\frac{1}{2}$ lb. on December 22, 1894, and rose to 7 st. $2\frac{1}{4}$ lb. on January 14, 1895. The last weight recorded (on January 22) was 7 st.

CHAPTER XIX

THE SULPHUR WATERS OF THE PYRENEES [1]

NOTHING is more gratifying in the treatment of disease than the occasional success which attends the employment of so-called alterative medicines, among which we may place mercury, iodine, arsenic, phosphorus, and the sulphides. It is common to see effects produced by these preparations which are at once tangible and decided, but unfortunately it is common also to see them utterly fail to produce the effect desired.

Anyone who has employed the sulphides in the treatment of disease must have been alternately pleased and disappointed ; for, although the effect produced is often exactly that which Dr. Ringer describes in his Handbook of Therapeutics, equally often, if not more often, the result appears to be absolutely negative. One reason for this is undoubtedly to be found in a faulty selection of cases ; and it may reasonably be hoped that an increasing knowledge may cause a diminution in the number of patients who, having been treated with sulphides, have failed to derive the expected benefit. Another reason also is to be found in the great instability of the sulphides themselves, which immediately oxidise on exposure to air ; and it may safely be said that a very large proportion of the patients for whom the sulphides are prescribed get scarcely a single dose of the medicine which it is desired to give them, but receive in lieu of it a quantity of harmless or inactive sulphate.

When prescribing sulphides it has been my invariable custom to advise the patient to get very small quantities made up at a time, and to warn him that unless the medicine has the familiar odour of rotten eggs it can do him no good. Better than giving a solution of the sulphides is to allow the patient

[1] Communicated to *Lancet*, 1879.

to have a small and well stoppered bottle filled with potassium or calcium sulphide, whence he may take his dose, roughly estimated on the point of a penknife, as required. Good service has undoubtedly been done by the introduction of the sulphides in perles, and I have reason to believe that the sulphide remains unoxidised in the perles for indefinite periods. I have lately, however, seen a sample of so-called 'perles of calcium sulphide' which on examination were found to contain no sulphide at all. The best way of all, no doubt, to prescribe sulphides is to send the patient, if possible, to a so-called sulphur spring, where he may drink the water, impregnated with hydrogen sulphide and holding other sulphides in solution, before oxidation has had time to occur. Then one may be sure that the medicine prescribed is actually being taken.

Having during the autumn of last year paid a visit to the sulphur springs of the Pyrenees, I have thought that possibly the information thus acquired might prove serviceable to others besides myself.

There are many sulphur springs in the Pyrenees, but the best known are those situated in the western half, in the departments of the Basses Pyrenées, Hautes Pyrenées, and Haute Garonne. Going from west to east, we have Eaux Chaudes, Eaux Bonnes, Cauterets, St. Sauveur, Barèges, Bagnères de Luchon, and Bagnères de Bigorre. Excepting at the last-named place, the prevailing waters are those containing sulphuretted hydrogen and other sulphides, and there is a strong family likeness between them all. These waters are excessively weak, and contain not more than from 0·5 to 0·2 grm. of dissolved matter in each litre of water, or from about $4\frac{1}{2}$ to 2 grains in the pint—thus offering a striking contrast to the waters of Harrogate and Aix-la-Chapelle, which contain respectively 137 and 37 grains in each pint. The amount of free hydrogen sulphide in the Pyreneean waters is exceedingly small, the salt which gives to them this sulphurous quality being sodium sulphide, of which there is from 2 to 5 centigrammes in each litre, or from $\frac{1}{5}$ to $\frac{1}{2}$ grain in each pint. The bulk of their small quantity of solid ingredients is composed of silicates, chlorides, and organic matters. The following schedule

will serve to show at a glance what are the chief ingredients of these springs :

GRAMMES IN A LITRE. (Parts in 1,000.)

