

## CHAPTER XXXI

### REPORTS ON PROBABLE CAUSES OF DEATH AFTER THE EVENT

ONE may be called upon to advise either an insurance company or a firm of solicitors without having an opportunity of making a medical examination of the person involved; indeed, it not infrequently happens that the action has arisen after the death of the patient. No question of deliberate fraud arises in these cases; but in attempting to hold the balance evenly, there is the danger of being misled by written statements of witnesses who are naturally very sympathetic with the widow of their late comrade, and whose powers of observation are much below the normal, especially with regard to what they may have considered, at the time, trifling circumstances.

The following cases, although they do not perhaps in one sense rightly find a place in a work devoted to malingering, and raise questions rather of medical jurisprudence than fraud, are, however, quoted at some length, for they illustrate interesting points which may be useful to the reader.

**Burn Alleged Cause of Carbuncle.**—The first case is that of a metal-worker, who was burned on the head and neck by an alloy of tin, lead, antimony, and copper. The injury was produced in the usual way, the spoon which was being used having been allowed to become moist with water. The molten metal was said to have fallen over the man's head, burning his neck and shoulders. He died six months later of acute septicæmia, following a carbuncle.

I was asked to advise upon this question: Was there a reasonable presumption of cause and effect between the burn and subsequent carbuncle? It appeared that the man did not stop working until forty-three days after the burn,

and there was some loose evidence to the effect that the wound healed up about four or five days after the accident. It also appeared that the deceased was suffering from Bright's disease in a fairly advanced stage at the time of his death. The plaintiff's case was that the carbuncle which produced the septicæmia arose from the penetration of a germ into the wound caused by the molten metal. On the other hand, the defence suggested that an advanced stage of Bright's disease, and the concomitant deterioration of the deceased's health, especially predisposed him, apart altogether from the burn, to the infection which produced the carbuncle.

In other words, the plaintiff's alleged direct causation was, in the defendant's view, a matter of coincidence.

In reviewing the case, the following statement was made :

D. J.—A carbuncle is produced by a small micro-organism (*Staphylococcus pyogenes aureus*). This germ is found naturally on the skin, even in health, but sometimes penetrates the skin at the seat of a hair-follicle, inflames the tissues beneath the skin, and in any condition where the health is lowered may end in a carbuncle.

It is very well known in the practice of medicine that the two conditions known as diabetes and Bright's disease especially predispose to carbuncles. The deceased D. J. was admittedly, by the post-mortem report of the superintendent of — Infirmary, suffering from Bright's disease. He was therefore predisposed to carbuncle.

It appears that D. J. sustained certain burns produced by molten metal. It does not appear that there was in any way a serious accident, for I cannot find that he even stopped work. The only remedial treatment he had seems to have been first-aid from his fellow-workmen. He did, however, put himself on the sick-list forty-three days after the accident above referred to.

I understand that it may be alleged that the germ producing the carbuncle found entrance into this man's body in consequence of :

- (1) A weakened condition of the skin, the result of the burn, or
- (2) The fact that the skin never had healed.

With regard to (1), I am prepared to state that it is impossible that the germ could be facilitated in its entry into the body by the mere fact that there had been a burn which had healed weeks previously. The presence of scar-tissue, which is really tougher than the skin, and free from the presence of hair-follicles (down which, as already explained, the germ penetrates), would *in itself* hinder rather than facilitate the entrance of the germ into the body.

(2) On the other hand, if I believed that the original burn had *not* healed prior to the commencement of the first symptoms of the carbuncle, it would be impossible to deny that an open wound, if it

existed, would form a ready entrance for the specific organism producing carbuncle.

The case, therefore, turns almost wholly upon the question as to whether or not this man did, in fact, wholly recover from the burn.

Assuming that the burn had healed, the fact that the carbuncle itself was found in the neighbourhood is not such a strange coincidence as would at first appear, for the back of the neck is by far the most usual site on which carbuncle appears.

**Death alleged Due to Fall in Subject of Old-standing Heart Disease.**—The following report, to a large extent, speaks for itself:

D. K., a vanman, was slowly driving up a hill, when he was seen to fall suddenly forwards from his seat in the van, striking the ground first with his head. A passer-by went at once to his aid, and although there was blood in the neighbourhood of the wound, the deceased admittedly never moved after reaching the ground. He was known to be suffering from marked heart disease, and the question which I was asked to decide was whether his death was entirely due to heart disease. It was stated in the evidence given by the doctor who attended him that the fact that there was some bleeding from the wound proved conclusively that the man was alive after the fall.

The following opinion was based upon the report of the coroner's inquest.

I am of opinion that this man died as the result of heart disease, that he died suddenly whilst sitting in the cart, and that his fall occurred, and his subsequent injuries were received, after death.

*The Statement as to Bleeding.*—It is important that the anatomical facts with regard to hæmorrhage should be clearly understood, otherwise the erroneous medical assumptions which have already been made in this case may be repeated.

The blood is contained in the arteries under pressure. This is proved by the fact that when an artery is cut the blood spurts, and may do so several feet. It will be noted that no witness speaks of actual spurting of blood, as occurs in life, and the impression formed by reading the depositions is, that blood *oozed* after death, and did not spurt out.

When, therefore, a scalp-wound some 3 inches long—as was found on this man's head—is inflicted on the body of a man who has died only a few seconds previously, a considerable amount of blood would certainly ooze from the wound—*i.e.*, he would certainly bleed.

It is a fact that as much as 2 or 3 ounces have escaped from the arteries of dead persons, and it can also be proved that an even larger quantity escapes from veins. Bleeding after an accident is no physiological proof that life is not extinct; of this there is no doubt.

The wound was on *the back of this man's head*, and was therefore in



the most dependent position, and the blood would naturally drain from the open wound, more especially when he was being carried, as he would be, face upwards.

A wound of this sort, occurring immediately *after* death, would stop bleeding (that is, draining from the wound), when, and only when, the blood in the artery coagulated—*i.e.*, clotted—which would not take place under a few minutes.

The vessels of the scalp are particularly prone to bleed; this is a well-known fact with regard to ordinary surgical wounds in that region, and the reason for this is that they are not (as in the limbs and the rest of the body) lying in large masses of flesh, muscle, etc., but upon the bone of the skull, and are embedded in the tough, firm tissues of the scalp. Their position, therefore, keeps them from contracting and collapsing as other vessels in other parts of the body do when cut or injured. Contraction, collapse and coagulation are nature's methods of stopping hæmorrhage.

It must not be forgotten that the witness who saw the accident would be shocked at the whole of the circumstances. He would, consequently, not be able to testify accurately as to details. The appearance of even a small quantity of bright red blood, which would smear the scalp and head generally, would give an *impression of quantity* out of all proportion to that actually present.

I can find no mention of any attempt being made to treat this wound even by first-aid measures; no one seems to have even *thought* of applying a handkerchief to the open wound, which they might have been expected to do, if they thought D. K. was alive.

It is very noticeable that no single witness suggests that he saw any evidence of life; indeed, the carman who saw the deceased fall distinctly states, "I saw no sign of life at all"; and his significant statement, "I can't say if he was alive," is followed by the further statement that "the road was in a good state," and gives the impression that at the moment he was carrying his mind back to the *time* of the actual occurrence of the fall. Indeed, this witness seems only to have been clear about two things—that he saw blood, and that there never was any sign of life.

His statement that the nurse at the hospital "saw the bleeding" means, of course, that she saw the blood, for had D. K. actually been bleeding on his arrival at the hospital, he must have been alive for some time after the accident.

*Post-Mortem Condition of Heart.*—The medical witness who made the examination admits that the heart "was diseased pretty extensively, of some years' standing." His statement that D. K. had "valvular disease and heart-muscle disease" is of considerable moment, for disease of the muscle of the heart is particularly prone to be followed by sudden, fatal syncope.

Disease of certain of the valves of the heart is more likely than that of some others to end in sudden death, and it is important to ascertain *which* valves were actually involved. This is not stated in the post-mortem report.



*The Medical Witness's Theory as to the Cause of Death.*—The medical witness has taken up a position which at first sight is difficult to controvert. What he says, in effect, is: "This man had a serious fainting attack, fell, and severely injured himself. I do not know that either the attack of syncope or the fall separately would have killed him, but I believe the fall was the last straw, as it were, and that he might have lived had he not had the fall."

It will be noticed that the value of his evidence depends entirely upon whether this man was alive when he reached the ground, and his sole reason for stating that the man was alive was that blood was found on his scalp at the post-mortem.

I have already dealt with this subject, but I would here add that so far from the blood which was found in the neighbourhood of the wound being an evidence of life, I can confidently affirm that, *assuming as a fact that this man died before he fell*, the accident could not have occurred as it did without blood being found in the neighbourhood of the wound.

The possibility of a man having a fainting attack, falling, and so injuring himself as to make the fainting attack a fatal one, cannot, of course, be denied. In this particular case—apart from the hæmorrhage, the presence of which I think I have explained—the statement of the alleged sequence of events cannot be anything but a theory. Against this theory there are the following points worthy of consideration:

1. The heart was doubly diseased—*i.e.*, both in muscle and valve.
2. Fatal syncope is extremely common in this condition.
3. No witness attempted to say that he ever saw the man give any sign of life after his fall. Even the carman who was passing at the time could not give any evidence of life to support the doctor's theory that the man was alive after the fall.
4. It is not suggested that the deceased was jolted off his cart, for the horse was proved to be quiet.
5. Had this man been killed by the fall, one would have expected a few involuntary movements, such as sighing, or deep respirations, to have been observed prior to his actual decease.

In this connection it is interesting to note that a case, identical in details, has recently been decided—*Thackray v. Connelly*, 1909, 3 B.W.C.C., 37, as reported in *Knocker's "Accidents in their Medico-legal Aspect,"* p. 958. In this case the point at issue was heart disease or concussion, and the Judge decided in favour of heart disease. In *D. K.'s* case we have the ingenious theory advanced that he died, not of heart disease, nor of concussion, but from the two taken together.

If it can be proved that the concussion was post-mortem, which I certainly think it can be, the verdict should go in favour of the company.

*Conclusion.*—I am of opinion that the evidence given at the inquest points to death from a sudden fatal attack of syncope—*i.e.*, stoppage of the heart's action as the direct result of previously existing serious heart disease, and that the accidental fall which followed upon his

dying while sitting on the cart neither caused nor accelerated his death.

The case came to Court, and the plaintiffs failed to establish their case.

**Tuberculosis of Alleged Traumatic Origin.**—The possibility of tuberculosis being caused by an injury opens a wide field for the speculative lawyer. It cannot be denied that, assuming the presence of tubercle bacillus, traumatism may, under exceptional circumstances, be the determining or predisposing cause of its renewed activity. Dr. Theodore Williams, at a recent meeting of the Insurance Medical Officers' Society, stated that he had seen symptoms gradually develop after traumatism where there had been an injury of some sort, such as a fall from a horse, or a blow; and said that when a patient had had well-marked tubercular disease in the right lung, even though there were but few signs remaining, he would warn such a patient of the danger of a kick from a gun or a blow in boxing, in case the slight traumatism might provoke to fresh activity the old focus of infection.

The following opinion was based upon medical certificates and statements in the case of a man who, whilst at work on board ship, suffered from an immediate hæmoptysis, as the alleged result of a strain. He never worked again, and died some nine and a half months after the bleeding. The widow claimed that the accident caused the hæmorrhage, and was accountable for her late husband's death. As acceleration was likely to be raised as an alternative plea, occasion was taken to deal also with that point.

The report—as all reports written to laymen should be—is expressed in popular language :

*Opinion*—D. L.—I have taken occasion to read over the whole of the papers submitted to me in this case, have consulted with Dr. X, who examined the deceased, and have searched the recent literature on the subject.

In this case two important questions at once present themselves :

1. Was the phthisis of which D. L. undoubtedly died caused by the accident ?
  2. Alternatively, did the accident *accelerate* the disease, if pre-existing ?
1. Did the accident cause the phthisis ?

There is but one answer to this, and that is, that it is impossible for the accident to have *caused* the disease.



Phthisis is an infectious disease produced by the tubercle bacillus, and it cannot be produced in any other way.

D. L. was first examined by Dr. X some six weeks after the accident. It is reported that this examination showed well-marked physical signs of what might be described as old-standing phthisis. Indeed, Dr. X on that occasion discovered the tubercle bacillus present.

Further, the post-mortem examination some eight and a half months after Dr. X's first examination showed unmistakable evidence, in one lung at least, of the disease having existed for many years. I refer to the fibroid condition—a condition which is well known to take years in its formation.

2. Did the accident *accelerate* the pre-existing disease ?

Assuming, then, pre-existing disease, the following points require consideration :

(1) *Nature of the Accident.*—The allegation seems to be that D. L. was lifting a heavy weight from the side of the ship with another man, or men, when the others suddenly let the weight go, and consequently the whole strain was put upon the deceased. Now, it must be admitted that, if this be true, a very severe sudden strain must have been put upon D. L. He was suffering at the time from phthisis, and the hæmorrhage which is alleged to have immediately taken place is not at all an unlikely event.

When considering the question whether this attack of hæmorrhage did or did not accelerate this man's disease, it must be remembered that we have here a man who had been medically passed as fit for sea; who was, in fact, working up to the moment the accident occurred; who has never worked since; and who actually died of acute phthisis within ten months after the accident.

(2) *Cause of Hæmorrhage in Phthisis.*—Physical strain in those already suffering from phthisis may lead to pulmonary hæmorrhage in at least two ways: (a) From the rupture of a dilatation of one of the small arteries in the lung. When this occurs the hæmorrhage is sudden and copious, and is most frequently found in the chronic fibroid form of the disease. (b) From mechanical disturbance of an old focus of disease followed by fresh inflammation and congestion of the part, and as a result of the congestion some of the small bloodvessels may give way and so produce hæmorrhage. If an *overdose* of tuberculin is given to a phthisical patient, fever results, followed by evidence of increased activity of the disease in the lung, not infrequently accompanied by hæmoptysis.

Physical overstrain or injury to a phthisical patient is frequently followed by the same sequence of events, and it is fair to assume that as a result of trauma an overdose of home-made tuberculin has been liberated into the circulation. In both cases the fever and focal activity may progress towards a fatal termination, often within a year.

According to Dr. X's notes, the deceased stated to him at an examination some five months after the accident that he had had other small hæmorrhages frequently for many days after the occurrence of the first hæmorrhage.



(3) *Dr. X's Conclusion.*—Amongst the papers submitted to me I find notes written in Dr. X's handwriting; these are evidently notes of a report, and state that at the examination he conducted six weeks after the accident the condition of the lung (though far advanced in phthisis) was quiescent. Dr. X bases his opinion that it was "a very doubtful question whether the accident accelerated the disease" (phthisis) on two grounds: (a) On account of the lack of sputum (expectoration), and (b) on account of the small number of tubercle bacilli which were found.

I do not agree with Dr. X's conclusions.

*General Conclusions.*—1. D. L. had phthisis before his accident.  
2. The accident accelerated the disease, but it is impossible to say to what extent.

This report was, of course, not satisfactory to the employer, who sent it, with all the documents, to another medical man. Quite a different report, expressing a totally opposite opinion, was given. This was supported in Court in the witness-box, and the widow was deprived of any compensation under the Workmen's Compensation Act. After the case was over, the employer sent me all the papers, including the second medical opinion he had obtained, and asked me what I thought *now*. I said I was more convinced than ever my view was correct, *with which he agreed!*

**Grounds for Medical Opinion should be Stated.**—A very common error made by those who are not frequently called upon to write reports in medico-legal cases is to give an opinion, but to give no reasons for the opinion. Not unnaturally many medical men argue that a layman, having referred a difficult case to medical authority, should accept that authority without question. I am confident, however, as the result of experience, that this is a mistake. It is always difficult for those who have for many years habitually expressed themselves in technical language to make medical matters clear to laymen; nevertheless, it is worth while doing so, because the fact must not be overlooked that in many instances reports are for the information of persons who have to decide whether or not claims shall be resisted. It is, therefore, of the utmost importance that a medical report should be in logical form, and clearly indicate the grounds upon which the ultimate conclusion has been arrived at by the medical examiner.

