and aunts with nieces or nephews, in which are included those instances where one or both parents of the nieces and nephews are also insane. The figures denote the percentage of cases whose first attack occurred within the given age periods.

The results of the above Table VII are shown graphically in Figs. 5 and 6 (p. 1058); the abscissae represent the age periods, and the ordinates the percentage of cases whose age at the time of first attack falls within the given periods.

Of the insane nieces and nephews of insane uncles and aunts, 103 out of 208, or 49.5 per cent., had their first attack at or before the age of 25 years.

Uncle—nephew or niece	...	51 out of 93
Aunt—nephew or niece ...	...	52 „ 115
Total ...	...	103 out of 208
		= 49.5 per cent.

Finally, curves of parents, male and female, and offspring have been constructed by taking as abscissae the numbers of cases (instead of percentages) of parents and offspring and the age-periods as ordinates; the result is shown in Fig. 7. It will be observed that the parents and offspring curves represent a blending of the two curves of Figs 3 and 4.

Fig. 7.—Parents and offspring curves, showing antedating.

Permit me to point out that this signal tendency of insane offspring to suffer with a more intense form of the disease and at an early age, as shown in the above figures and tables, is of great importance for the following reasons: First, it is one of Nature's methods of ending or mending a degenerate stock; secondly, it is of importance to the physician, for he can say that there is a diminishing risk of the child of an insane parent becoming insane after he has passed 25, a matter of great importance in the question of marriage; thirdly, it is of importance in connexion with the subject of social surgery of the insane, for when the first attack of insanity occurs in the parent the children for the most part have all been born, as these tables and curves show. Sterilization would therefore be applicable in relatively few parents admitted to asylums.

In conclusion, I should like to call attention to the fact that the importance of the study of mental diseases in this country has within recent years received an impetus in several directions. First, the universities have just

commenced to grant diplomas in psychological medicine. Impressed by the great value the D.P.H. had proved in the advancement of public health, I advocated five years ago the establishment of a similar diploma for psychological medicine, in the preface of the *Archives of Neurology*, vol. iii, and I interviewed some of the heads of the profession, but, like many other innovations, it received a plentiful douche of cold water. Secondly, Dr. Henry Maudsley four years ago most generously offered £30,000 to the London County Council if they would build a hospital with pathological laboratory attached for the treatment and study of acute curable cases of insanity. This was accepted; the site has now been obtained, and the plans for this hospital have been recently sent to the Commissioners. In connexion with this, I may mention that a hospital for acute cases has recently been built in Baltimore, and the State of Massachusetts has built a hospital for acute mental diseases in Boston. Thirdly, the Royal Society of Medicine has established a Section of Psychiatry, which should do much to stimulate in this country the scientific study of mental diseases.

The Royal Sanitary Institute, I am sure, will recognize the great importance of these movements and approve of them and all others which aim at encouraging the scientific study of the causes, individual and social, of mental defects and disorders and their inheritance, for by this way alone can we hope to promote the relief of the present and the protection of future generations. And if we should feel despondent and listen to the cries of racial degeneracy, we can reflect upon the magnificent heroism displayed in the recent terrible *Titanic* disaster, which shows that the same spirit of calm resolution, devotion to duty, and unselfishness in the hour of supreme danger and death that was shown fifty years ago at the foundering of the *Birkenhead* still remains with us as an inborn character in all classes of our Anglo-Saxon race.

A Post-Graduate Lecture ON COMA AND ITS DIFFERENTIAL DIAGNOSIS.

BY
DAVID FORSYTH, M.D., D.Sc., F.R.C.P.,
CHARING CROSS AND EVELINA HOSPITALS.

I.

GIVEN a comatose patient, perhaps a stranger and, for the time being at any rate, with no friend to tell his history, how is his state to be diagnosed? The situation, though familiar to us all, can never abate its claim on our serious attention. For these patients often die, and yet, at best, our diagnostic insight is not always keen enough to pierce the obscurity of their symptoms; and sometimes, in addition, we discern across their couch the shadow of legal complications. Not, however, that the cases mostly lie near the boundary of our resources. On the contrary, the majority fall well within it, and the difficulty presents itself rather in the fact that now and again we are at a loss to recognize the patient who is dying from him, for example, whose cure can safely be left to nature within the four walls of a police cell.