—	Eaux Chaudes (Le Clot)	Eaux Bonnes	Cauterets (César)	St. Sauveur	Barèges (L'Entrée)	Luchon (La Reine)	Bigorre (Labassère)
Sodium sulphide .	0·0080 (?)	0·0214	0·0226	0·0218	0·0344	0·0508	0·0464
Sodium chloride .	0·0900 (?)	0·2640	0·0178	0·0695	0·0544	0·0624	0·2058
Silicates . . .	0·0041 (?)	0·0500	0·1114	0·0867	0·1087	0·0614	0·0555
Sodium sulphate .	0·0234	0·0277	0·0080	0·0400	0·0169	0·0312	—
Calcium sulphate .	0·0791	0·1644	—	—	—	0·0312	—
Organic matter .	(?)	0·0480	0·0450	0·0320	0·0510	(?)	0·1450
Total solids . .	(?)	0·5760	0·2019	0·2500	0·2654	0·2511	0·4813
Temperature (C.) .	36·25°	32·00°	48·00°	34·60°	40·00°	51·00°	15·00°

A typical spring has been taken as a representative of each of the localities, but these form only a very small proportion of the total number. Thus at Barèges there are upwards of a dozen springs ; at Luchon there are nearly thirty ; at Eaux Chaudes, six ; at Eaux Bonnes, six ; at Cauterets, thirteen ; and at St. Sauveur certainly two or three. There are differences in the compositions of these several waters, and the local practitioners profess to be able to select the cases which are best treated at each of them, and make the most of the milligrammes and fine shades of temperature which constitute the differences.

At Bigorre there are a great many different springs, the majority of which belong to the group of saline waters, the chief saline ingredient, however, being sulphate of lime. These should properly be classed among indifferent waters. There are a few sulphurous springs also, the chief of which is that called Labassère.

It will be noticed that the analyses make mention of organic matter found in the springs, which is generally present in considerable quantities. This organic matter collects in the reservoirs and conduits through which the water is brought on its way to the baths and drinking fountains, and seems to be spoken of by various authors under the names of ' Barégine,' ' Glairine,' or ' Sulfurine.' These names would lead the unwary to suppose that this organic substance is the active principle of the water, and indeed we find it so spoken of in more than one monograph, and notably in the advertisement of the

Barèges waters, which says :—' Cette barégine donne aux eaux de Barèges une onctuosité particulière, qui est un des éléments de leur efficacité thérapeutique.' The director of the bathing establishment at Barèges very kindly ordered one of the attendants to collect for me a sample of this so-called barégine, and in a very short time after the order was given I was furnished with as much as could be put in a half-pint bottle. To the naked eye it looked slimy, and was of a grey greenish tint, with here and there portions which were nearly black. It felt like mucus, and under the microscope was found to be composed of the filaments and reproductive organs of various algæ. These algæ must be regarded as purely accidental, and are merely due to the fact that the sulphurous waters form a suitable nidus for their propagation. The growth of algæ in the water of hot sulphur springs has been noticed elsewhere than in the Pyrenees. In the fourteenth volume of the ' Journal of the Linnean Society' will be found an interesting paper by Mr. H. N. Moseley, F.R.S., who accompanied the *Challenger* expedition in the capacity of naturalist, ' On Fresh Water Algæ obtained at the Boiling Spring at Furnas, St. Michael's, Azores, and their neighbourhood.' The whole group of the Azores is pronounced to be decidedly volcanic in character, and in the largest island of the group (St. Michael's) hot springs abound in many parts, but notably at the western extremity of the island in the valley of Furnas, where are situated the boiling fountains known as the Caldeiras, which have enjoyed for a considerable period no little reputation for their curative action upon palsy, rheumatism, scrofula, and similar maladies. Mr. Moseley describes how he cut away portions of a vegetable deposit from the sides of some of the boiling springs in situations where the heat of the water was so intense that great care was necessary not to scald the hands while collecting it. These hot waters contained an abundance of sulphuretted hydrogen. Mr. Moseley estimates the temperature of the water in which the algæ were found to have been from 149° F. to 158° F. This estimate was made by dipping the finger in the water, since unfortunately he had no thermometer with him at the time of his visit. Near the margin of the lake of

Furnas are several spots where hot gas, charged with sulphuretted hydrogen, is discharged through the water, and, curiously enough, the algæ were found to grow more luxuriantly in the immediate neighbourhood of these discharges.

The alga peculiar to the Pyreneean sulphur springs has been described by Camille Montagne in the ninth volume (Botanique) of the 'Annales des Sciences Naturelles,' and has been named by him Leptomitus sulphurarius. It is said to grow especially in waters having a temperature between 50° F. and 100° F. It cannot exist without exposure to air, and sulphur seems also to be indispensable to its proper development, as well as an azotised substance dissolved in the water. An alga of a very similar description has been found growing in the hot water of Carlsbad, but it has been decided not to be precisely similar to that which constitutes barégine. Not only do vegetables flourish in these sulphurous waters, but animals as well, and many species of anguillula have been discovered by various observers in the waters of Barèges and other bathing resorts of the Pyrenees.