Further, there is nothing undignified in giving the reasons for one's judgments. The decisions given by the Judges in all the Courts are nearly always accompanied by their reasons, and the fear of explaining why one has come to a certain decision reminds one of the advice given by a Scotch attorney to a friend who had recently been elevated to the Bench, and about whose decisions he was somewhat anxious: "Never add any reasons in your judgments, Donald; for, whilst they will probably be right, your reasons may be wrong!"

**Empyema alleged Due to Accident to Hand.**—The following case was submitted to a competent medical man, and a very definite, sound opinion was given by him upon the facts; but his certificate merely stated the opinion, giving no grounds for the decision, and failed in that it did not discuss the facts as they were stated in the depositions of the various witnesses. Consequently, it did not satisfy the employers, who referred the case to me; indeed, it was only subsequent to their receiving my report that I learned that they had had a previous opinion which had not inspired them with sufficient confidence to take action.

*Opinion*—D. M.—I have carefully perused the papers submitted to me in the case of the death of D. M.

It appears that on January —, 19—, the master of the ship is said to have received a superficial graze on the back of his hand. This apparently is sworn to by two witnesses, one of whom, F., was at the opposite side of the ship at the time of the alleged accident. Both witnesses speak of the abrasion having rapidly healed; there is not the slightest evidence to connect this abrasion with the master's subsequent death nine weeks and three days later.

It is quite apparent, in view of the symptoms described—persistent pain in side, difficulty in breathing, disregard of food, increasing pallor and emaciation, and the subsequent removal at hospital of a large quantity of pus—that the deceased died from empyema.

Empyema consists of a collection of matter (pus) in the chest, between the outer surface of the lung and the inner wall of the chest.

Now, whilst I have no doubt that the alleged accident to the hand on January —, 19—, cannot be connected in any way with the death, there is just the *possibility* that a blow on the side said to have happened three days later, might, by breaking a rib or ribs, have caused traumatic pleurisy, with effusion, and effusion not infrequently results in empyema.

In this connection, however, the important points to my mind are the history of the alleged injury to his side, and the conduct of the injured man immediately afterwards. What do we find?



F. states: "The sheet struck him [the master] on the right side. He did not seem to be much hurt, and he then went to his cabin, and *had his dinner*. This would be about 12 o'clock. The master came up on deck again at about 6 p.m., and I did not hear him make any complaint of feeling any effect of the blow he had received."

L. states: "... Caught the master a sharp blow on the right side. . . . He stopped working for a few minutes, after which he did not appear to be in any pain, and made no complaint of feeling any effects of this blow during the remainder of that day."

It is true that he complained of pain the *next morning*; but if he had had a traumatic pleurisy, the result of a fractured rib, he must have had severe pain at the time of the blow, disabling him forthwith from further work.

After due consideration, therefore, I have come to the conclusion that one cannot fairly say that this man's death was in any way attributable to any accident.

Pleurisy, with effusion, is most frequently of tubercular origin. I think this poor fellow was probably ill at the time, and that, as his symptoms appeared to develop coincidentally with his comparatively trifling bruise, the men working with him not unnaturally, after thinking the matter over, put the two together, and came to the erroneous *post hoc ergo propter hoc* conclusion. The entries in the log give sufficient evidence that *at the time* the writer considered the matter of no importance.

I have no doubt that want of early proper medical attention, and the delayed removal of the pus, and so forth, were material factors in bringing about the fatal issue.

The deceased's doctor suggests that the empyema was an evidence of blood-poisoning produced by the graze on the back of the hand. Empyema is sometimes the result of blood-poisoning, but it is the *last* stage of a very serious general disease—i.e., general blood-poisoning—which would have attacked the whole system, and shown itself by numerous abscesses in different parts of the body.

If the empyema had been the result of the hand injury, the glands of the arm-pit would have become inflamed; it would, indeed, have been the *last* symptom of a general blood-poisoning, which the master certainly did not have.

I am satisfied the case *does not* come within the four corners of the Workmen's Compensation Act, 1906.

The case was compromised by the payment of £100.

**Death from Syncope accelerated by Fracture.**—The following case illustrates two points: (1) The importance, as already emphasized, of setting out in detail one's reasons for arriving at a conclusion which a layman could not be expected to accept without explanation; (2) the importance of recognizing the legal liability of employers where disease, which is not necessarily the cause of death, has accelerated it,



D. N., the deceased, aged forty-seven, teacher at a technical school, was showing a pupil how to file piping when the file slipped, and he fell with his legs crossed, fracturing his thigh. He died twenty-six days later, and I was subsequently asked to consider the depositions taken at the inquest, and to give an opinion as to whether the circumstances attending the deceased's death were such as to indicate that the death resulted from or was accelerated by the accident.

*Opinion.*—The deceased was evidently a heavy and plethoric man. The post-mortem examination showed that he had a fatty heart—that is to say, not fat *round* the heart (as is popularly supposed), but part of the muscle fibre of the heart was replaced by fatty deposit, thus weakening the contracting power of the heart.

I see that the witness S. states that during life he examined the heart once or twice, and that the sounds were “rather faint.” This medical witness states that there were no signs of shock for some time prior to death. Were it contended that this man died of shock in the ordinary sense I should not agree, for it is quite apparent from the evidence that there were no symptoms of shock.

But there are other considerations.

Here is a man, unhealthy, flabby, well advanced in middle-life, who is suddenly, as the result of a serious accident, laid on his back. His whole environment is changed. His secretions and normal tissue changes are thrown out of gear. His heart is already fatty. The amount of normal muscular tissue has therefore been diminished. The enforced rest in bed, the sudden change, and the pain he must have endured, cannot but have tended to enfeeble what healthy muscular tissue remained in his heart. People with fatty hearts always have faint heart-sounds, and the impulse is not powerful, because the heart has not the normal driving force. Such persons not infrequently die from any cause which disturbs the balance of vital energy which is necessary for the innervation of the heart.

In this case I think the question is: Did the circumstances of his accident disturb the balance between the natural nervous force which regulates the heart's action, and the processes which tended towards syncope? I think it is impossible to deny that the fracture had just that influence which produced the syncope which ended in death.

I am of opinion, therefore, that this accident undoubtedly accelerated the man's death, and that from the post-mortem picture, as I see it in the report, there was, apart from the intervening accident, no reason to anticipate a fatal issue for some considerable time.

The sum of £300 was paid to the widow under the Workmen's Compensation Act. The case did not go to arbitration.

**Aneurism alleged Due to Strain.**—The following case illustrates the uncertainty of the result when a medical question has, of necessity, to be decided by a layman. I think my

professional brothers will agree with me that there was very little doubt, from a medical point of view, as to the cause of the death of the deceased; but the evidence of a fellow-seaman, whom the Judge specially called in the case, seems to have influenced His Honour in favour of the deceased's dependants.

Our judgments are arrived at by one of two methods—either by reason or sentiment. These two parallel methods of arriving at a conclusion cannot possibly converge. Defendants are heavily weighted when the Workmen's Compensation Act is invoked in the interests of a widow and dependants.

*History*—D. O.—The deceased seaman, aged twenty-eight, was ordered to repair the topsail after a gale whilst at sea. He went aloft with the usual foot-ropes, his chest and stomach rested on the iron, and he had to lean over the yard in different positions to mend the sail. He was aloft some five hours, considerable seas rolling meantime, so that he required to keep a tight hold. Thirty-six days later he first complained, saying he had pain in his chest, and a cough. In a few days he seemed all right, and made no further complaint to the captain. Forty days later—i.e., seventy-six days after repairing the sail—he was found looking very ill, suffering from acute pains in the stomach. He was taken to the water-closet, and then to his berth, and given a dose of castor-oil. The evening of the same day he died. He had always been a strong, healthy man. The day before his death he ate cold meat and potatoes.

The case set up for the applicant was that in consequence of the deceased's work in repairing the sail an aneurism was produced, which caused his death.

*Opinion*.—I have carefully perused the papers submitted to me, and am requested to give my view as to whether the statements contained in these papers show that the deceased died from an accident or from natural causes.

I have come to the definite conclusion that there is no evidence whatever that this man died as the result of an accident. It will be noticed that the deceased boatswain went aloft to repair a sail (E.'s statement); this, I take it, would be in the ordinary course of a sailor's duties. It will be noticed that he complained vaguely of pains in his chest, but not, apparently, until two days later (M.'s statement). From that time until eleven weeks later he appears to have been pretty well, and kept at his work. Indeed, he was at work right up till five hours before his death.

The sudden onset of the illness, the severe abdominal pain (entry in official log), the faintness, the collapse, the desire to go to the lavatory, the sudden termination of the disease, ending fatally—all point to an internal rupture, rapidly supervening peritonitis, accompanied (as it always is) with collapse, and finally cardiac failure.



The exact nature of the rupture it is impossible now to state. The likelihood is that it was one of three things :

1. Ruptured abdominal aneurism.
2. Gastric ulcer, which perforated ; this would be consistent with the alleged indefinite pains in the chest.
3. Abscess of the appendix suddenly bursting into the abdominal cavity ; this in all probability was the real cause.

To my mind there is not a vestige of real evidence upon which the deceased's dependants can in any way substantiate a claim for compensation in respect of an accident.

Had the condition been produced by pressure on his chest whilst in the rigging, severe pain would inevitably have been felt *at the time*. It will be noted that though two witnesses (G. and M.) mentioned that the deceased did, within a few days of the date he went aloft to repair the sail, refer to some indefinite pains in his chest, yet he was not incapacitated. Moreover, even whilst in port on two occasions the deceased did not avail himself of the opportunity of seeking medical advice ashore. Indeed, no treatment of any sort appears to have been applied or desired, except the homely remedy of alcohol at night.

I am of opinion that a claim for compensation cannot be seriously maintained in this case.

The County Court Judge held that death arose out of and in the course of employment, and awarded the widow the sum of £213.

In the following interesting cases it is only necessary to relate the facts and to state the deductions arrived at therefrom:

#### **Death from Heart Disease alleged Due to Fractured Ankle.—**

*History*.—D. P.—On a certain date (a year before I was asked to report) the deceased woman fell and fractured her tibia at the ankle-joint. She never walked again after the accident, and died some three and a half months later.

At the inquest the family doctor stated that the deceased D. P. was a healthy woman, but admitted that he had not attended her prior to the accident.

*Opinion*.—I was asked, after perusal of the depositions in the case, to state whether, in my opinion, the death was due to traumatism.

Now, assuming the facts to be correctly stated, it cannot be denied that the accident did contribute to, and in some measure accelerate, the fatal issue ; but, on the other hand, the result of the post-mortem examination left very little doubt that the woman had been a hard drinker, so that the accident could not be regarded as more than a contributory factor in the causation of death.

The deceased's doctor appeared to be of opinion that this woman became insane and had delusions as a direct consequence of the accident, and that her death was, in point of fact, attributable to her accident and ensuing insanity.



Now, the post-mortem revealed that the "heart-muscle"—by which is meant practically the whole heart, for, with the exception of the valves, it is all muscle—was almost entirely replaced by fat; and that the cause of death was "cardiac fatty degeneration of heart, failure of circulation, congestion, and coma." Presumably (according to the medical man) this followed the onset of insanity. I think this is an entirely mistaken view of the case.

It is well known that a fatty heart in a woman of fifty-one might at any moment prove fatal in one of two ways: (1) by sudden failure to overcome the resistance of the arteries; (2) by gradual failure to distend the arteries through a more or less prolonged illness, ending finally in syncope.

In the latter case it is a very usual condition for patients, as a consequence of failing circulation through the brain, to ramble, to become confused, and mistake their surroundings; and by those not accustomed to deal with mental disease this degree of dementia might quite honestly, but erroneously, be termed "delusions."

This point is more important than at first appears, for this reason: Delusions are universally accepted as being evidence of insanity. Now, if this woman's insanity was attributable to the accident, it would entail legal liability which it would not if the heart disease took its natural course and ended fatally, with the usual symptoms of weakness, mental confusion, etc. It must be admitted that the shock of the accident hastened the fatal issue; but the findings of the doctor at the post-mortem examination afford evidence that the woman could not in any case have lived much longer than she did, for he found a fatty heart and atheroma of the aorta. On the other hand, were the jury persuaded that death resulted from insanity brought on by the accident, the liability would obviously be considerably enhanced.

There are certain points that have a bearing on the question of causation of death, and which, I think, should be put to the doctor at the trial. For instance, had the patient a temperature? If so, it is obvious that the death may have been brought about by other causes than delusional insanity, which *per se* is not accompanied by a rise of temperature.

It would be interesting to know whether the doctor would seriously maintain in cross-examination that a woman with a heart which was "almost entirely replaced by fat," with "atheromatous patches in the aorta," was, as he seems to have stated at the inquest, a "healthy woman," and whether the atheroma could have developed in three months!

**Result.**—High Court action. The doctor repeated the evidence he gave at the inquest as above. The jury awarded £128.

**Tight-Lacing; Asphyxia; Death.**—F. W., aged thirty-three, was discovered dead at seven o'clock one morning in a small dressing-room at his residence. An electric radiator was alight, three electric lights were burning, the window was either closed or very slightly open, the month was September. The deceased, who had purposed

attending a fancy-dress dinner, was found to be wearing, *inter alia*, a woman's heavy wig and a pair of corsets, beneath which were a man's vest and a woman's combinations. The last occasion upon which he had worn the corsets was when a youth of nineteen years. His body was discovered in a seated attitude, with his back to a chest of drawers. He had apparently been using some electrical contrivance, as a portable battery was found in an almost closed drawer behind him, the two electrodes of which were slipped down each side of the chest inside the corsets.

A post-mortem examination was made, at which the material facts disclosed were—intense congestion of the brain, congestion of the lungs, slight dilation of the right side of the heart, fluidity of the blood, petechiæ on the elbows, arms, and skin of the chest. Petechiæ were not noted on the outer surface of the lungs or on the pleura. The doctor making the post-mortem examination certified the cause of death as asphyxia brought about by the tight corsets. The deceased was the holder of a policy covering death from accident, but, on a claim being preferred against the company, payment was resisted on the ground that the death was due to cardiac failure, in that, having regard to the conditions discovered at the post-mortem, this was caused by Bright's disease, epilepsy, angina pectoris, or the use of an aphrodisiac such as electricity, chloroform, strychnine, or prussic acid.

In the course of the arbitration proceedings which ensued in consequence of the dispute, the matter was placed before me, with a request to give an opinion as to whether it could be stated with certainty that the death was caused by asphyxia, or whether the probabilities of that being the case were greater or less than the probabilities of the natural causes suggested by the company. After a careful consideration of the papers laid before me, I came to the following conclusions as to facts and the inferences to be drawn therefrom.

1. *As to the Battery.*—This was not of sufficient power to administer a fatal shock; this I proved by actual experiment on myself. The reason for its use was a mere matter of conjecture; but having regard to the fact that the deceased was known to dabble in an amateur way with electricity, and on one occasion had attended a fancy-dress ball with an electric star over his head, I suggested that he had conceived the idea of using the battery in connection with the corsets, with a view to giving electric shocks to his partners in the not unlikely event of a dance following the dinner he had proposed to attend.