However, the whole subject is too important to allow any of its clinical forms, even the simplest, to be passed over, though, by putting aside all cases of such merely fleeting insensibility as fainting, I shall restrict myself to those in which, defining them roughly, the unconsciousness is not transitory and is generally profound. The clinical picture I wish to paint is that of a patient rapidly bereft of his senses, except, perhaps, of pain, with no power of voluntary movement, and unable, whatever the provocation, to respond by blinking, swallowing, or coughing. The condition is common enough, and its causes spread over a pretty wide field. Let us begin by arranging them, not in any anatomical or even pathological order, but with the everyday, and therefore to this extent most important, varieties in the front.

In the foreground of our minds we should keep this round half-dozen: Vascular derangements of the brain

(haemorrhage, thrombosis, embolism); injuries to the head; epilepsy; diabetes; poisons; Adams-Stokes disease. A short list, from which, nevertheless, our choice will be made in the large majority of cases.

While, however, it includes the majority of the cases of coma, the majority of the causes are still to be named; and since any given case may be, for all we know beforehand, some rarer instance, these others also must be kept in mind. First, we have meningitis in its various forms, encephalitis, cerebral abscess, and cerebral tumour. Then the specific fevers, eclampsia, cholaemia, and epidemic enteritis, while to these must be added, for a reason to be given later, general paralysis and disseminated sclerosis.

Finally, in the background, as of no great consequence on account of their rarity, but stored up rather in the hope than the expectation of making use of them, these others—pernicious malaria, muscular exhaustion after, say, a long foot-race, and heat-stroke.

With this summary, which, for easy reference, I have tabulated, we have run over the list of the pathological causes.

I.	II.	III.
Derangements of the Cerebral Circulation—	Meningitis.	Pernicious malaria.
Haemorrhage.	Encephalitis.	
Thrombosis.	Cerebral abscess.	Muscular exhaustion.
Embolism.	Cerebral tumour.	Heat-stroke.
Mechanical Injury—	Fevers.	
Fractures.	Eclampsia.	
Compression,	Cholaemia.	
Epilepsy.	Epidemic enteritis.	
Uraemia.	General paralysis.	
Diabetes.	Disseminated sclerosis.	
Poisons.		
Adams-Stokes disease.		

II.

Very often, of course, if only we knew the patient's history, present or bygone, his symptoms would be obscure no longer. Suppose, for example, the relatives speak of epilepsy or of diabetes, our perplexity may fade almost to nothing. Yet, on the other hand, let us not forget that it is just when the epileptic fractures his skull or the diabetic suffers a cerebral haemorrhage that the history may beguile us. Remember, too, that a patient may lie unconscious from more than one cause at once. Of this I will give an instance later. But, with this reservation, we are justified in conceding no small weight to the history. Nevertheless, even when this is altogether lacking, the loss, though considerable, is most often repairable. After all, not many of these patients, these derelicts sunk in a sea of coma, are without something to show for their buffetings by the storms that have at last prevailed against them. Our business is to search high and low for the dents and splinters, piecing together their scraps of testimony, and so declaring the whole story of the disaster.

Before setting about this, however, it is well to study attentively the broad features of the case. Sometimes from these alone it will be possible to learn as much—possibly more—as by any elaborate quest of little details, which, however useful by way of corroborative evidence, may play us false if too much trust is reposed in them. Depend upon it, the patient, unconscious as he is, has, after all, a tale of his own to tell. He is, let us say, a burly man, past middle age, profoundly unconscious, and with his limbs lying anyhow, as they were laid. His chest heaves tumultuously and his breathing, though noisy, comes slow and deep. He puffs out his breath from a corner of his mouth, for all the world like a smoker well content, and his cheek flaps slackly in the current. Where the collar of his shirt has been loosened there is seen a violent commotion of his arteries, in keeping with the flush over his hot face. With such a patient which of us, though never so cautious, would keep or wish to keep his mind unbiassed until he had compared the pupillary reflexes, applied chemical tests to the urine, and recorded the temperature in both groins?