It is much to be regretted that a nonsensical and misleading name has been given to the aggregate growths which occur in these sulphur waters. There is no evidence whatever that they have any part in the therapeutic value of the waters, and it is very undesirable that a word should be retained which, in our present state of knowledge, not only savours of quackery, but is undoubtedly used for quackish purposes. Another regrettable fact is the part which limited companies are beginning to play in the management of mineral springs. It has been discovered that a spring of water, if it be sufficiently puffed, is a surer and more valuable source of income than a gold mine, and the statements put forward in advertisements are very often absolutely unwarrantable. It is desirable that the profession on the Continent, and especially in France, should unite for the purpose of restraining the statements of those who have merely a money interest in mineral springs, for it will become exceedingly difficult to prescribe this or that water if, by so doing, one should seem to countenance the unwarrantable assertions which are made

concerning them. It would be satisfactory if such rash assertions were made only by the trading companies, but this, unfortunately, is not the case; for many of the medical monographs appear to show a very undue amount of local bias, and to lend a too ready credence to startling therapeutic theories. It would be a great boon if the examination of the mineral springs of Europe could be made by an international commission which should be entirely without interest in the popularity or earnings of any popular spring.

Looking once more to the nature of the waters, we see that there is only one ingredient—the sodium sulphide—which can fairly be credited with any therapeutic value, and if catalogued in the order of their sulphide strength they would read thus—Luchon, Labassère (Bigorre), Barèges, Cauterets, St. Sauveur, Eaux Bonnes, Eaux Chaudes. The amount of sulphides contained even in the strongest of them is but small —so small as to call forth the remark from Braun ('Curative Effects of Baths and Waters,' p. 419, Weber's translation) that 'only a few of them contain an amount which admits the possibility of the effect of sulphuretted hydrogen.' The writer can see no reason for this opinion; for there is no more difficulty in believing in the therapeutic effect to be derived from a tenth or a quarter of a grain of sodium sulphide than in that which is known to result from the thirtieth of a grain of phosphorus or a twelfth of arsenious acid. The chief use of sulphur water, according to Braun, is for treating 'stasis of the portal system,' and conditions which are secondary thereto, and he seems to have no knowledge of their very great utility in combating the various manifestations of a scrofulous diathesis. It is equally remarkable that Birch-Hirschfeld, in his treatise on Scrofulosis in Ziemssen's Encyclopædia, makes no mention of the sulphides in the sections devoted to treatment.

The power which the sulphides possess of stimulating nutrition in scrofulous subjects whose nutrition is sluggish must have been often observed by anyone who has made a patient trial of their therapeutic value. At the Royal Infirmary for Children I have repeatedly ordered them for

patients with enlarged glands ; and although I have frequently failed to obtain any result I have at least as frequently seen, as an immediate consequence of their administration, activity replace torpor and absorption or suppuration be quickly produced, these effects being usually accompanied by cutaneous hyperæmia, and sometimes by a slight tingling of the skin.

Faith in mineral waters is far more easily produced when the alleged effect of the water is obviously due to the chief ingredient, and when the administration of the chief ingredient *per se* produces results identical with those which arise from taking a course of the waters. The fact, therefore, that the sulphur waters of the Pyrenees are found chiefly useful n those cases for which sulphides are administered at home is greatly in favour of the genuineness of the therapeutic action of the waters.

The majority of the monographs which deal with this group of waters claim for them that they may be used as a panacea against every known ailment ; and the reader who has any yearnings after an approach to scientific therapeusis soon throws them away in disgust, having only learnt one important fact from them—viz. that patients visiting the Pyrenees are to be warned against authors as persons of a dangerously sanguine temperament. There are, of course, exceptions, and amongst these may be mentioned the very excellent and temperate monograph on the Waters of Barèges by Dr. Armieux, and the scarcely less able treatise on Eaux Bonnes by Dr. Prosper de Pietra Santa. Andrieu, who published an essay on the Eaux Bonnes some thirty years ago, says : ‘The stimulating properties of the Eaux Bonnes make them of value in cases where it is necessary to rouse the torpid vitality of organs, and to contend with a chronic condition by impressing on the sluggish and obscure disease a quicker, freer, and more sharply marked course.’ The idea seems to have been held, at one time, that the best way to cure a chronic malady was to convert it into an acute one, and this power was claimed by Andrieu for the waters of Eaux Bonnes. Pietra Santa, in summing up