2. *As to the Corsets.*—They were last worn when he was a youth of nineteen. In the intervening fourteen years his chest and abdomen must have greatly developed, especially as he had held a commission in His Majesty's forces. He was an athletic man; and having regard to his increased size, and the corsets being put on over a considerable amount of underclothing, there must have been considerable constriction of the chest and consequent embarrassment of his breathing.



The diaphragm by its movements up and down is the chief factor in drawing air into, and ejecting air from, the chest, and this muscle must have been seriously embarrassed by the pressure of the corsets.

3. *As to his Position.*—The sound of a buzzing coil of an electric battery is very penetrating, especially at night; and probably, so as not to attract the attention of his wife, who was in an adjoining room and was convalescing from an illness, he put the battery in the second drawer of a somewhat low chest of drawers, leaving the drawer only sufficiently open to allow the wires leading to the electrodes to emerge. He then commenced experimenting, and found that the length of the wires necessitated his sitting with his back to the chest of drawers, in order to get the electrodes over his shoulder and down the front of the corsets. Sitting in this position the abdominal breathing would be reduced to a minimum, and we thus have both his abdominal and thoracic breathing badly hampered.

Having this condition of affairs, it was necessary to consider how a fatal termination could be induced. A most important physiological fact to be remembered is that, during the process of death by gradual suffocation (such as occurs in smothering or suffocation by the pressure of a crowd), there is no line of demarcation between consciousness and unconsciousness; the one merges imperceptibly into the other. In the Sunderland disaster of 1883, where a large number of children were crushed to death, one of the survivors reported that she remembered being crushed and falling to sleep, and then found herself in bed. The unfortunate man in this case could not have noticed that he was becoming partially asphyxiated and was slowly losing consciousness. A syncopal attack may have precipitated the unconsciousness. This would explain the absence of any attempt to remove the corsets or assume a less constricting position.

The dilation of the superficial bloodvessels would cause the corsets to become tighter, and these embarrassing causes would act and react on each other, accentuating their effects. Once drowsiness and unconsciousness overcame him, the stages leading to death are obvious.

The grounds put forward by the insurance company for contending that death could not have been caused by asphyxia were that petechiæ were not found in very large numbers on the outer surface of the lungs and on the pleura; that the appearance of petechiæ on the surface of the body was a very uncommon characteristic; that the lungs can only become engorged with blood by movement of the chest wall, and this was impossible owing to the constriction by the corsets.

I quoted Taylor's "Principles and Practice of Medical Jurisprudence" (6th edit., p. 636), under the heading "Post-mortem Appearance in Suffocation," to the effect that "In a set of cases examined by Tardieu, the skin of the face, neck, and shoulders, presented dotted or punctiform ecchymoses." These are what are known as "petechiæ," and in this case they were found on the elbow, arms, and chest.



With regard to the suggested absence, in any quantity, of petechiæ on the outer surface of the lungs and pleura, it was contended that the authority quoted states that subpleural, subpericardial, and meningeal bleedings were moderately common in cases of asphyxia, but that petechiæ in these positions are very small, and require to be carefully looked for. I contended that the fact that they were not noted in the post-mortem in this case was accounted for by the statement of the doctor who performed that task, that he "had not looked for them."

The quotation from Taylor's "Principles and Practices of Medical Jurisprudence" was particularly effective, as it appeared for the first time in the *sixth* edition, and that prior to my evidence the *fifth* edition only had been used by our opponents!

I urged further that the congestion of the lungs, together with the slight dilation of the right side of the heart, strongly supported the theory of slow suffocation; for Taylor in the before-mentioned volume, at p. 622, states: "If death has occurred slowly, the lungs are found intensely engorged; if death has occurred very rapidly, the lungs may be quite anæmic, their condition varying inversely with that of the right side of the heart, which in the first case is only moderately distended with blood, but in the latter is so distended as to seem almost on the point of bursting."

No bottle or receptacle was found which could give colour to the suggestion of poison. It was in the highest degree improbable that poison could have produced the effects discovered, which were obviously of a mechanical origin. There was no evidence of the existence of Bright's disease, angina pectoris, status lymphaticus, or any similar condition, and there was strong presumptive evidence against the deceased having suffered from any such disease at any time.

Having regard to all the circumstances, I came to the conclusion that the constriction of the chest, and the partial obliteration of the lumen of the abdominal vena cava, combined with the congestion of the upper part of the chest, had produced a serous apoplexy, the sequence being—congestion of bloodvessels of the brain, gradual unconsciousness, coma, asphyxia.

An eminent Government expert and others gave evidence testifying that, in their opinion, death was due to status lymphaticus, but my reading of the enigma apparently commended itself to the board of arbitrators, who made an award in favour of the claimant.

**Syncope after Immersion—History.**—F. X., aged about fifty, was returning to his ship (of which he was chief engineer) at about 6 p.m. on a February evening. The means of access was a ladder about 20 feet long, one end of which firmly rested about 1 yard from the edge of the quay; the other reached to the stern of the vessel, and was there fastened to a stanchion. The ship was about 6 feet from the quay, and the ladder was therefore at a slight angle. According to the statement of a witness of the occurrence, F. X. proceeded to mount the ladder, holding the rungs as he went up. He got up about three or four steps, when his left leg slipped off the ladder,

his right arm dropped, and he swung partly round, and probably struck his hand on the side of the ladder. He hung by the left hand for about three seconds, and then fell, flat on his face, into the water. One of the ship's firemen got on to a ledge in the dock wall, and succeeded in reaching the immersed man and pulled him on to his knee. Both men were taken off in a boat and put on to a barge, from which they got ashore by means of an iron ladder against the dock wall; from this F. X. walked alone to the ladder from which he had fallen. A rope was fastened round his waist, and the two ends were held by men on the ship, and he then walked up the ladder. He proceeded to his cabin and took off his jacket and waistcoat, but was helped to remove the rest of his things. He gave information as to where his dry clothes were to be found. He was then laid in his bunk and became unconscious. He never spoke again. A sick-bay steward came on board and commenced applying artificial respiration, and a doctor came soon afterwards and assisted. It was not until after the doctor's arrival that the water (stated to be a large quantity) was drained from the man's lungs. Fifteen minutes after the artificial movements were commenced the man died.

The captain of the ship proved that the man had been subject to frequent and sudden attacks of illness, consisting of sickness and prostration, but they usually only lasted for about twelve hours, and he recovered without medical assistance. On one occasion, when he was apparently quite well, he vomited violently without any warning. A doctor had attended him for gastritis during two years, but this was five years before the date of the fatal occurrence.

I was asked to advise whether from the facts stated there was a probability of the deceased having suffered a seizure, which was merely coincidental, and not due to the exertion of going up the ladder.

In my opinion the narrative of the eyewitness was equally consistent with a seizure or an accidental slip. In the absence of post-mortem examination I had difficulty in accepting what took place as a seizure. The symptoms of gastritis do not partake of the nature of sudden disabling pain, such as is common, for instance, in angina pectoris, and the suggestion that there was a sudden seizure of an unknown disease, so severe as to cause him to release his hold of the ladder, was inconsistent with the clinical symptoms of his alleged attacks of illness. It is possible that if the man had suffered from some acute cardiac or abdominal distress he would have acted as described, but it must be remembered that he was practically on all fours on the ladder, and, unless he lost consciousness, would know that his safety lay in remaining *in statu quo* and calling for assistance. If he lost consciousness, his recovery was very rapid, for within a few minutes he was walking unassisted and directing his mates where to find his dry clothes. Any suggestion that the sudden immersion restored his consciousness is not scientifically acceptable. He was probably trusting more to his feet than his hands, in climbing on board. I believe that his left foot slipped from the rung of the ladder while his right hand and foot were in process of removal to



the next rung; this would violently tilt his body to the left. The strain was too much for the one hand, and caused him to release his hold of the ladder, and thus he fell into the water. The right hand being disengaged, would probably be thrust up in an endeavour to regain a hold, and as he turned would give the impression that it was raised in pain or despair.

This was by no means a typical case of death by drowning, but it is well known that in such deaths fatal syncope is accelerated by the presence of water in the lungs, which mechanically embarrasses the circulation through the lungs, and consequently through the heart. Probably the quantity of water which the deceased inhaled did not at first seriously affect the heart's action; but when he was put on his back in a horizontal position the mechanical effect of the water in the chest was fatal, for to all intents and purposes it was then that he died. I believe that the cause of death was syncope, brought about by neglect of the well-known necessary initial precaution of immediately draining the fluid from the chest. There was, at any rate, not sufficient evidence, from a medical point of view, that the deceased was the victim of any cardiac or other seizure, except that brought about by the result of the immersion. I could not, therefore, support the attempt to resist the defendant's claim.

The case came for arbitration before a County Court Judge in the country, and I was not called. Judgment was given against the employers.

**Typhoid Contracted on or off Duty—History.**—F. Y., a seaman, died of typhoid fever on December 25, and, having regard to the facts detailed, I was asked to advise as to the probability of the disease having been contracted on the ship or on land.

The vessel left London on November 2, and arrived at Montevideo on November 23. The next day, the 24th, it was at Buenos Ayres, and remained there until December 4. The ship then proceeded to La Plata, and was there from December 5 to 9. On December 13 the fourth engineer showed symptoms of typhoid fever, on the 14th another seaman was attacked by the same disease, and on the 17th F. Y. was taken ill. These were the only cases which occurred, but an analysis of a sample of the ship's water made by the Board of Trade disclosed that it was an exceedingly bad one, and indicated probable contamination with sewage.

The incubation period of typhoid fever ranges from four to twenty-eight days, the usual period between infection and the first display of symptoms being about fourteen days. The minimum period of incubation would have admitted of all the cases being contracted on board after leaving La Plata, and the maximum would have put it before the arrival at Montevideo, but the probable period of contraction in all the cases was from about December 1 to 3, when the vessel was at Buenos Ayres.

In F. Y.'s case, he probably received the poison on either the day before he left Buenos Ayres or on the day he sailed to La Plata.

Typhoid is of course a water-borne disease, and, although very

prevalent in Buenos Ayres, it seemed to me impossible, having regard to the dates, to prove that the water (if the man drank it) did not cause the disease, having regard to its more than doubtful quality. It appeared to me that the most that could be said in favour of the man having become infected on land was that during the incubation period the patient may experience no inconvenience of any kind, and therefore it was quite possible for the man to perform his duties until the 17th, although having contracted the disease on land. To allow of the possibility of his having become infected from the fourth engineer, the minimum period of incubation must have operated.

In the absence of any evidence to the effect that the water was not partaken of by the deceased, I did not find any reason to lead me to the conclusion that any cause had operated in producing the disease other than the contaminated water supplied by the ship.

I was not called at the arbitration proceedings, and whether evidence beyond that supplied to me was laid before the Court I know not, but in the result an award was made in favour of the employers.



## CHAPTER XXXII

### EXAMINATION BY X-RAY PHOTOGRAPHY

**Radiographic Examination.**—A skiagram is not a photograph in the ordinary sense; it is a reproduction of a series of shadow-pictures which depend for their production upon the varying densities of the structure penetrated by the X-rays. The shadows cast, by crossing or overlapping one another, may destroy the characteristics of each. The view presented is the same as if the whole subject were on one plane; therefore form, size, distance, and position, have all to be reasoned out.

When the plate is printed, the shadows are always reversed. The bone shadows, which are white or transparent upon the plate, are black or dense upon the print. The denser the tissue, the less does it permit the X-rays to pass through it, and fewer rays reach the plate to take effect on the silver emulsion. Those parts of the plate that have no tissue over them show the full effects of the ray on the emulsion, and consequently appear very black and dense. Radiograms do not as a rule show injury to soft tissue, but with a good quality of plate an extensive rupture would be shown in a muscle or in the thicker ligaments, such as the ligamentum patellæ or tendo Achillis.

In investigating cases where injury or disease of the bones is suspected, the wisdom of having an X-ray photograph taken is becoming more and more appreciated. We have thus at our disposal a more or less certain means of proving or disproving the existence of an organic basis for the symptoms complained of. It is advisable that there should be such an examination in all cases where there is the slightest prospect of its affording any assistance.

**Importance in Court Proceedings.**—It should be an invariable rule to have all bone or joint injuries photographed by X-rays before going into Court; for whether the defendant takes this precaution or not, the plaintiff probably will.

C. U. had her thigh broken as the result of gross carelessness. Ten months later I was asked by the defendant's solicitor to examine the case in consultation with their medical man, and to report whether I could support him in the suggestion that the incapacity was being unnecessarily prolonged. In my report I was able, however, to point out, without the help of the X-rays, that there was an ununited fracture, and furthermore that the plaintiff's solicitor had very wisely obtained an X-ray photograph of it a few days before! It amused me to discover that, by the instructions of the plaintiff's solicitor, a portable apparatus had been taken to a house some distance from London, and the photograph taken with the utmost secrecy. As my report advised that from a medical point of view we had no case, the defendants, under the circumstances, were naturally glad to settle; and the rod in pickle (over which, no doubt, there had been much gloating) was never presented to me in the witness-box.

C. V., a dock labourer, sustained an accident owing to a large piece of chalk (weighing over one hundredweight) falling on his *legs*. He had no bones broken, but sustained injuries from which the first medical man who examined him reported he might be expected to recover within a few months. He continued to complain of pain and inability to use his left leg, and was examined at intervals of every few months by numerous surgeons, who found wasting and shortening of the left thigh. Sixteen months after the accident payment was stopped on the advice of a medical man who saw him on behalf of a company. Arbitration proceedings were commenced. It was claimed for the plaintiff that the condition of his leg was due either to a local neuritis or to injury of the head or spine as a result of the accident. The defence, I believe, relied on the contention that the condition was congenital. The County Court Judge decided in the man's favour, and ordered the weekly allowance to continue.

Some months later he was sent to me for examination. There was considerable wasting and some shortening of the left thigh bone. A radiogram, taken at the suggestion of an eminent surgeon who saw the case with me, showed clearly that the whole condition was due to arthritis deformans of the hip-joint, probably of some years' duration. X-ray examinations showed the presence of the same conditions, but to a less degree, in the other hip and in both knee-joints. He was, in fact, the subject of constitutional articular disease, as shown by radiograms of four different joints of his body. The appearance in each case pointed to the conclusion that the disease could not have existed for less than two years; but the pain at the left hip-joint, the shortening of the left leg, and the wasting of the muscles of the left thigh, all of which were proved to be present as early as the first arbitration proceedings, indicated the commencement of arthritic



hip-joint disease long prior to the date of the accident. There was no evidence that the accident even aggravated this condition; therefore, so far as the accident was concerned, no incapacity for work remained.

Payment was by my advice stopped, and I suggested that if arbitration proceedings were again taken, a totally different defence should be set up from that presented at the first proceedings. A medical assessor sat with the Judge. I produced the X-ray photographs, and proved that the man's disabilities resulted from old-standing hip-joint disease, which had neither been produced nor accelerated by his accident. The County Court Judge decided in the company's favour, and discontinued the man's allowance.

In view of the fact that radiograms are useless, and are very apt to mislead, unless skilfully taken, it is obviously prudent always to employ a competent radiographer. The radiologist with a small apparatus should realize his limitations.

X-ray examinations should always be thoroughly conducted. For example, the use of the fluorescent screen alone may lead to most inaccurate conclusions; and it is possible, if a screen only is used, for even a skilled observer to overlook such an obvious injury as a transverse fracture of the shaft of the tibia. The screen examination should, therefore, never be solely relied upon, but in all cases the radiographic method should be employed. By this means accurate recognition of injury or disease of bone is assured; moreover—and this is very important in medico-legal cases—a permanent record is obtained.