Or, again, take a little child, 3 or 4 years old, deep in coma. She lies on her side with her limbs drawn up and her head thrust far back. Her open eyes are vacant and filmy, with shreds of half-dried secretion lying across them, while every now and again one eyeball turns until the child squints and then turns back again. At

intervals, and between her moanings, she heaves a long, broken sigh, whereupon her breathing stops for a while until one wonders if she be dead. Time and again, too, she carries through some pitiful little movement—perhaps passing a trembling hand slowly to and fro, or with her lips making as though she ate. With this much and no more before us need opinion be reserved until the leucocytes in the cerebro-spinal fluid have been counted and the chamber of the vitreous explored with ophthalmoscope and lens?

Not that I wish for a moment to make little of a painstaking physical examination. On the contrary, this must never be omitted; for even though it tell no more than can be learnt with the naked eyes and ears, this very fact, when it can be asserted of a particular case, carries its significance. The examination, however, apart from any special investigations, need not go very wide. As a first step, the depth of the coma should be estimated as near as possible by trying to rouse the patient, say, by tweaking his hair or cuffing his cheeks. Next, compare the muscular tone of his limbs, in the hope of detecting some relative limpness on one side. Then listen to what his heart has to tell—maybe the diagnosis itself—and pay special heed to his pulse and his breathing. For the rest, if his tendon-jerks are feeble on one side than the other, or his pupils unlike in size or mobility, or if, being alike, they are very small or very large, or if his tongue goes to one side or his temperature rises—these observations will sum up most of what can profitably be learnt from the examination. Taking them altogether, their significance lies not so much in betraying the cause of the coma as in marking off those that are unilateral from those that are bilateral. Yet even on this ground we must not trust them too far.

With respect to the special investigations, the two first of the following four are practically always required; the other two only in particular cases. These are they: (1) An analysis of the urine for albumen and sugar; (2) an inspection of the retinae for albuminuric retinitis, optic neuritis, or specific choroiditis; (3) an examination of the cerebro-spinal fluid obtained through a lumbar puncture; and (4) an analysis of the stomach contents for poisons.

This completes the collection of evidence. If, having gone thus far, you are still in reasonable doubt—and, in some cases, not wide clinical experience backed up by a searching examination will place the diagnosis beyond doubt—return to your patient's bedside and watch again. Perhaps, fingering over the points in your mind but uncertain how to string them together, you may, even now, come upon the clue.

III.

How, then, having thus come to close quarters, are we to know one case from another? First, let us settle with the rarer cases in order to clear the ground for those of greater practical importance. Indeed, with regard to the rarest of all, our Group III, I need add nothing; these cases, when they occur, will almost certainly be so circumstanced as hardly to be confounded with the rest.

In Group II the cases share this much in common, distinguishing them from the principal group—that the coma appears, not without warning, but rather as the culmination of an illness already suspected and, probably, identified. For examples take the coma of typhoid fever, of meningitis, of cerebral abscesses and tumours, of cholaemia. Clinically, these cases do not often stand in the category with apoplexy and the others. Nevertheless, you should particularly remember that with some of them, albeit exceptionally, the coma is every whit as sudden and unforeseen as with, say, uraemia. Thus, I recall instances of tuberculous meningitis and cerebral tumour first showing themselves in this way, and of course most of the specific fevers of childhood may lay hold of the patient by a convulsion passing into coma. Moreover, with the two last items in the group—general paralysis of the insane and disseminated sclerosis—a convulsion leading to coma may be the first smoke from the fire, rising long before suspicion would otherwise have been roused. This prognostic is, perhaps, not always borne in mind when a patient, man or woman, previously healthy, is taken with an apparently inexplicable convulsion.

The kernel of the whole subject, however, is Group I. Here, as a first step in the differential diagnosis, it is useful, as a rule, to separate, on the one hand, cerebral haemorrhage, thrombosis, embolism and injury from, on the other, epilepsy, uraemia, diabetes, poisons, and Adams-Stokes disease; or, broadly speaking, the unilateral from the bilateral lesions.