the indications and counter-indications for employing these
waters, makes use of the very words of Andrieu, and says :
'Chronicity, asthenia, catarrhal conditions, the scrofulous
diathesis, a lymphatic habit, laxity and habitual passive
congestion of the tissues, an obtuse sensibility with slight
tendency to irritability, the "herpetic diathesis," rheumatic
and hæmorrhoidal affections, . . . these are the conditions
in which the administration of the waters of Eaux Bonnes
is especially indicated. . . . A state of inflammation, an
exaggerated nervous erythism, excessive pain, violent spasm,
copious discharges, pyrexia, pronounced plethora, colliquative
sweats—these are the main counter-indications, absolute or
relative, to the administration of these waters.'

The waters of Eaux Bonnes have a great repute in the
treatment of follicular pharyngitis, and in those forms of
phthisis which are the result of the scrofulous diathesis.

Armieux, whose position as head of the Military Hospital
at Barèges allows him to take an entirely unbiased view of
the utility of the sulphur waters, is quite in accord with Pietra
Santa. He says : 'One can say in general that the waters of
Barèges are most favourable for lax-fibred and lymphatic
patients suffering from sluggish affections, for the lesions
resulting from scrofula, and for traumatic lesions occurring in
subjects without rallying power or reaction in whom the
fibrous tissues, the lymphatic glands, and the articulations
readily swell, but are slow to subside.'

In the Military Hospital at Barèges great success attends
the treatment of the various scrofulous affections of bones and
joints, and during the late war it was found that sluggish
wounds occurring in scrofulous subjects soon became active
when removed to Barèges for treatment.

Hitherto the probable effect of drinking sulphur waters has
alone been considered, and we have found that the results
obtained by drinking the natural water are distinctly similar
to those which are brought about by taking artificially pre-
pared solutions, so that there is no necessity, in accounting
for the action of the former, to make any reference to an
inscrutable *divinum quid*. It must be remembered, however,

that a visitor to the Pyrenees comes under other influences besides those of the sulphur in the water ; and chief among these are the temperature of the water, which allows him to combine hot-bathing with water-drinking ; and the height above the sea-level of the place where he sojourns while undergoing the cure.

That warm bathing is a valuable auxiliary in the treatment of many chronic ailments nobody will deny, and especially in those conditions occurring in scrofulous patients where the nutritive processes are sluggish. It has been said by Braun that 'heat favours function,' and there can be little doubt that a hot bath is admirably calculated to assist the action of the sulphide, which is given with a view of stimulating function. There is no reason to suppose that the sulphide contained in the water of the bath exercises any specific effect, although possibly the vapour of hydrogen sulphide which is inhaled during the act of bathing may excite some beneficial influence. For bathing purposes the chief merit of the Pyreneean waters is their heat, the contained sulphide being wholly inactive. The fact that the waters when drunk are taken hot may help to produce the desired effect.

Of the various methods of bathing and applying the waters it is not necessary to speak, for they do not materially differ from those which are in use at other baths. There are piscines, swimming baths, private baths, foot baths, ascending and descending douches, inhalation rooms, and pulverisation rooms. With regard to the pulverisation of sulphur waters, it may be mentioned that Pietra Santa proved conclusively that the act of finely dividing the water brought about at once the oxidation of the sulphide into sulphate.

All the Pyreneean bathing resorts are at a considerable elevation above the sea-level, and it has been supposed that this fact has more influence on the welfare of the visitors than the sulphides or other ingredients of the water. Mountain air, like hot bathing and the sulphides, is a powerful stimulant to function, so that all three of these factors in the

treatment of a patient in the Pyrenees work in the same
direction. As regards height above the sea-level, Barèges
stands first, with an elevation of upwards of 4,000 feet; then
follows Cauterets, with 3,000 feet; next come in order
St. Sauveur, Eaux Bonnes, and Eaux Chaudes, at a height of
2,500 feet, 2,400 feet, and 2,200 feet respectively; then Luchon,
with an elevation of 2,000 feet; and lastly Bigorre, which is
only 1,800 feet above the sea.