**Sources of Error.**—Radiograms are often found to be valueless for diagnostic purposes owing to the fact that sufficient attention has not been paid to the relation between the source of the X-ray and the part which is to be examined. Striking distortions may be the result of such an omission. In dealing with bone injuries, it is always advisable to have *two* views of the same limb, one secured in a direction at right angles to the other; for when radiographed in one direction only, a fracture may be invisible; further, an accurate estimation of the amount of displacement of the fragments in a case of fracture can in this way only be obtained. This cannot be done in the shoulder, hip, etc.; the difficulty, however, may be overcome by obtaining a stereoscopic view, but, unfortunately,

this cannot be conveniently demonstrated in Court. The illustrations (Figs. 51 and 52) represent the same injury—namely, fracture of the left fibula. In Fig. 51, which is a postero-anterior view, there is no evidence of fracture, the broken ends of the fibula being in perfect apposition. In Fig. 52, the lateral view, an undoubted fracture, with a certain amount of displacement of the fragments, is plainly seen in the position marked  $\leftarrow$ .

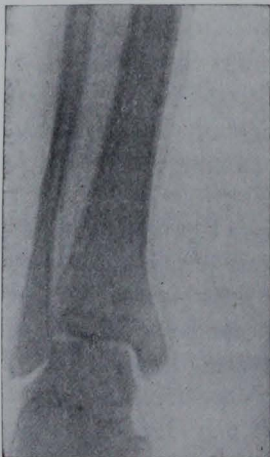


FIG. 51.



FIG. 52.

(From Ironside Bruce's "X-Ray Examination" in Choyce's "System of Surgery.")

Until recently the only guide to the best position for the X-ray tube has been the observer's experience, and the method usually followed has been to place the tube in some indeterminate relation to the part. Now, however, there is a disposition to adjust the tube definitely and accurately. When this is done, the advantage gained is obvious. Dr. Ironside Bruce has produced a work in the form of an atlas, which presents many advantages, for it contains radiograms of the normal at different ages (five, fifteen, and twenty-five), each secured with the tube in a definite anatomical relationship. Thus a certain knowledge of normal radiographic appearances is afforded, and enables the radiologist to secure a radiogram of any part of the body in a given case identical with that to be found in



the atlas.\* The position in which the subject should be placed has been made clear by photographs, and the relation of the tube to some familiar and accessible anatomical point has been defined in each instance.

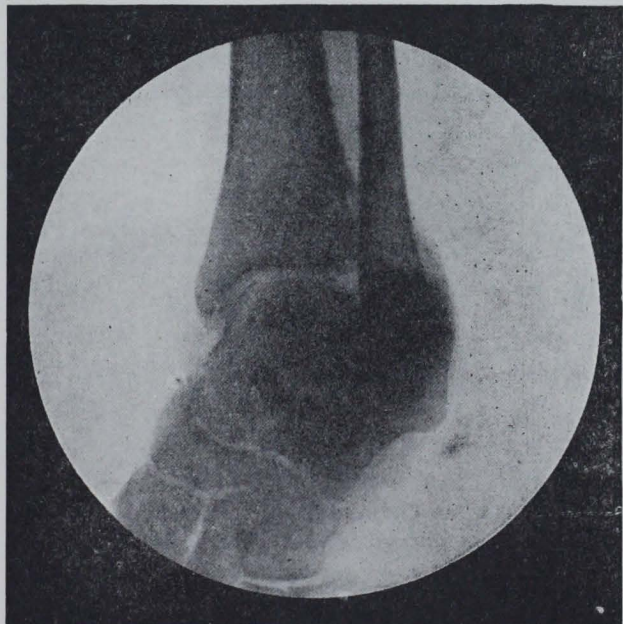


FIG. 53.—RADIOGRAM OF NORMAL FOOT, BADLY CHOSEN FOCUS-POINT AND UNSUITABLE POSITION OF LIMB.

I have permission from Dr. McKendrick, Surgeon in charge, Radiographic Department, Royal Infirmary, Edinburgh, to reprint the following paragraphs from his "Radiography of Normal Parts." I am grateful for this opportunity, for it sets out with remarkable clearness the dangers of amateur or dishonest radiography.

"It is well known that an ordinary photograph of any familiar object may be exceedingly difficult to interpret, if an unusual point of view has been chosen by the photographer."

"Some time ago a series of such photographs was printed

\* "A System of Radiography," Ironside Bruce. London: H. K. Lewis, 1907.

“ by the publishers of a well-known magazine, and valuable prizes offered by them to anyone who interpreted the greatest number correctly. In this case the point of view, or, as we might call it, the focus-point, was the only factor causing difficulty in the correct interpretation.

“ In radiography we have to deal, not only with suitable focus-points, but with the position of the limb or joint, as the case may be, at the time of making the exposure.

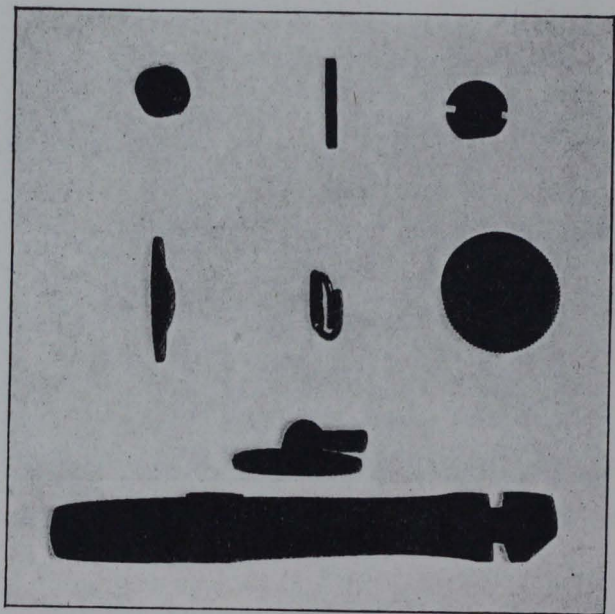


FIG. 54.—RADIOGRAM OF OBJECTS WITH X-RAY TUBE DIRECTLY OVER CENTRE OF PLATE.

“ A badly chosen focus-point, in conjunction with an unsuitable position of the limb, may result in a radiograph analogous to the puzzle pictures mentioned above. This gives rise to what is called “distortion” in the radiograph. . . . The shadow, although it may appear unrecognizable, is not actually distorted, but only apparently so. See Fig. 53 of a normal foot taken from an unsuitable focus-point. Anyone might be excused for expressing the opinion that the ankle had sustained a severe dislocation, or that the case was one of a badly united Pott’s fracture. This picture



“ shows how far a surgeon might be misled by a radiograph  
 “ through his not knowing the conditions under which the  
 “ exposure was made. Imagine the case of a malingerer  
 “ who alleged injury to his foot. Several surgeons might  
 “ examine the foot, and be able to swear in Court that the foot  
 “ was absolutely normal. An X-ray picture similar to Fig. 53  
 “ might be taken of his normal foot, and the discomfiture  
 “ of the various surgeons who pronounced the foot normal  
 “ can easily be imagined, if such a radiograph were put into  
 “ their hands in the witness-box after their evidence had been  
 “ given. . . .

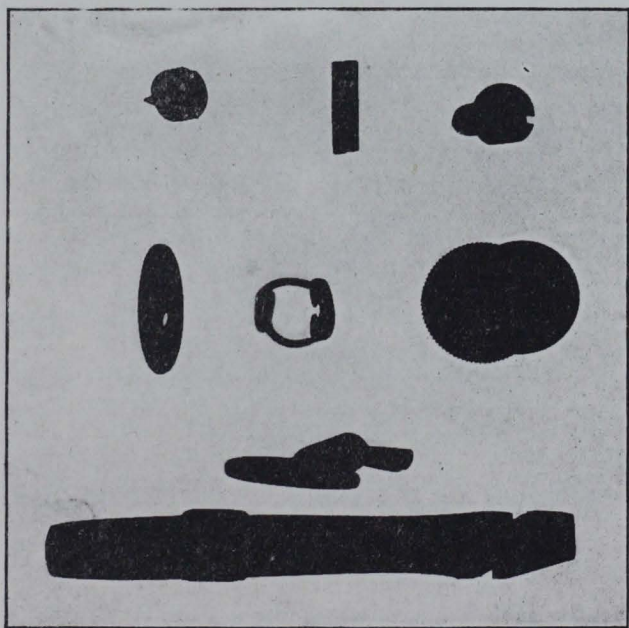


FIG. 55.—RADIOGRAM OF SAME OBJECTS AS IN FIG. 54, WITH X-RAY TUBE  
 FOUR INCHES TO ONE SIDE.

“ Fig. 54 is a puzzle radiograph, similar to the above-  
 “ mentioned magazine puzzle pictures. In it, eight familiar  
 “ objects have been placed upon an X-ray plate, and an ex-  
 “ posure made with the tube 24 inches directly over the centre  
 “ of the plate.

“ An expert radiographer will have difficulty in recognizing  
 “ even one of these objects.

“ In Fig. 55 all the original relations have been retained, but  
“ the tube has been displaced 4 inches to one side. Any-  
“ one accustomed to the examination of radiographs will now  
“ be able to recognize at least two of the objects—viz., the  
“ button and the safety-pin.

“ In Fig. 56 the same objects have been placed in such posi-  
“ tions that each of them can be easily recognized.

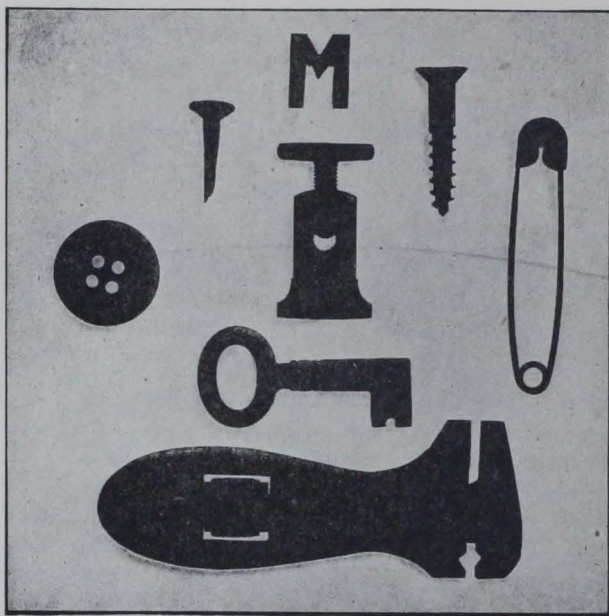


FIG. 56.—RADIOGRAM OF FIG. 54, IN A DIFFERENT POSITION.

“ If now the tube be moved 4 inches to one side, the  
“ objects cast almost exactly similar shadows (as in Fig. 57)  
“ to those cast in Fig. 56.

“ We learn from this that radiographs of any object which is  
“ lying with its long axis in a plane parallel to that of the plate  
“ will be apparently little altered in shape if a different focus-  
“ point is chosen. If, on the other hand, an object such as the  
“ neck of the femur, which is not usually lying in a plane  
“ parallel to that of the plate, is being radiographed, an altera-  
“ tion in the focus-point will make a marked difference in the  
“ shadow cast. The radiographer should be able to place



“the limb in the best position for attaining the best relationship of the part to be examined to the plane of the plate.”

**Radiography not Infallible.**—Even X-ray photographs carefully taken in planes at right angles to each other do not in certain rare cases absolutely exclude the possibility of fracture, as the following case related to me by my friend Major McKechnie, of the Indian Medical Service, exemplifies:

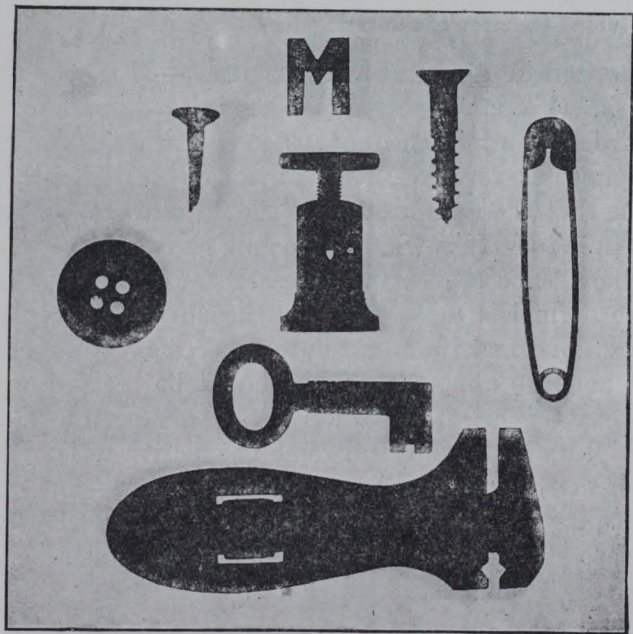


FIG. 57.—RADIOGRAM OF SAME OBJECTS IN SAME POSITION AS IN FIG. 56. BUT WITH X-RAY TUBE FOUR INCHES TO ONE SIDE.

A soldier had a gunshot wound of the right forearm which had healed. He was very emphatic that he could not use his arm, and insisted on having it in a sling. The forearm was helpless, and yet there was no obvious cause. Later a swelling developed at the site of the injury. X-ray photographs taken in planes at right angles to one another failed to show any injury to the bone. When the swelling developed it was cut down upon, as in view of the X-ray examination it was assumed that a septic condition must have developed, possibly owing to the retention of some septic foreign material in the wound. The operation showed that the swelling was caused by muscular spasm, and exposure of the ulna in its middle showed that there was a transverse fracture of that bone. It was cracked without any displacement in any direction. There was no callus.

**The Value of Stereoscopic Radiographs.**—A soldier said his foot had been crushed by a waggon. After somewhat prolonged treatment in hospital, he was discharged to a convalescent dépôt for the purpose of being drafted to the front. An X-ray had been taken, and the diagnosis of no injury was made. He, however, still complained bitterly of pain on walking, and held his foot in the position of *talipes equinus*.

As he persisted in saying he could not do his drill, he was sent by the camp commandant to be X-rayed by another radiographer. A stereoscopic radiograph revealed the fact that he had an impacted fracture of the neck of the astragalus.

**Comparison of Abnormal with Normal.**—If a patient complains that his knee-joint has been injured, and on X-ray examination the presence of osteo-arthritis in the alleged injured knee is revealed, it is obviously of the utmost importance that the *uninjured* knee should *also* be so examined, for not infrequently it will show equal, or it may be still more marked, evidence of the same disease.

If the normal is to be used for the purpose of comparison with the abnormal, it is absolutely necessary that both the normal and the abnormal views should be secured with the tube in exactly the same relation to the part examined. There often appears in a radiogram some peculiarity which can only be explained by comparison with the normal—for example, when dealing with a case of injury to bones in which the epiphyses are still separate, and in which one cannot be certain whether there is displacement or not.

**Interpretation by Experts.**—It should be remembered that it is one thing to look at a radiogram, and quite another thing to be able to interpret it correctly, and it is amusing to see skiagrams solemnly handed round to a jury, who examine them with much anxiety and apparent intelligence. Radiograms should always be interpreted to the Judge and jury by those accustomed to dealing with them.

An injustice is likely to be done if those unaccustomed to taking or reading skiagrams essay to give expert advice, for dangers lurk at every corner. Fractures and abnormal conditions are not infrequently pointed out which do not in fact exist. Inexperienced radiologists often detect these where they are too often the result of defective technique.