With a *cerebral haemorrhage* the patient, perhaps while making some special effort or if stirred by excitement, is seized by a rapidly deepening coma, which, by the time the doctor reaches him, has brought him to the state already pictured. Over and above these features you may find that his limbs hang more loosely on the one side, that his head and eyes are turned from this side, and, perhaps, that his pupils are unequal. If the blood, instead of ploughing through the internal capsule, has torn up the pons, the symptoms will be modified, and the damage may be located by a crossed facial paralysis, tightly contracted pupils, a rising temperature, and not infrequently convulsions. In either event, if, as is likeliest, Bright's disease lies at the bottom of the mischief, the proof must be sought in the hypertrophied heart and tense, degenerated arteries, in the urine, and in the retinae. Nor, if still in doubt, must a chance be overlooked that the cerebro-spinal fluid may be tinged with blood that has trickled down from the head. Before drawing any conclusion from this, however, assure yourself that the blood has, in fact, come from the brain and not oozed from the lumbar puncture. This latter contingency would be proved if the blood clotted in the fluid as it stands in the test tube, or if, after centrifugalization, the supernatant liquid were left bright and clear. Alternatively, blood that had found its way from the brain would have already clotted or at least lost its power of clotting *in vitro*; and as some of its pigment would have dissolved in the spinal fluid, the latter, even after long centrifugalizing, would still be stained yellow, and would yield the test of haemoglobin, or, if not this, of its derivative bile pigment.

Cerebral thrombosis, whatever its ultimate effects, takes time to come about, in this respect differing from most cerebral haemorrhages. Usually the patient, before reaching the state of coma, passes through a few hours or days of premonitory symptoms, more especially headache and dizziness with ill-defined sensations of tingling or numbness on one side, while his mental confusion may be apparent to his friends as well as felt by himself. Later, as the clot spreads and more and more of his brain is sapped of its blood, he becomes hemiplegic and finally subsides into coma.* As to the distribution of the symptoms, this must depend on the vessel that has become plugged. With a median artery like the basilar the effects will be spread over both sides, and, since in this case the pons bears the brunt, the symptoms will be severe like those of a pontine haemorrhage. On the other hand, with the thrombus in the posterior cerebral artery carrying blood to a part of the occipital lobe, the symptoms are likely to be hemianopia with sensory aphasia; or, with obstruction in the middle cerebral artery supplying the motor cortex, hemiplegia and, when the left artery is stopped, motor aphasia; and so on. These clots most often form in people who are not so old as apoplectics, but as a rule are syphilitic. In fact, as I pointed out in an earlier lecture on the spinal cord, syphilis, by bringing endarteritis in its trail, makes itself one of the commonest causes of arterial thrombosis, whether spinal or cerebral. On this account we should be at pains, in cases of suspected thrombosis, to collect the evidence attesting syphilis.

With *embolism*, the third of the cerebral vascular lesions, the symptoms come on as suddenly as the embolism itself. The patient quickly, even instantaneously, loses consciousness, and, if he comes round again, finds himself powerless on one side and, perhaps, speechless.† The symptoms are open to be interpreted as a haemorrhage, but, as a matter of fact, the diagnosis is not often obscure, inasmuch as the embolus usually comes from the heart (especially from the mitral valve or from a clot in the left auricular appendix), where will be found proof enough to satisfy you; indeed, in cases with the suggestive presystolic thrill, you will probably draw your own con-

clusion as soon as you lay your finger on the point of the heart. If, as sometimes happens, the embolus has broken away from an ulcerating vegetation, you will find, almost for a surety, other indications of infective endocarditis, including, perhaps, embolisms elsewhere.