As a matter of course, these Pyreneean health resorts are
used by a great variety of invalids other than those of the
scrofulous class. Whoever may expect to find benefit from
a sojourn in a mountain climate or from a systematic course
of warm baths will find it here as well as elsewhere.
Patients suffering from rheumatism or gout, or from the
effects of paralysis, come to the Pyrenees in great numbers,
as well as a vast crowd of semi-invalids who have lived too
freely or worked too hard. To these must be added many
more who make some real or fancied ailment the excuse for
spending a month or six weeks in localities which are at
once pleasant and fashionable. I have merely tried to in-
dicate that class of patients which, more than any other,
may expect to find relief from drinking Pyreneean sulphur
waters.

Since it is very necessary to know something more about
a health resort than its elevation above the sea-level and the
analysis of its water, I feel that a few words of description of
the various places named will probably prove acceptable.
There are certain points which they all have in common.
Natural beauties abound on every side, and in the Pyrenees,
as in the Alps, the wonders of nature form pleasing objects
for excursions and occupation for the mind. The expense of
getting to the Pyrenees is rather greater than that of getting
to Switzerland, and the time occupied in the journey is
rather longer. The expense of living is rather greater than
in Switzerland, and the cost of locomotion is considerably
more. All the bathing resorts are united by a magnificent
road, the Route Thermale, as it is called; but there are not
the same facilities for the transport of persons and baggage as

are found in Switzerland. The public diligences are few in number and second-rate, and the only really comfortable mode of locomotion is by private carriages, for which the tariff is decidedly high. It is always advisable to ask what price is to be paid for a room before engaging one in an hotel, and the only way to guard against imposture is always to ask beforehand what price is to be paid for everything. The following passage, taken from Dr. Armieux's monograph, gives an excellent notion of some of the worse qualities of the inhabitants. My own short experience fully accords with that of the writer : 'Les Barégeois sont intelligents, leur imagination est vive, ils sont très près de leurs intérêts, et ne se font aucun scrupule quand il s'agit d'obtenir le plus d'argent possible des services qu'ils rendent aux étrangers. Dans un pays où chacun est propriétaire il n'y a pas de pauvres ; cependant on voit beaucoup de mendiants, et les petits enfants sont instruits de bonne heure à tendre la main aux passants, avec cette particularité caractéristique que, quand on leur refuse, ils vous insultent, et que si l'on se fâche ils vous jettent des pierres. L'argent gagné pendant la saison thermale n'est pas employé à améliorer le bien-être de la famille et à amasser des réserves pour la morte saison. De déplorables habitudes se sont introduites dans ces paisibles vallées, et l'intempérance des deux sexes est un objet d'étonnement et de dégoût pour le voyageur.'

As regards accessibility, Eaux Bonnes and Eaux Chaudes are each about twenty miles from Pau, which is the nearest railway station. Cauterets, St. Sauveur, and Barèges are at distances varying from five to ten miles from the railway terminus at Pierrefitte, while Luchon and Bigorre have each a railway station in the town.

Eaux Chaudes is a quiet place, situated in a very narrow gorge, and has all the disadvantages of places so situated—viz. great cold before the sun's rays reach the bottom of the gorge, great heat during the middle of the day, and incessant draughts. It is not a place for persons with weak lungs.

Eaux Bonnes is fashionable and lively, and is built into a

rocky *cul de sac* with sides which are absolutely perpendicular. Many of the hotels have rooms literally hewn out of the rock. The centre is occupied by a garden, where the band plays. The situation of the town is very picturesque, and the huge rocky summit of the Pic de Ger overhanging it forms a most striking object. Nearly every house in the place is an hotel or a lodging-house, and in the height of the season the population is almost dangerously dense.

Cauterets is a large, fashionable place, attracting thousands of visitors during the season. It is surrounded by hills on every side, but it is less closely hemmed in than Eaux Bonnes. Here, as at Eaux Bonnes, every kind of amusement is provided.

St. Sauveur is a quiet place, built on one side only of a tolerably wide valley. From it the Cirque of Gavarnie, one of the most interesting natural curiosities of the Pyrenees, is easily reached.

Barèges consists of one long, straggling, old-fashioned street, built on the side of a hill facing the west. There are several hotels, but few of them are really first-rate. Barèges is more frequented by genuine invalids, probably, than any other point in the Pyrenees. Owing to its elevated situation the climate is rough at times, and but little suited for persons with delicate lungs. There is a famous military hospital here, and the Pic du Midi and some of the grandest scenery in the Pyrenees are within easy reach.