No radiogram should be produced in Court unless it is the



work of a competent medical radiologist; yet hundreds of pounds are often obtained from defendants by means of untrustworthy radiograms taken by amateurs or unqualified persons. It is a good plan, when an opponent intends to make use of a radiogram taken, let us say, by a local chemist, to subpoena that gentleman, and to prompt counsel with a few conundrums to be solved by the witness when in the box. Radiograms are as easily faked as are the trick representations of a cinematograph show.

Radiograms are specially useful in cases of fracture, for they show whether or not good union has taken place, and how far the bones have returned to their proper position. In cases of osteo-arthritis the radiogram often shows the presence of bony abnormalities.

Radiograms cannot, of course, demonstrate muscular injuries, but when the attachment of a muscle to the bone has been torn away and it carries with it some portions of periosteum or bone, the outline of the bone is irregular and wavy.

Dr. McKendrick has kindly supplied me with the following note on a common source of error in the interpretation of radiograms.

**Adventitious Shadows.**—There are several pitfalls arising from accidental shadows which the examiner must always be on his guard against. Calcareous glands or hardened faecal masses, if these happen to lie near to the spine, may be diagnosed as traumatic myositis ossificans. Examinations at different dates and under altered technique will elucidate the nature of such shadows.

Dressings are often a cause of accidental shadows upon an X-ray plate. Even when the superficial dressing has been removed, if the depth of the wound has been dressed with iodoform or bismuth, a fairly dense and misleading shadow will be cast.

Phleboliths, which cast a fairly dense shadow, are common in the pelvic veins.

Where the shadow of a muscle is superimposed upon that of a bone, a fracture is often closely simulated at the point where the edge of the muscle crosses the bone.

**Identification of Negative in Court Cases.**—It is an advantage to be present when the radiogram is being developed, for an opportunity is thus afforded of viewing the plate whilst it is still wet, it being then at its best for this purpose, as the subsequent drying process causes some loss of detail. Prints often fail to reveal changes which are manifest in the plate, and their use for demonstrating purposes is to be deprecated. Yet how often are they produced for the Judge and jury to inspect, and erroneous statements made, and damaging opinions formed, which too frequently end in a miscarriage of justice.

I make it a rule to be present when the radiograph is taken, to follow the plate into the dark-room, initial it, and wait until it is developed. Under these circumstances, no mistake can be made, such as the wrong plate being produced, and one is able in the witness-box to identify the skiagram. There is also the additional advantage that during the time the plate is wet, after it has been removed from the fixing-bath and washed, infinitesimal changes can there and then be discussed with the radiographer. Plates should always be labelled L. or R. when dealing either with a limb or with the trunk. Small leadfoil letters, R. or L., should be stuck by adhesive plaster on the plate cover previous to exposure.

**Deviations from the Normal.**—I am indebted to Dr. Gilbert Scott for the following hints. He points out that a large number of variations from the normal constantly occur, that they are congenital in origin and are not indicative of disease, and that this can often be proved when examining a limb by showing that a similar condition exists in the sound one.

Amongst the many deviations found, he mentions the following:

**Injuries of the Spine.**—An antero-posterior and lateral view should always be taken. This is not sufficiently appreciated. No satisfactory lateral view can be obtained of the upper dorsal region on account of the shoulders, but a lateral view of the rest of the vertebral column, including the sacrum and coccyx, can be obtained.

**The Laminæ.**—Those of the fifth lumbar vertebra are very frequently found to be incompletely united in the middle line,



with the result that there is a gap on the plate between the two ends of the laminae; or they may both reach the middle line, and as they have not united they appear on different levels. This condition, which is a form of incomplete spina bifida, is a congenital defect, and may easily be mistaken by the inexperienced for evidence of old fracture.

**Transverse Processes.**—Fracture of these, the result of direct or indirect violence, often passes undetected unless radiographed. The condition is more common than is generally thought. It should never be forgotten that the last rib varies greatly in length. Sometimes it is so small that it may be readily mistaken for a fractured transverse process of the twelfth dorsal vertebra; for it has the appearance of nothing more than a small bone element in close relation to, but separated from, the transverse process of the vertebra. This rib, the twelfth, may be congenitally crooked or twisted. Any transverse process may be developed from a separate centre, and fail to unite with the body of its vertebra. A gap appears in the radiograph, and this congenital defect is apt to be wrongly diagnosed as fracture.

**Sacrum.**—Gas in the intestine, being more transparent than the surrounding tissues, throws a shadow of varying density, which, if it happens to lie in certain positions, implants on the radiographic plate an apparent defect in the bone shadow, which has the appearance of the results of injury or disease.

**The Coccyx.**—This bone is difficult to radiograph satisfactorily, owing to the density of the various structures lying in front of it. To detect dislocation, it is essential to obtain both an antero-posterior and lateral view. The arrangement of the individual segments of this bone vary within wide normal limits, both antero-posteriorly and laterally.

**The Sacro-Iliac Synchrondrosis.**—This often shows a puzzling shadow, and the two sides may vary considerably within normal limits.

**The Knee-Joint.**—Care should be taken not to mistake sesamoid bones for fragments of bone chipped off the tibia, fibula, or femur. Sesamoids are usually situated posterior to the joint, and are generally flattened on the under surface.

Thickened synovial fringes eventually become calcareous, and are then opaque to the X-rays. These so-called "loose" bodies frequently occur in osteo-arthritis, and are sometimes mistaken for fragments of bone the result of injury.

**The Ankle.**—The extreme tip of either malleolus may, apart from the epiphysis, develop from a separate bone centre of ossification. It usually remains separated from the shaft during life, with the result that the radiographic appearances are strongly suggestive of bone injury.

**The Foot.**—An addition to the normal number of the small bones of the foot is common. These are generally found posterior to the astragalus, and the tuberosity of the scaphoid. In extreme cases of long-standing flat-foot, the scaphoid may become much deformed and irregular, giving an appearance which is suggestive of injury by crushing. Normally there are two large sesamoid bones under the big toe; in a large number of cases one or other appears in either two or three pieces, which is merely the result of unusual ossification.

**The Phalanges.**—These in a normal condition are often very irregular in outline, and this should not be attributed to injury.

**The Subdeltoid Bursa.**—Inflammatory deposits the result of inflammation of the bursa, when of long standing, become opaque to the X-rays, with the result that it is often difficult to say whether they are merely ossified inflammatory deposits or are fragments torn off the bone.

The extreme tip of the condyles of the humerus, the styloid process of the ulna, and rarely that of the radius, are sometimes developed separately from the shaft, and remain so during life.

Finally, Charcot's joints are sometimes first discovered by X-rays.

**Apparatus for Demonstration in Court.**—I have, by modifying an existing apparatus, produced what I hope may prove a useful instrument for the purpose of the inspection of radiograms in Court (Figs. 58 and 59). It consists of a small triangular-shaped metal box, the base of which is some 10 by 9 inches, with two sloping opal glass sides. On one side, in front of the opal glass, the negative of the radiogram of the part

in question is fixed, and on the other the negative of the corresponding part of the same person, or, if this happens to be abnormal, a skiagram of the same part of a normal subject. Inside the box are placed four small incandescent electric lamps of eight candle-power each, the current being supplied by a 60-ampère H.R. 8-volt accumulator, which is carried separately from the instrument. This affords a brilliant light, and shows

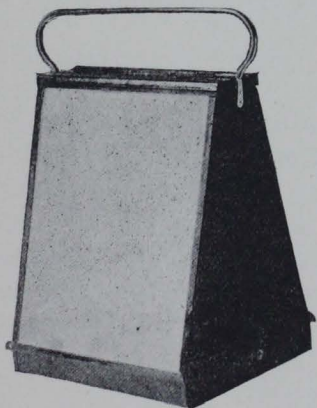


FIG. 58.

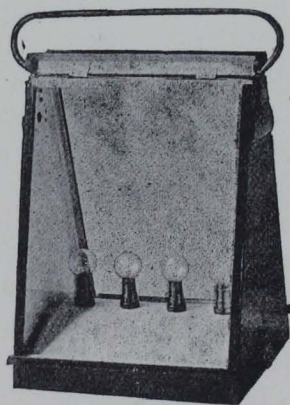


FIG. 59.

up every detail in a very clear way. The apparatus is a little heavy, but portable, and is handed by the usher to the Judge and jury. Small slips of paper, with arrows and a word or two of description, can be pasted on the plate; this helps to elucidate any point which is being made.

It is a good plan to offer to explain personally to the tribunal these details, and actually point out any abnormalities that may exist.



## CHAPTER XXXIII

### THE USE OF ELECTRICITY IN ALLEGED MALINGERING

THE utility of the electric battery in assisting to unmask the malingerer will have been noticed in many of the illustrations which have been given. A battery producing the faradic current only is all that is required, and this may be obtained at very little cost. It should have a break key in one electrode; thus it will be possible to cut off completely the current from the patient without his knowing what has happened (whilst the coil is buzzing loudly), by simply depressing a small knob with the thumb (see Fig. 60).

**Method of Using.**—The indifferent electrode—that is, the one which is simply used to complete the circuit—may be of any shape or size.

Before actually proceeding to use the test, the battery should first be tried on oneself to ascertain what is the exact strength of the current about to be used. Moisten the skin of the patient over the area to be tested; the water should be warm and freely used, both on electrodes and skin. It will need frequent renewal during the examination. No salt need be added; a weak current is all that is necessary.

When a patient is malingering he may simulate either increased sensibility or loss of power.

**Alleged Increased Sensibility.**—If a man under examination complains of pronounced tenderness at a particular spot, it is well known how difficult it is to deny or affirm the reality of such a purely subjective symptom.

A weak current of electricity would slightly increase pain or sensitiveness if genuine. The malingerer often complains that his alleged hypersensitive area is rendered much more

painful by the electric current. The truth or falsity of this statement may be demonstrated by the following manœuvre:

A noisy faradic battery is set in motion, and the indifferent electrode applied to the body at some distance from the alleged painful spot; the other handle, in which is the break key, is gradually, from some distance, approached to the painful spot. The break key is depressed and the current entirely cut off,

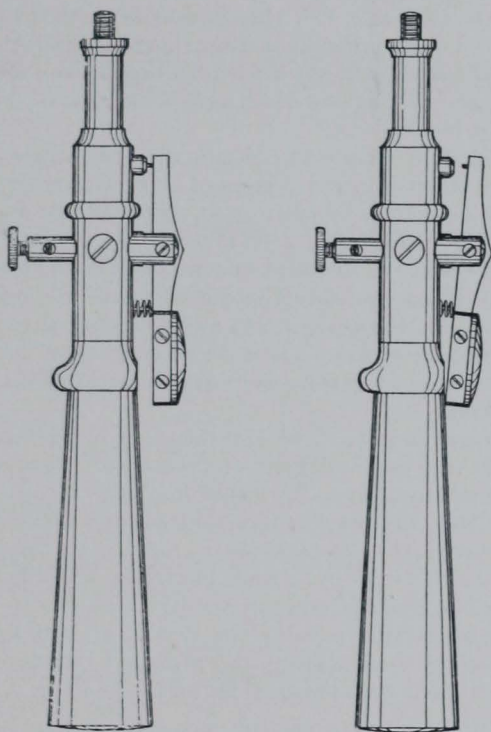


FIG. 60.—TESTING ELECTRODE HANDLE WITH BREAK KEY.

although the battery is still left in noisy action. The patient is asked to state definitely whether the current is felt to a greater or less degree as the handle approaches the painful area.

In a surprising number of cases ignorant but determined malingerers, hearing the battery still in action, and assuming that the current *must* therefore reach them, have told me that the pain produced over the alleged painful spot was so intense that it amounted to “agony.” The pain is sometimes

the malingerer; it is not an uncommon thing to find a man who has really recovered from nystagmus producing a rapid spasm of the eyelids during inspection by the medical examiner. The deception can, however, be readily detected; for if the patient's attention is diverted, it immediately ceases, which is not the case in genuine nystagmus. The feigned twitching of the eyelids cannot be kept up for more than a few minutes, and those accustomed to the condition will be able at once to detect the feigned nature of the attack. The genuine movement is quicker and finer than the malingerer is able to produce, who blinks his eyes and often brings other muscles into play.

At first sight miner's nystagmus does not appear to be a disease that could be easily simulated, and this is true so far as the main objective symptom, oscillation, is concerned. A few individuals can at will produce a nystagmus, but these are exceptional cases. The type of nystagmus produced when an attempt is being made at simulation is generally a lateral one, and differs from the rotatory form which is so common in true miner's disease.

Although oscillation of the eyeballs can scarcely be simulated, the symptoms which accompany it can, and often are.

It should not be forgotten that, although it may be difficult to elicit oscillation at one time, it is often comparatively easy to do so at another. Therefore cases alleged to be examples of miner's nystagmus, which do not, on repeated and thorough examination, show any oscillation of the eyeballs, must be viewed with suspicion.

In doubtful cases the following methods, suggested by Llewellyn in his book on Miner's Nystagmus, are useful:

Genuine oscillatory movements may be elicited by getting the patient either to raise his eyes gradually towards the ceiling or to converge them.

Perhaps the best way is to ask the patient to fix a small body like a pencil, held a foot in front of and on a level with his eyes. If then there is no oscillation, the object should be gradually elevated, and the patient asked, not to move his head, but to follow it only with the eyes.

If this fails, he should be asked to stoop to the ground two or three times in succession, when it will often be manifested,



occasions the current is cut off prior to reaching this spot, it may be (or at any rate the patient's counsel may claim) that the first application of the current acted as a mental suggestion, and the subsequent complaints were the result of the powerful stimulus given to the mind by the initial application of the current to the alleged painful area. When no current reaches the spot prior to the complaints of knife-like sensations, etc., this objection cannot be raised, especially if, as indicated above, the experiment is repeated several times.

I have found this test very valuable in demonstrating a malingerer to his own doctor—a procedure, however, which is not always wise. It should never be given away in the case of a doctor attached to a running-down solicitor.

Occasionally—very occasionally, when by intuition I feel that the circumstances are exceptional, and that I can effect a *coup d'état*—I tell the patient. I do not, however, recommend this, for the result of the experiment tells with more effect in the witness-box, especially if elicited in the course of cross-examination.

*History.*—C. X., as the result of a collision at sea some eight months previously, fell on an iron railing, injuring his back. At the time of my examination he complained of pain in his back and chest.

*Examination.*—Whilst waiting his turn for examination he pretended he was so feeble that he could not stand even for a few minutes. During the examination, except when called to order, he puffed and blew as if he were short of breath.

With regard to his chest he indicated no special area as being painful, but complained of the whole of the front of his chest. The result of the examination, however, revealed nothing abnormal as the result of the accident.

With regard to his back, he complained of pain at the fourth lumbar spinous process. Suspecting him, and in order to test him, I applied the faradic current from an electric battery to his back, and, whilst the coil was still buzzing loudly, switched off the current as the pole of the battery reached the alleged painful area. On two separate occasions as the electrode approached its vicinity he complained bitterly. He yelled so loudly, and spoke so boisterously, that I had to insist upon his controlling himself; indeed a patient who was waiting in a room some distance from my consulting-room heard him yelling. After repeating the experiment, I explained to him what had actually taken place. He evidently appreciated the mistake he had made, for upon applying the current once more he made no complaint, and the accuracy of his answers on this last occasion *showed the dishonesty of his previous replies.*

In order to satisfy myself with regard to the alleged tenderness in the chest I used a stethoscope over the whole of the front of his chest. Believing that I was merely examining his chest in the ordinary way, he made no complaint, though I took occasion to press with very great firmness.

It was, therefore, perfectly obvious that C. X. was a very bad actor, that he was not suffering as the result of his accident, and I advised his employers accordingly.

On receipt of my certificate his weekly allowance was stopped, and he made no protest.

The experiment sometimes fails. It seems almost incredible, but recently, on two separate occasions, I satisfied myself that claimants had actually been coached in the details above described so as to thwart the discovery of an imposture. But even so, it affords a convenient way of solving the very difficult question as to whether a patient really suffers the pain he alleges when pressure is applied on a particular spot.

Assuming the patient has been coached in the test above described and it does not have the desired result, the following procedure should be adopted: His eyes are covered, or he is asked to look well to one side; he is then directed to say definitely and at once when he feels the electric current, which, it is explained, is to be switched on and off at intervals as the electrode is moved over and in the neighbourhood of the painful area. It is surprising how often very firm pressure by the electrode will be borne without the slightest complaint, while the patient's attention is taken up in attempting to decide whether a weak current of electricity is or is not flowing on a certain area which he has, either consciously or unconsciously, taught himself to believe is sensitive to the slightest touch.

**Alleged Loss of Power.**—If complaint be made of loss of power, the test should be carried out as follows: Start the current. Place the testing electrode (in which there is the break key) upon the muscles in question with the thumb depressed (for motor points see Chapter XXXIV.). The patient is, of course, receiving no current. Now sharply raise the thumb; if the muscles are normal, a contraction will immediately result. The patient, of course, does not at the precise moment expect the sudden stimulus, and has no time voluntarily to inhibit it. Moreover, if the current is fairly strong, he cannot prevent the muscle contracting; besides, if he attempts



to do so, the *opposing* muscles will be seen to enlarge. Assuming there has been a normal response to the electrical stimulation, *any loss of power cannot be due to a local lesion*, for any physiological interruption would (assuming the injury were not less than two days old) have resulted in diminution, if not in entire absence, of response to the faradic current.

*History*.—C. Y.—I was asked by an insurance company to examine, in consultation with a doctor who was familiar with the case, a man who was said to have been suffering from lead poisoning for fourteen months. An opinion as to his fitness for work was required.

*Examination*.—He complained of weakness, inability to do anything, accompanied by complete loss of power in the whole of the upper right arm.

When asked to move the arm, elbow, and hand, he declined even to try, and when the arm was taken up it flopped down as if it did not belong to him. Giving him to understand that I was satisfied with regard to the paralyzed condition of his arm, I proceeded to examine his lungs, and lifted up his arm at right angles to his chest, to examine the side of his chest. He continued to hold it up himself! He was next asked to bend both elbows, and upon my attempting to straighten the arm alleged to be paralyzed, he prevented my doing so—presumably because he thought he must oppose everything.

Absence of muscular wasting negated the presence of local disease. In testing his sensation he was directed to look to one side, was lightly pricked alternately on the right and left arms, and asked to say "No" when he did not feel it and "Yes" when he did. When the right side was touched he said "Yes," and when the left side was touched he said "No," thus giving himself entirely away.

I now resumed the examination of the right arm. As is well known, in genuine lead poisoning the nerves which supply certain muscles become so diseased that nervous impulses cannot be sent to them from the brain, and, similarly, nervous impulses cannot be made to travel along the nerves even by means of a faradic current. The current was therefore applied, and the freest contraction of the muscles was obtained under the stimulus of the faradic current, showing that if lead palsy had ever been present, he had now wholly recovered from it; indeed, when the current was applied with some strength, he drew his arm away so violently that a strong steel rod belonging to the instrument was bent.

The date of the onset of his alleged paralysis was inconsistent with the development of true lead palsy. Notwithstanding the fact that a few days before he had complained of paralysis of the right leg, when he was instructed to strip and walk round the room, feeling no doubt that he would have an inappreciative audience, he did so without lameness. There was no doubt he was clumsily acting.

He was living in a small room, the temperature of which could not have been less than seventy when we called; it contained seven



people, including a sympathetic wife who obviously believed in him. There was nothing amiss with him except the mental deterioration of prolonged idleness and introspection, and the physical disabilities produced by an environment in which it was impossible to maintain health.

*Result.*—Compensation was stopped, and he took proceedings under the Workmen's Compensation Act. At the hearing I gave evidence as above, and judgment was given for the defendants.

If the response to electric stimulation is doubtful, compare the effect of the same strength of current on the muscles of the opposite side of the body—that is, convert them into a standard. It should be remembered that a response does not preclude injury to the central nervous system, nor does it exclude functional disease—*e.g.*, hysteria.

If no reaction can be obtained with the faradic current, a further examination should be made with the galvanic current, and the muscles thoroughly and carefully tested for the reaction of degeneration.

The condition of a patient who emerges from this test successfully requires further investigation. If he has a definite nervous lesion, the diagnosis and prognosis may be matters of some considerable difficulty, and it has not been found possible within the limits of this book to discuss them fully. A short account of electric testing will be found in the next chapter.

## CHAPTER XXXIV

### ELECTRICAL TESTING

A DESCRIPTION will be found in Chapter XXXIII. of the use to which electrical reactions may be put in the detection of malingering.

At the end of this chapter a short description of the apparatus is given for those who are not familiar with its use.

It is proposed here to give a brief description of the method of electrical testing adopted when a patient is thought to be suffering from some definite nervous affection.

It must be clearly understood that it has not been possible within the limits of this work to do more than indicate the general principles which guide us in this branch of the subject. Sufficient has, however, been included to enable a practitioner who follows the directions to detect the reaction of degeneration, and in a straightforward case to localize the lesion.

#### **Method of Electrical Testing.**

**Test with the Faradic Battery.**—Start the current in the primary coil, and see that the rate of interruption is satisfactory.

Join up the electrodes by wires to the secondary coil, the large or indifferent one to be placed at some distant part of the body, and the other, with a make key in its handle, to be used as the testing electrode.

Have a bowl of warm water at hand, and freely moisten the electrodes themselves, and the skin of the patient over the muscles to be tested, and also, if possible, over the corresponding muscles on the other side of the body. The skin and electrodes must be kept moist throughout the experiment.

**The Reaction of Degeneration.**—The test for this condition is first by means of the faradic, and, later, if necessary, by the galvanic current. First test the strength of the faradic battery.

Test with a weak current the corresponding muscles on the sound side of the body. If these respond by a moderate but quite definite contraction, test with the same strength of current the muscles under examination. If they respond as briskly as those on the other side, there is no *reaction of degeneration* (R.D.). In such cases it is not necessary to proceed further with electrical tests.\*

\* When a motor impulse is sent from the brain to a voluntary muscle it passes through two neurons—*i.e.*, two motor cells and their main processes. One cell is situated in the cortex of the brain, and the impulse passes through it and its main branch (the axon), which ends in a fine meshwork of fibrils round the other nerve cell situated in the spinal cord. The impulse passes across from the meshwork to the spinal motor cell, and through its axon, to end in a specialized organ in the substance of the muscle itself—the muscle spindle.

If a lesion occurs to the former of these neurons, the cell or its axon, the muscle will not receive the impulse. It will therefore be paralyzed, but its nutrition *will not* suffer. It will therefore not waste more than can be accounted for by disuse, and its electrical reactions will be unchanged. If a lesion occurs to the latter of these neurons—namely, the motor cell in the anterior horn of the spinal cord, or its axial prolongation through the nerve to the muscle—the muscle will, as in the former case, fail to receive the impulse, and will, therefore, be paralyzed, but in this case its nutrition also *will be affected*, causing it to waste rapidly, and its electrical reactions will be changed. This alteration is known as the “Reaction of Degeneration” (R.D.).

R.D., then, indicates the presence of a lesion in the lower motor neuron; and this may be in the anterior horn of the spinal cord, in the cell itself, in any part of its nerve process, or in the muscle.

It should be noted that the interruption of the impulses is the essential thing, and that the lesion may be one of partial or complete severance, or severe pressure, or, in fact, any lesion causing physiological interruption of the axon of this trophic neuron.

It is the nerve which responds to faradic stimulation, and although the muscle responds by a contraction, it in all probability receives the stimulus only through its nerve. When this has been so injured that impulses cannot reach the muscle through it, some response to faradic stimulation can generally be obtained for one or more days afterwards by direct application of the current over the belly of the muscle (see R.D.). This reaction is, however, probably still due to nerve fibrils in the substance of the muscle whose nutrition has not yet suffered from the accident.

The galvanic current causes a contraction through direct stimulation of the muscles themselves, the nerve exercising only a controlling influence.



If, however, the reaction of the muscles under examination differs from that on the other side of the body, either in character or in strength, a nervous lesion must be suspected, and it is wise to proceed to test the muscles in question with the galvanic current.

It may be noted in passing that—

The reactions to the faradic current are occasionally exaggerated. This may indicate any state of:

1. Undue irritability, such as chorea and the early stages of spastic paralysis.

2. Disease characterized by profuse sweating (which diminishes the resistance of the skin to the electric current)—*e.g.*, Graves's disease.

3. Hysteria. The reactions in this affection are variable, but in some forms there is an increased susceptibility to faradic stimulation.

The reactions to the faradic current may be diminished or absent. It is this condition which would make one suspect reaction of degeneration, and therefore makes it necessary to proceed to testing with the galvanic current.

**The Galvanic Current.**—These tests need only be undertaken where the reaction obtained with the faradic current is diminished or absent.

The conditions here are somewhat different. The current is a constant one, and as contractions only occur when the circuit is closed (*i.e.*, “made”) or opened (*i.e.*, “broken”), and not while the current is passing steadily through the muscle, it is necessary for the operator, in order to observe the character of the contractions, to close or open the circuit himself.

The galvanic battery should be tested on oneself, and, if found in good working order the electrodes *and* the skin of the patient are moistened as in the case of the faradic battery.

**The Reaction of Degeneration.**—It is convenient to begin the examination by connecting the testing electrode with the negative pole of the battery.

As in the case of the faradic current, *test first the corresponding muscles on the other side of the body* until the strength of current

required to cause a *minimal contraction* under normal conditions has been ascertained.

Now apply the testing electrode with the make key to the muscles under observation, and note the following points :

1. Amplitude of response.
2. Mode of reaction.
3. Polar susceptibility.

**1. Amplitude of Response.**—The reaction of the galvanic current may be either (a) increased or (b) decreased.

**2. Mode of Reaction.**—The changes observed in the mode of reaction are the most characteristic of all in R.D. Whereas normally a muscle reacts to galvanic stimulation by a quick reaction quickly terminated, when R.D. is present the reaction to be obtained is a sluggish one, starting slowly, of long duration, and *sometimes*, in the early stages, of greater amplitude than that to be obtained under normal conditions.

**3. Polar Susceptibility of Muscle.**—Under normal conditions a muscle responds—

To the Kathode (negative pole) at Closure (make) of Current most readily = K.C.C.

Then to the Anode (positive pole) at Closure (make) of Current next = A.C.C.

Then to the Anode (positive pole) at the Opening (break) of the Current = A.O.C.

And least readily to the Kathode (negative pole) at the Opening (break) of the Current = K.O.C.

This is generally stated shortly as follows :

$$K.C.C. > A.C.C. > A.O.C. > K.O.C.$$

the sign  $>$  signifying “ greater than.”

When, however, there is a lesion in the lower motor neuron, the order of susceptibility becomes—

$$A.C.C. > K.C.C. > K.O.C. > A.O.C.$$

In practical testing it is the first two of these (viz., the closing contractions) on which the attention is usually centred, for, if A.C.C. is greater than K.C.C., that in conjunction with the other evidence is sufficient to indicate abnormal reaction without troubling about the opening contractions.

When the nutrition of the muscle has been very seriously impaired, the reactions to the galvanic, as well as to the faradic, current become extremely slight, and ultimately entirely cease, because there is no muscle to respond. The last, or complete, stage of R.D., then, consists in the entire failure of the muscle to respond to either form of electric stimulation.

**When does Reaction of Degeneration show Itself?**—It is impossible to give exact time limits for these stages, but, roughly speaking, it may be said that no change in electrical reaction can be noted for about forty-eight hours, or sometimes longer, after the occurrence of the lesion.\*

The typical reaction of degeneration is generally established between a week and ten days after the accident, and will last for a good many weeks—six or more.

If complete severance of the nerve has occurred, the condition will, of course (unless remedial measures are adopted), go on until there is complete muscular atrophy. If, however, the cause of the physiological interference can be removed, gradual recovery will take place.

R.D. may be considerably postponed by passive exercises of the muscles at the earlier stages, either by massage and passive movements of the limbs, or by directly stimulating the muscles to contract with the electric current.

It is important to bear in mind the curious truth that, as recovery progresses, voluntary power is first re-established, and is then followed by normal susceptibility to electric stimulation.

To aid facility in diagnosis, a table of the reactions is appended, and it is very important to remember that with the galvanic current the change in *mode* of reaction is the most characteristic sign.

\* As the reaction to faradic stimulation is almost certainly that of the nerve, and not of the muscle itself, this at first appears contradictory; but it is probably explained by the fact that this reaction, which occurs for several hours after the nervous lesion, is due to the nervous elements in the muscle itself, whose nutrition is at first sufficiently good to yield a response.





**Practical Considerations.**—Several practical points with regard to the application of these tests should be mentioned.

1. If at first too strong a current be used, no reliable information can be obtained, as the resulting contraction, owing to the spread of the current to the surrounding muscles, is massive, and the reaction of any particular muscle or group

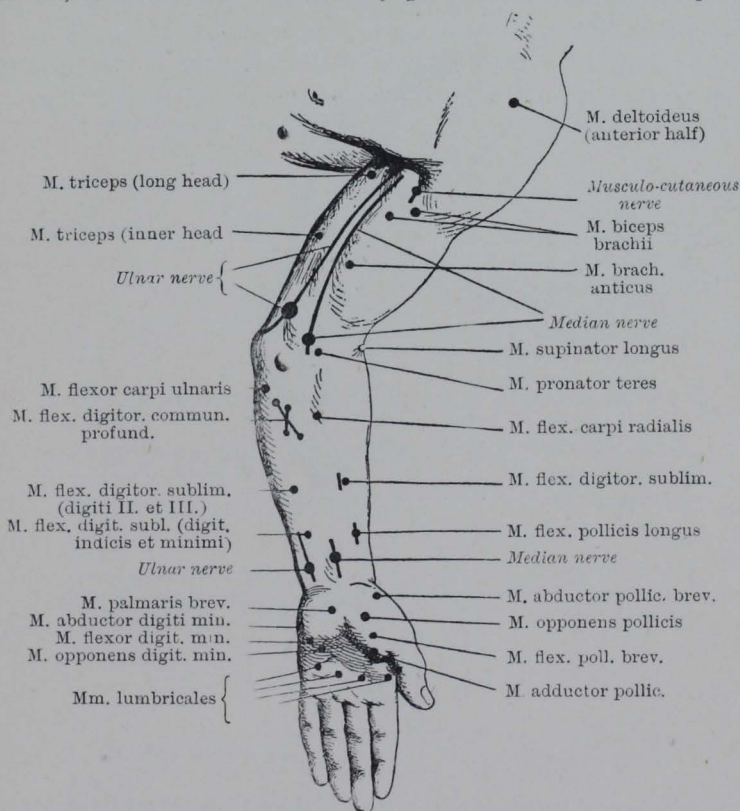


FIG. 61.—MOTOR POINTS OF UPPER LIMB. (ERB.)

of muscles, is lost in the general effect. The unpleasant sensation causes the patient to lose confidence, making further testing difficult.

2. The farther away the muscle under observation is from the central axis of the body, the greater (even under normal conditions) is the strength of the current required to produce a reaction. Thus, in a perfectly healthy subject, the small muscles of the hand will only react when a much stronger stimulus is used than need be applied in the case of the biceps.

The standard of comparison should then be, whenever possible, the corresponding muscle on the unaffected side of the body.