Luchon is decidedly the most fashionable of all the Pyreneean health resorts, and for beauty of situation it certainly eclipses all the others. It lies in a wide, well watered, and verdant valley, facing the north, and being in close proximity to some of the highest mountain peaks it is as much frequented by tourists as invalids. The hotels are numerous, and are mostly first-rate, and the visitors are largely composed of French and English fashionables. Few pleasanter places than Luchon for spending a summer holiday could be found.

Bigorre is situated on the Adour just where the mountain slopes subside into the plains of Tarbes. Unlike the places I have previously mentioned, Bigorre is not a mere bathing

resort, or *ville d'été*, but is the capital of an arrondissement, and a town of some local commercial importance. Its permanent population is considerable, amounting to nearly 10,000 persons. Its sources of water are very numerous, and the waters are varied in character, only a few of them being sulphur waters. Its hot springs were well known to the Romans. The town is picturesque, and is well provided with good hotels. Some of the winter residents from Pau take up their quarters in Bigorre for the summer months.

CHAPTER XX

LARYNGEAL SPASM[1]

THE involuntary contraction of a muscle, which we call 'spasm,' is of two kinds, prolonged and momentary, tonic and clonic. A typical tonic spasm has occasionally been seen and felt by everybody during an attack of cramp in the calf of the leg. The muscle is hard and contracted, and there can be no doubt about the condition. 'Clonic spasms' are often so rapid that the condition of the muscle is rather to be inferred than demonstrated. The various forms of tremor, the jerking movements of nystagmus, or of histrionic spasm, the unpredicable jerking of chorea, post-hemiplegic movements (co-ordinated or otherwise), and the rhythmical movements to which the name of clonus is given are all samples of clonic spasm. In the two acts of yawning and laughing we have, as it were, the physiological prototypes of tonic and clonic spasm. Both these acts, when fully developed, are accompanied by laryngeal sounds, but neither of these sounds is ordinarily attributed to 'laryngeal spasm,' although there can be little doubt that in both instances there is involuntary movement of the laryngeal muscles, in one case tonic and in the other clonic.

In yawning and laughter the sound is truly vocal, and is produced in each instance mainly by expiration, any inspiratory sound being only occasionally interpolated.

The word 'spasm' is also often applied somewhat loosely to any sudden attack of pain or difficulty ; but it is well to remember that when we speak of intestinal, renal, biliary, or pulmonary spasm the actual condition of any muscle con-

[1] *Medical Chronicle*, 1895.

cerned is more a matter of inference than demonstration. The term 'laryngeal spasm' is usually reserved for certain sudden attacks of inspiratory dyspnœa, or for certain laryngeal sounds suddenly produced during inspiration. It is hardly too much to say that every inspiratory crowing sound is liable to be spoken of as 'laryngeal spasm,' although any condition of spasmodic contraction on the part of any of the laryngeal muscles is incapable of actual demonstration.

The act of inspiration is generally noiseless. All the inspiratory muscles act in harmony. The act may be either forcible or feeble, but the strength or weakness of the act depends on a harmonious conspiracy of many muscles, which contract strongly together, or feebly together, as the case may be.

Let us confine our attention to two sets of inspiratory muscles only—the diaphragm and the abductors of the vocal cords. With every descent of the diaphragm the abductors come into play and the glottis is opened. These muscles are both actuated through the respiratory centre in the medulla, and the width of the glottic opening always bears a direct proportion to the force with which the diaphragm descends. So long as this direct proportion is maintained the inspiratory act is noiseless, but so soon as the glottis fails to open to a degree which is proportioned to the strength of the diaphragmatic contraction the act of inspiration is accompanied by *stertor*.

The patient who has some slight thickening of the vocal cords or abductor paralysis of one or both cords may breathe quite naturally and noiselessly, notwithstanding the narrow glottis, so long as he is at rest and the diaphragm is working gently and slowly ; but if he is asked to walk upstairs or to run a few steps the breathing quickly becomes stertorous or stridulous, because the glottic opening no longer has a width proportionate to the strength of the diaphragmatic contraction, and the incoming air rushes through the comparatively narrow glottis with a rapidity which is sufficient to produce noise.

In the cases we have imagined the noisy breathing is due to the fact that the orifice of the pipe is too small for the

blast, but there is no question of muscular spasm, the relative smallness of the orifice being caused in the one case by exudative thickening, and in the other case by *muscular paralysis* (!).

If the diaphragm descend with a force which is abnormally great, the incoming blast may be sufficiently strong to produce stertor in a normal glottis. This is heard in whooping cough and in vomiting.