3. The resistance of the skin to the passage of an electric current is very great. Some attempt to diminish this is made by moistening and warming the skin by warm water. Also in actual practice it is found that the mere passage of the

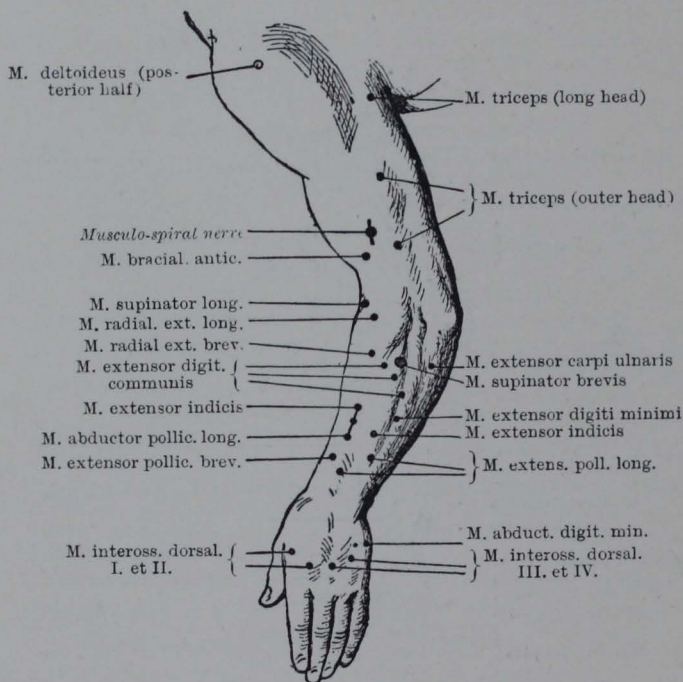


FIG. 62.—MOTOR POINTS OF UPPER LIMB. (ERB.)

electric current itself rapidly diminishes the normal resistance of the skin. When, therefore, repeated testing of one muscle is to be carried out, the current will in all probability have to be reduced during the progress of the examination to counteract this increased susceptibility.

4. **Motor Points.**—If the reaction of degeneration (R.D.) be discovered, it is important to ascertain the extent of the lesion.

This is done by carefully testing all the muscles in the affected area, with a view to discovering which nerve is injured. It is found that each muscle responds more readily to either form of the electric current when stimulated at one particular



spot than at any other. This is at the neuro-muscular spindle, and is known as the "motor point" of the muscle.\*

It is necessary, then, in carrying out these finer tests, to apply the current over the motor point of each muscle tested. These points have been carefully worked out by several investigators, and those for the limbs are reproduced here (Figs. 61-65).

5. With these before one, and bearing in mind the nerve-supply of the muscles under observation, it is now necessary carefully to work over the affected area.

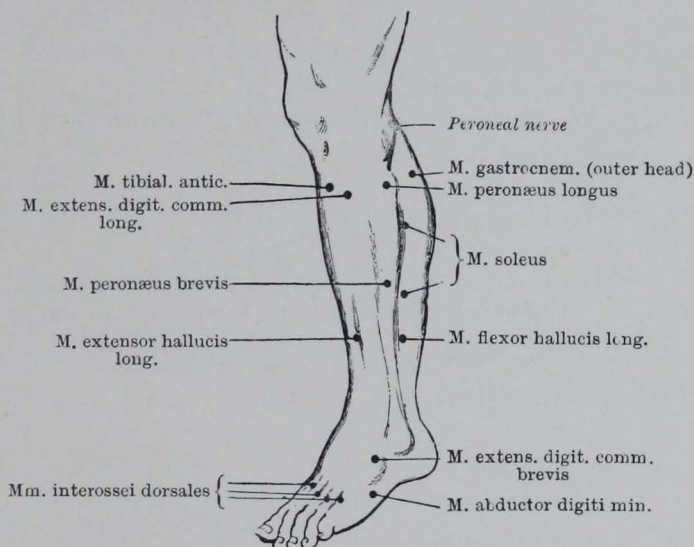


FIG. 63.—MOTOR POINTS OF LEG. (ERB.)

It is well to remember that the injury may involve—

- (1) One or more of the peripheral nerves.
- (2) One or more cords of a plexus.
- (3) One or more nerve trunks as they emerge from the spinal column.

(4) A group of motor neurons in the anterior horn of the spinal cord.

6. When drawing conclusions one should remember that where a muscle receives fibres from more than one nerve R.D. will not be obtained unless the nerve injured is the

\* A neuro-muscular spindle consists of a bundle of fine muscular fibres enclosed in a thick sheath of connective tissue. It varies in length from  $\frac{1}{8}$  to  $\frac{1}{3}$  inch, and is about  $\frac{1}{125}$  inch in diameter. Each receives a nerve fibre. It is believed that these are the end-organs of the nerves.

one mainly responsible for maintaining the nutrition of the muscle.

This varies slightly in different individuals, but a diagnosis seldom depends on the reaction of one muscle alone.

7. Difficulty may be experienced when muscles are closely grouped together in ascertaining which muscle has responded or failed to respond. This may be decided by attention to the following points :

(1) Whether the current is applied to the motor point of a muscle.

(2) Whether the contraction obtained corresponds with the known action of that muscle.

(3) Whether the contraction passes in the direction of the fibres of that muscle.

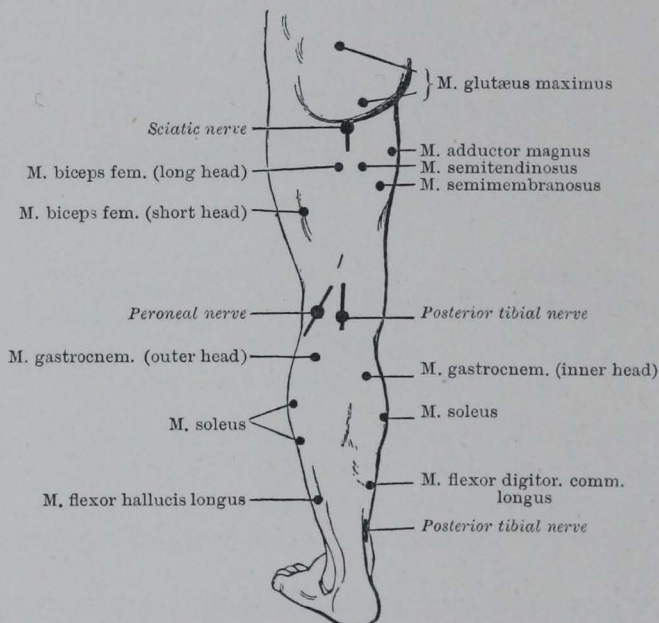


FIG. 64.—MOTOR POINTS AT BACK OF THIGH AND LEG. (ERB.)

In deciding the question of the localization of the lesion, it is also important to bear in mind the fact that reaction of degeneration only gives us information as to the condition of the motor nerves ; for the condition of the sensory fibres other tests are necessary, such as for the presence of anæsthesia, etc.

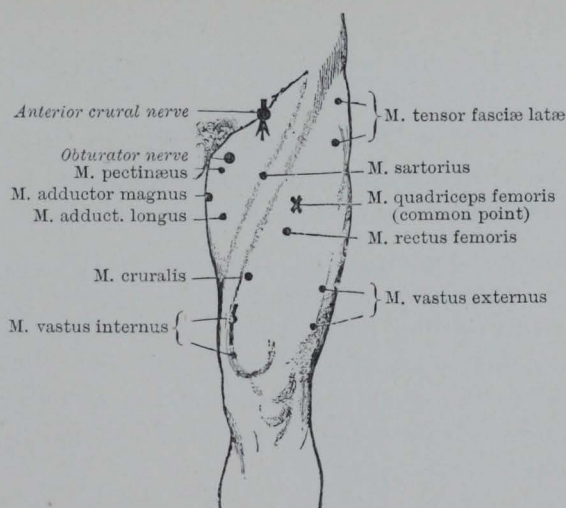


FIG. 65.—MOTOR POINTS OF ANTERIOR THIGH MUSCLES. (ERB.)

To take some practical examples of the way in which these tests may be applied :

**Injury to Median Nerve.**—The median nerve supplies the following :

Pronator teres.	Flexor longus pollicis.
Flexor carpi radialis.	Flexor profundus digitorum
Palmaris longus.	(outer half).
Flexor sublimis digitorum.	Abductor pollicis.
Pronator quadratus.	Opponens pollicis.
Lumbricals of two outer fingers.	Flexor brevis pollicis (outer head).

Result of injury : Loss of power of—

- Pronating forearm.
- Flexing wrist (this loss is almost complete).
- Flexing fingers firmly.
- Opposing or abducting the thumb (flexion also much reduced).
- Flexing the first phalanx of the two outer fingers.

N.B.—If the nerve is injured at the wrist, the first seven of the above muscles are not affected.



## Deformity :

Forearm held in position of supination.

Wrist extended.

Wasting of thenar eminence.

N.B.—It will be noticed that when the nerve is injured at the wrist the deformity is slight.

A malingerer feigning loss of power in the hand not infrequently holds it in a position similar to that produced by injury to the median nerve. The electrical reactions of the muscles which appear to be paralyzed will clear up any doubt as to whether the disability complained of is genuine or not. The contractions so obtained in the long flexors of the forearm are very easily observed.



FIG. 66.—SHOWS THE AREA OF LOSS OF SENSATION FOLLOWING LESION OF THE MEDIAN NERVE. (AFTER HEAD.)

The total area of loss is contained within the continuous thick line. The black is the area of protopathic and epicritic loss. The zone between the black area and the continuous line is the area of epicritic overlap.

Protopathic sensibility signifies the recognition of painful cutaneous stimuli and extremes of heat and cold. Epicritic sensibility signifies the recognition of light tactile stimuli and intermediate degrees of heat and cold.

(From Turner and Stewart's "Nervous Diseases.")

It must be remembered that a current of considerable strength will be required, even under normal circumstances, to produce a reaction in the small muscles of the hand.

**Injury to Ulnar Nerve.**—The ulnar nerve supplies the following :

Flexor carpi ulnaris.

Flexor profundus digitorum (inner half).

Intrinsic muscles of little finger and palmaris brevis.

Interossei.

Lumbricals of two inner fingers.

Adductors transversus and obliquus pollicis.

Flexor brevis pollicis (small inner head).

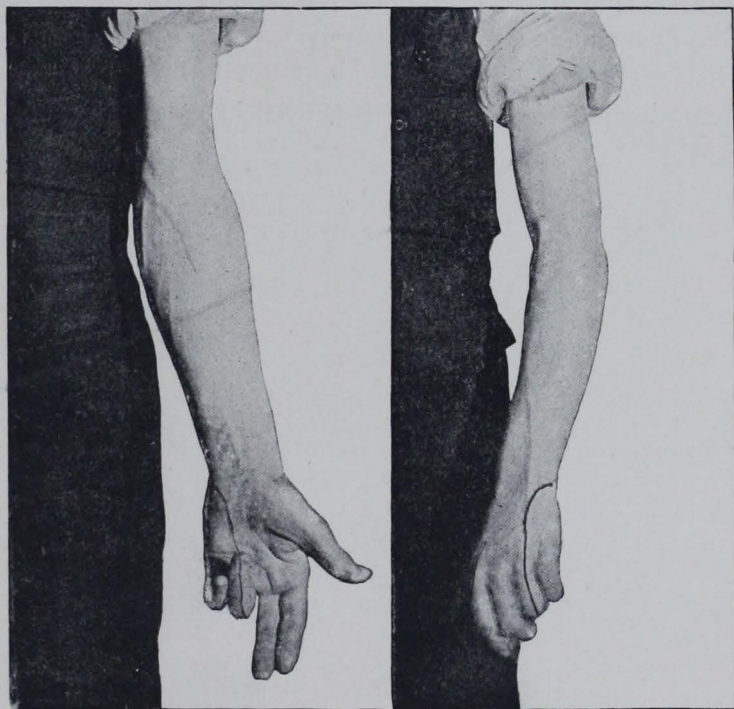


FIG. 67.—ULNAR PARALYSIS FROM A WOUND OF THE NERVE BEHIND THE INTERNAL CONDYLE OF THE HUMERUS.

The area within the black line is anæsthetic.

(From Purves Stewart's "Diagnosis of Nervous Diseases.")

Result of injury : Loss of power of—

Flexing wrist (this loss is very slight, see Median Nerve).

Adducting the thumb.

Adducting and abducting all the fingers.

Flexing the first phalanx of the two inner fingers.

Moving the little finger.

## Deformity :

Wasting of the hypothenar eminence of the hand.

Apparent prominence of the bones and tendons of the hand owing to the wasting of the interossei.

Hyperextension of the first phalanx of the two inner fingers owing to the action of the extensor unchecked by the long flexor, the interossei, or the lumbricals.

Flexion of the second and third phalanges of the two inner fingers, due also to the paralysis of the lumbricals.

Hyperextension of the thumb at the first joint.

Sensation is affected in the areas marked in the figures.

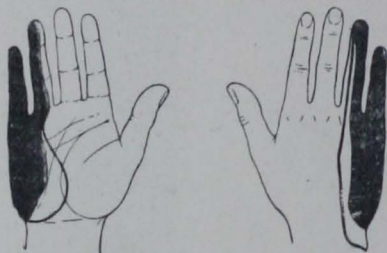


FIG. 68.—SHOWS THE LOSS OF SENSATION FOLLOWING A LESION OF THE ULNAR NERVE. (AFTER HEAD.)

The total area of loss is contained within the continuous thick line. The black is the area of protopathic and epicritic loss. The zone between the black area and the continuous line is the area of epicritic overlap.

(From Turner and Stewart's "Nervous Diseases.")

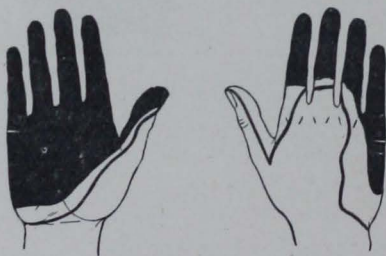


FIG. 69.—SHOWS THE LOSS OF SENSATION FOLLOWING LESION OF BOTH THE ULNAR AND MEDIAN NERVES. (AFTER HEAD.)

The total area of loss is contained within the continuous thick line. The black is the area of protopathic and epicritic loss. The zone between the black area and the continuous line is the area of epicritic overlap.

(From Turner and Stewart's "Nervous Diseases.")



**Musculo-Spiral Nerve.**—Supplies :

From its main trunk—

Triceps—long head, inner head, outer head.

Anconeus. (Supplied also by posterior interosseous nerve.)

Supinator longus.

Extensor carpi radialis longior.

Brachialis anticus (this muscle, however, receives its main nerve-supply from the musculo-cutaneous).

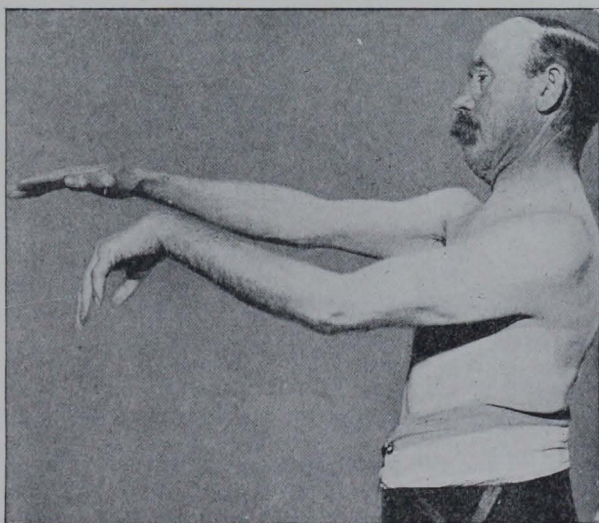


FIG. 70.—LEFT-SIDED MUSCULO-SPIRAL PARALYSIS, SHOWING DROP-WRIST AND ATROPHY OF SUPINATOR LONGUS.