In whooping cough the continued and repeated action of the expiratory muscles has the effect of emptying the thorax to its maximum extent. The arch of the diaphragm is driven upwards to the highest possible point, and when the patient seems to be on the very verge of asphyxia down comes the diaphragm with a force and rapidity far in excess of the normal ; there is a disproportion between the incoming blast and the size of the pipe, and the stertorous noise known as the 'whoop' is produced. This 'whoop,' time out of mind, has been spoken of as being caused by a 'spasm of the glottis,' a statement which is a mere assertion incapable of proof. That the glottis is too small to allow the incoming blast to pass without noise is true. That the incoming blast by which a paroxysm of whooping cough is terminated must of necessity be one of exceptional force is certain, but whether the glottic aperture be opened to its normal extent or be lessened either by abductor insufficiency or adductor spasm must always remain a matter of speculation.

In vomiting the abdomen is compressed, and the whole of the lower part of the thorax is in a position of extreme expiration. The vomiting effort being ended, down comes the diaphragm, and not infrequently a 'whoop' is generated by the exceptional blast passing through the relatively small glottis.

In the same way, and from similar causes, one has heard a 'whoop' occasionally interspersed among the noises of a paroxysm of uncontrolled laughter.

Perhaps the most common instance of laryngeal stertor is 'hiccup,' when the contraction of the diaphragm, taking place suddenly and irrespective of any respiratory need, is not accompanied by any corresponding abduction of the vocal

cords. Hiccup has frequently been spoken of as a laryngeal spasm; but of this there is no evidence whatever. It is probably much nearer the truth to speak of it as a diaphragmatic spasm caused by local or reflex irritation, and occurring at a time when the glottis is not sufficiently open to allow the noiseless passage of the sudden incoming blast.

In the administration of chloroform, when the patient is deeply under the influence of the anæsthetic the act of inspiration is liable to be accompanied by a slight laryngeal stertor, a sound which warns the administrator to be careful, and tells him that the respiration is encumbered. This stertor has been spoken of as due to glottic *spasm*, but, in reality, it is almost certainly due to paralysis of the abductors, which succumb to the influence of the narcotic earlier than the diaphragm. The diaphragm contracts vigorously at a time when the posterior crico-arytænoid muscles, following the lead of the other voluntary muscles, are beginning to fail; the glottis is not opened, the volume of the blast is too big for the orifice, and stertor is the result.

The laryngeal stertor of chloroform narcosis, which is always a sign of danger, will often disappear if the chin of the anæsthetised person is pulled upwards; and if this fails the tip of the tongue must be seized in a pair of forceps and be pulled forward with a sharp sudden jerk. This manœuvre is effectual by its reflex effect upon the abductors of the glottis, and not by a mere mechanical pulling forward of the mass of the tongue. It was Sir Joseph Lister who pointed out that for the removal of laryngeal stertor the effect produced upon the tip of the tongue must be of a sharp stimulating character.

So far reasons have been given for doubting the dogma that laryngeal stertor is caused by a spasmodic action of the adductor muscles of the glottis. Direct observation of the larynx at the time of the stertor is impossible, and even were it possible we should only be able to infer the condition of the laryngeal muscles from the position of the vocal cords, and the difficulty of determining whether any abnormality of the glottic

aperture was due to over-action of one set of muscles, or under-action of their antagonists, would still confront us.

There still remains for consideration one condition which is enshrined in all the text-books of medicine as a spasmodic disease, and is regarded by many as a disease of the nervous system allied to epilepsy, angina, or chorea, and which has received the name of *laryngismus stridulus*. This is a disease which is met with almost exclusively among very young and rickety children. As a rule the child is awakened by, or is seized on waking from a sound sleep with, an acute difficulty of breathing, accompanied by a crowing noise. The difficulty of breathing is extreme, so that evidence of an asphyxial condition is apparent in the countenance, and if this condition is prolonged there are not uncommonly contractions of the hands and feet, which have been spoken of as carpo-pedal contractions, and the occurrence of which has been considered as affording a reason for considering the laryngeal condition spasmodic, and for placing the disease among the neuroses.

Laryngismus stridulus is a symptom of rickets, and occurs only in those who have the soft bones and cartilages, the flabby muscles, and the general anæmic feebleness which are characteristic of that condition.

Many rather fine spun theories have been put forward to account for laryngismus by referring its pathology to the nerve-centres, but it is not too much to say that none of these are entirely satisfactory.