(From Purves Stewart's "Diagnosis of Nervous Diseases.")

Through its branch, the posterior interosseus—

Extensor carpi radialis brevior.

Supinator brevis.

Extensor communis digitorum.

Extensor minimi digiti.

Extensor carpi ulnaris.

Extensor indicis.

Extensor ossis metacarpi pollicis.

Extensor longus pollicis.

Extensor brevis pollicis.

Result of injury : Loss of power of—

- Extending forearm.
- Supinating forearm.
- Extending wrist.
- Extending thumb and fingers.
- Weakness in flexing forearm.

Deformity :

Very characteristic position known as “ drop-wrist ” (hand flexed and pronated on forearm).

Sensation sometimes impaired at base of index-finger and thumb on dorsal aspect.

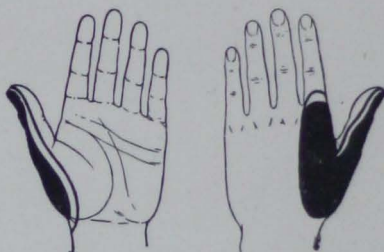


FIG. 71.—SHOWS THE LOSS OF SENSATION FOLLOWING A LESION OF THE RADIAL AND EXTERNAL CUTANEOUS NERVES. (AFTER HEAD.)

The total area of loss is contained within the continuous thick line. The black is the area of protopathic and epicritic loss. The zone between the black area and the continuous line is the area of epicritic overlap.

(From Turner and Stewart's “Nervous Diseases.”)

**Circumflex Nerve.**—Supplies—

- Deltoid.
- Teres minor.

Result of injury : Loss of power of—

- Abducting arm.

Deformity :

Only that caused locally by the wasting of the muscle.

Sensation affected in area over the insertion of the deltoid muscle.

**External Popliteal Nerve.**—Supplies—

Through its branch the musculo-cutaneous :

Peroneus longus.

Peroneus brevis.

Through its branch the anterior tibial :

Tibialis anticus.

Extensor longus digitorum.

Extensor proprius hallucis.

Peroneus tertius.

Extensor brevis digitorum.

Result of injury : Loss of power of—

Dorsal flexion of foot.

Extension of toes.

Eversion of foot.

Deformity :

Toes drop and are flexed, and the foot is raised high above the ground to prevent tripping.

Sensation impaired on outer side of leg and dorsum of foot.

Even if the reaction of degeneration be detected and localized, the problem is not completely solved, as a further point remains to be investigated—namely, whether interference with the passage of the nervous impulse is *completely* or only *partially* interrupted, and this it is often extremely difficult to determine.

The best way to arrive at a decision is to consider the amount of the reaction of degeneration (that is, whether partial or complete) in conjunction with the length of time since the occurrence of the lesion. The practical value of this point is chiefly in deciding whether, after an accident, complete or only partial physiological interruption has resulted.

The prognosis is very difficult in these cases. If complete severance of the nerve has taken place, complete loss of power of the muscles supplied thereby will occur, unless the cut ends remain in close apposition or by operation the two cut ends of the nerve are sutured to each other, so that the new fibres as they grow from the central portion of the cut nerve may find their way into the old sheath.

If only partial severance of the nerve has occurred, the



remaining portion, if uninjured, even though quite small, is sufficient to keep the two ends together, and therefore regeneration will usually take place without any operation.

It is frequently impossible to be perfectly certain what has occurred by simply testing on one occasion. Repeated examinations have to be made before it is possible to come to a decision.

These cases require considerable judgment and experience, and it is wise, if possible, to obtain the advice of an expert.

**Treatment.**—With regard to the use of electricity in the treatment of motor nerve lesions, the tendency is generally to credit the electric current with much more power than it really possesses. Electricity will not generate nerve, nor will it even hasten its regeneration.

Its great use is to prevent the muscles from degenerating while their nerve-supply is temporarily in abeyance. By applying the current, such muscles may be thrown into action frequently, thus maintaining their nutrition and efficiency, until the course of the motor nerve is re-established. Massage and passive movements are important adjuncts to the electric treatment, and should always be used along with it to promote the circulation through the affected muscles. If, in such cases, neither electricity nor massage has been employed, when the nerve impulses are again enabled to reach the muscles in question, they find the latter shrunken and flabby from non-use, thus causing much delay in the recovery of full power.

### A Short Description of the Apparatus.

These tests are carried out with (1) the faradic intermittent current, and (2) the galvanic or constant current.

**1. The Faradic Battery.**—This may be obtained in a very convenient and light form, and without great expense. The apparatus here illustrated is recommended from personal experience. If the battery is likely to be little used, a smaller and less expensive form will do, or one may be hired.

It consists essentially of a primary coil (Fig. 72), through which a current passes either from one or more (battery) cells or from the main (after modification by a transformer), and a secondary coil surrounding the primary one.

By means of binding screws two wires are inserted into this secondary circuit, which terminate in electrodes through which the current is applied to the point.

When the current is started, it passes (see Fig. 73) in the direction shown by the arrows. Passing through *M* it makes it magnetic, and this attracts *K* (Neef's hammer), thus causing it to descend in contact with *M*. When this happens, contact with the point of the screw *T* is broken and the current interrupted. *M* then ceases to be magnetic, and the hammer

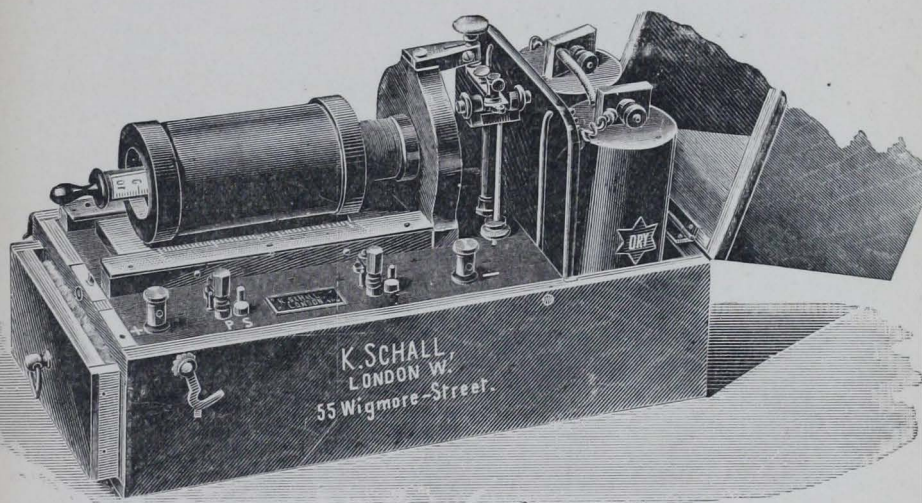


FIG. 72.—BATTERY FOR FARADIC CURRENT ONLY.

*P*, Primary coil ; *S*, secondary coil.

*K* being attached to a spring resumes the position of rest. This starts the current again by remaking contact at the point of the screw *T*.

When the current is *started* in the primary coil, it induces a weak current in the secondary coil in the opposite direction to itself.

When the current is *interrupted* in the primary coil, it induces a stronger current in the secondary coil in the same direction as itself.

Owing to the presence of the interrupting key (Neef's hammer) the current in the primary coil is started and interrupted many times a second. The stimuli to the nerve

are therefore so frequent that the muscular contractions resulting from them fuse in a form of tetany.

For this single continuous contraction to be obtained, the primary circuit must be interrupted at least twenty times a second. The strength of the current in the secondary coil increases and decreases directly with that of the current in the

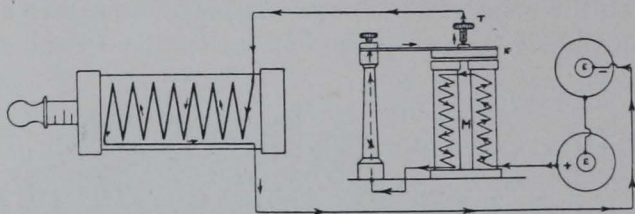


FIG. 73.

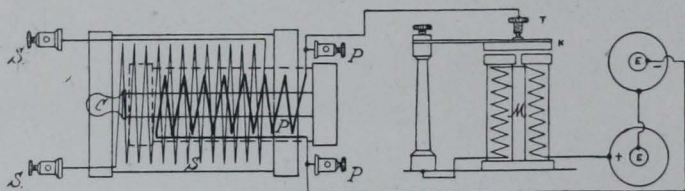


FIG. 74.

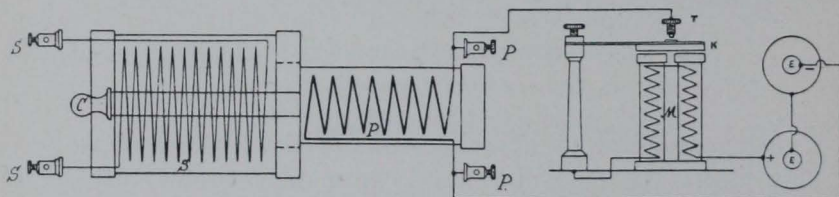


FIG. 75.

primary coil, but may also be varied by the following means : By varying the proximity of the two coils. When the secondary coil immediately surrounds the primary coil (see Fig. 74), the induced current is strong ; when, on the other hand, the secondary coil is partially withdrawn from around the primary coil (see Fig. 75), the induced current is proportionally weakened. In many batteries the current strength may further be varied by a magnetic iron core (*C*) (Fig. 75),



which, when inserted within the primary coil, increases its current strength, and consequently that of the secondary coil. Still another way of varying the strength of the current in the primary coil is by adjusting the rate of the interruption of the current by Neef's hammer.

**Electrodes.**—These are of different materials, forms, and sizes. One is rather the larger, and is applied to an indifferent

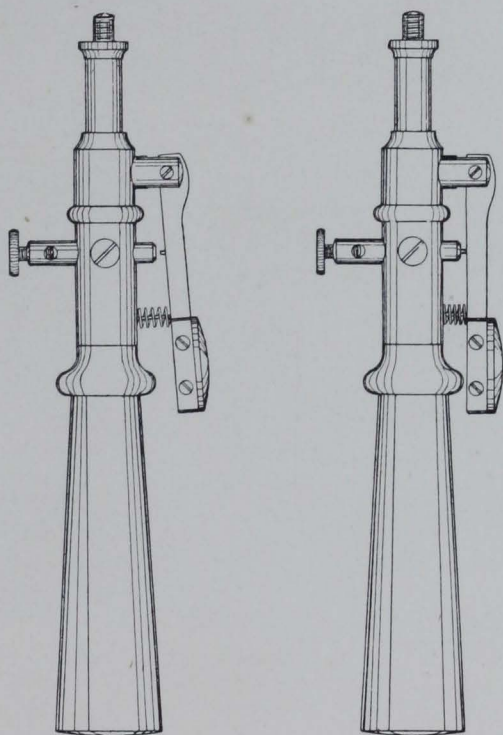


FIG. 76.—TESTING ELECTRODE HANDLE WITH MAKE KEY.

part of the body ; the other is smaller, and applied exactly to the region or structure to be tested. The intervening portion of the patient's anatomy completes the circuit.

**The Indifferent Electrode.**—This is used simply for the purpose of completing the circuit, not for the purpose of testing neighbouring structures, and is placed as far as possible away from the point to be tested, in order that the result at the two poles may not be confused, and because the unpleasant effects of

the current are minimized if, owing to a long course through the body, it is somewhat dispersed in transit.

This indifferent electrode is most conveniently made in the form of a flat piece of flexible metal covered with flannel or wash-leather, as this can be placed at the back of the neck,

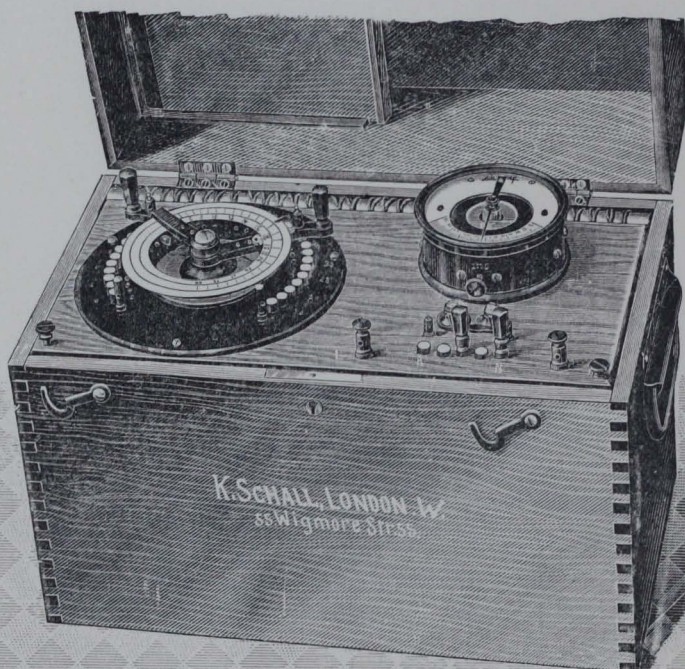


FIG. 77.—BATTERY FOR CONTINUOUS CURRENT ONLY.

*N*, Normal; *R*, current reversed.

over the sternum, the sacrum, or at any fairly central part of the body.

**The Testing Electrode.**—This should consist of a small round pad fixed to a handle several inches in length, and in this handle there should be a make key by which, upon pressure with the thumb, the current in the secondary coil may be made when required for use.



**Make Key.**—In this form the current is interrupted when the key is at rest, and it can only be completed by throwing the key into action by depression of the thumb. The advantage of this key in testing is that while it is at rest the electrode can be moved freely over the skin without causing any stimulation,



FIG. 78.—BATTERY FOR COMBINED CONTINUOUS AND FARADIC CURRENTS.

To the left, faradic battery : *P*, primary circuit ; *S*, secondary circuit.  
To the right, galvanic battery : *G*, galvanic terminals ; *F*, faradic terminals ; *R*, reversed direction of galvanic current ; *N*, normal direction of galvanic current—*i.e.*, + and - terminals as marked.

and the current therefore can be applied just when and where it is desired.

**2. Galvanic or Constant Battery.**—Electrical power for this can, if desired, as in the case of the faradic current, be obtained by a suitable transformer from the main supply, if the current is a continuous one ; but if cells are to be used, a battery of



not less than thirty cells should be employed, as considerable strength of current is necessary in some cases to demonstrate the Reaction of Degeneration.

A galvanometer should be inserted in the circuit to show that the current is properly established, and also to indicate its strength.

It is also advisable to have the cells joined up in such a way that either end of the series can be used as desired. If such an arrangement is not made, the first cells in the series get exhausted, while those at the far end have perhaps hardly been used, thus necessitating very frequent renewal, with its accompanying waste of time and other inconveniences.

The current being a continuous one, muscular contractions are only produced if the examiner himself makes or breaks the circuit; therefore, polar effects can be observed, and are of considerable importance. It is therefore necessary to have the anode and kathode clearly marked, and it is convenient to have some arrangement for automatically switching the current from the anode to the kathode cord, and *vice versa*, thus reversing the current without having to remake the connection.

The electrodes should be the same as those described for the farad'ic current.

It is possible to have the batteries for these two kinds of currents (the faradic and galvanic) combined in one instrument, and this is a very convenient arrangement if the tests are to be invariably carried out in one's own consulting-room; or the power may be used from the main, through a suitable transformer, if the current is a continuous one.