It seems to me to be more reasonable to assume that this sudden difficulty in breathing arises from the softness and feebleness of cartilage and muscles and other tissues, and that when the child calls, so to say, upon its diaphragm for a more vigorous contraction than usual the glottis fails to open adequately and the soft parts sink inwards.

When Marshall Hall was experimenting on 'asphyxia' he tied the nozzle of a big syringe into the tracheal end of a donkey's trachea and larynx removed from the body. He found that when the piston of the syringe was withdrawn gently air passed freely through the larynx, but that if the

piston were withdrawn suddenly the upper end of the larynx
was driven inwards by the pressure of air, and the passage of
air through the larynx was arrested. This experiment is
interesting, but one must remember that there is a great
difference between a dead larynx and a living one, and that
the posterior crico-arytænoid muscles, whose duty it is to open
the door for the incoming blast, play a most important part in
the act of respiration.

We have only to look at the pigeon breast of a rickety
child in order to be reminded of Marshall Hall's experiment
alluded to above, and to be impressed with the fact that the
softest part of the too flexible thorax of these flabby little
patients cannot withstand the external pressure of the atmos-
phere when the diaphragm descends. The respiratory power
of rickety children, in consequence of their soft bones and
feeble muscles, is necessarily imperfect ; it is very difficult (if
indeed it is possible) for them to inflate their lungs completely ;
they are very prone to suffer from partial collapse of the lung,
and it is well known that rickety children form a large pro-
portion of the victims of respiratory disease. Children
circumstanced as are the rickety, who cannot play, who dislike
to be moved, who habitually keep perfectly still and work
their diaphragms at half pressure, now and again feel the need
of a really deep breath, and this is especially the case when
the feeble respiration has been still further reduced by sleep.
When the diaphragm descends with extra force the crico-
arytænoidei postici do not contract with proportional force,
and the flabby larynx closes before the external pressure of
the air and the soft epiglottis is driven down upon the larynx
before the inspiratory current. The tube of the larynx, not
being stiff enough, collapses, just as the indiarubber tubes of
an aspirator are liable to do. Incarceration of the epiglottis
in the upper part of the larynx has been described as occur-
ring in this condition, a fact which would lend considerable
support to the argument which I have advanced. All the
phenomena which follow the closure of the rickety child's
windpipe are consequent upon it, and are bred of terror *plus*
asphyxia, and any convulsions from which the child may suffer

may be regarded as a consequence of the asphyxial condition, which, as a cause, is perfectly adequate, and renders it gratuitous on our parts to seek another explanation.

Few of us have had the experience of suddenly awaking from sleep with an impermeable windpipe. It is difficult, however, to imagine anything which can be more distressing or alarming, or bring more vivid conviction of impending death.

This question as to whether in laryngismus stridulus the phenomena are caused by muscular cramps in the larynx or by the collapse of the soft tissues out of sheer feebleness is by no means unimportant, because the treatment to be adopted will necessarily be influenced by the view which is taken by the practitioner of the pathology of the disorder. Is the child to have antispasmodics or tonics? Is it to have chloroform for the spasm and to be drenched with chloral, bromide, belladonna, *et hoc genus omne*, or is the tongue to be pulled sharply forward, as in chloroform stertor, the respiration to be roused by cold affusion, and, subsequent to the attack, the body to be strengthened by fresh air and the various means, dietetic and therapeutic, which need not be dwelt upon here?

The fact that the strengthening and rousing regimen—cod-liver oil and iron, milk, and cold bathing—is that which finds most favour with writers on the subject is perhaps another argument for abandoning the spasm theory in relation to the stridulous dyspnœa of the rickety.

So many reasons have been brought forward for doubting whether many of the conditions which are associated with laryngeal stertor are really due to a spasmodic cramp-like action of the adductors of the cord that one is compelled to ask whether spasmodic closure of the glottis ever occurs? Its occurrence, as has been said, must always be a matter of inference rather than demonstration. To use the laryngoscope during an attack of spasm is impossible, and even could this be done the cause of a narrow or closed glottic chink would be doubtful.

When a crumb 'goes the wrong way,' and there occurs that cataclysmic explosion of coughing and stridor which we

have all witnessed, it is in the highest degree probable that a spasmodic action of the adductors of the vocal cord is one of the elements of the crisis ; but as this is in the highest degree purposive, and is, as it were, Nature's therapeusis to prevent the crumb being drawn inwards, and to enable the patient to expel it by coughing, it can hardly be regarded as pathological.

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