With the surgical *injuries* to the head only one of these three vascular conditions—haemorrhage—is at all likely to be confounded, but with this the distinction is sometimes uncertain. True, the history of an injury counts for much; but then there may be no one to speak to this. Moreover—and this is important—the injury itself may be the result of some other cause of coma. Thus, in a recent case, a gentleman, turning from the counter at his bankers, slipped (so the bystanders averred) and fell, striking his head against the mosaic floor. He was carried unconscious into the manager's office, but, after a while, seemed as though he was reviving; not for long, however, for coma began to deepen rapidly, and he soon expired. After death the vault of his skull, which was unnaturally thin, was found fractured, and, as was expected, a copious meningeal haemorrhage lay beneath. On looking further, however, we came upon what was not expected—namely, a pontine haemorrhage, the result of old-standing granular nephritis. This haemorrhage, I cannot doubt, was responsible for his slipping.

Commonly, however, the history of an injury will serve to distinguish the surgical conditions, while, with a fracture of the base, you will probably see blood coming from the ear or nose—possibly even a dribble of cerebro-spinal fluid—or beneath the conjunctiva or about the mastoid process, and, if a cranial nerve has shared in the injury, the resulting palsy. Again, in compression from a meningeal haemorrhage, the patient probably recovers temporarily from the concussion, and only after an interval, perhaps of several hours, becomes comatose; though now, with his meningeal artery bleeding furiously, he goes under very fast, his symptoms at first being hemiplegic, with the corresponding pupil widely open. In compression by a fragment of bone, on the other hand, the coma will probably follow hard on the injury.

In *epilepsy*, the patient's history of previous attacks, confirmed possibly by scars on his head or body—the legacies from earlier seizures—the testimony of eye-witnesses, the saliva churned up in his mouth, the bitten tongue, the voided urine, the symmetry of his nervous symptoms, and, as a rule, the gradual return of consciousness—some or all of these should make the diagnosis relatively easy even while the coma persists.

In contrast to this simple form, we must recognize in *uraemia* one of the most misleading varieties of coma. No doubt in some cases it is recognizable readily enough, but in others, especially when it affects a hemiplegic distribution, it succeeds in passing itself off as a unilateral lesion, most probably as a haemorrhage. Perhaps on these occasions the imposition is to be set down to a limited oedema in one hemisphere, but, whatever the explanation, this much must not be forgotten—that uraemia, at one time or another, simulates most organic cerebral conditions. It assumes many guises, and I cannot tell you how never to be deceived. In a difficulty you must put your trust mainly in the evidence for Bright's disease, supported, negatively enough, no doubt, by the absence of any sufficient reason for making another diagnosis. One further point of some little value—uraemic coma is not always very profound, and sometimes the patient, if briskly stimulated, will stir.

The coma of *diabetes* brings us once more on firmer ground, provided, that is to say, we remember that not all that reduces Fehling's solution is diabetic sugar. Other chemical bodies with this property are ready to mislead the unwary, while, of course, sugar itself may be present in the urine of a patient comatose from some pontine lesion, a cerebral haemorrhage or even from epilepsy. With none of these, however, should we expect that really copious deposit of the hydrate of copper that goes with diabetes mellitus. Yet, with diabetes proved, it were well to be not over-hasty in drawing a conclusion, inasmuch as some diabetics meet death by way of apoplexy.

Next, among the *poisons* responsible for coma (a family which, thanks to the fecundity of synthetic chemistry, grows larger almost every year by the birth of a new hypnotic) only three need be considered—alcohol, opium, and lead. The alcoholic patient, however deep in his

* By no means all cases of thrombosis, of course, any more than all cases of haemorrhage, become comatose. The smaller the lesion and the less important the cerebral areas affected the less probable is the coma.

† Here again, of course, in the milder cases coma may never develop

cups, can generally be roused, at any rate to mutter and grumble. Moreover, his nervous signs are no more on the one side than the other; and his urine, though of low gravity, is probably free from albumen and sugar. The same points hold good for opium, except, perhaps, that the patient may have sunk too far *Lethe-wards* to be recalled at all. In addition, his pupils will be narrowly contracted, his skin cold, moist, and livid, his pulse slow and weak, and towards the end his breathing flickers out. As to lead, it will be enough to remember that, now and again, instead of its customary attacks on the outposts of the nervous system, it gathers its forces to storm the citadel itself. And so hot is the assault that the brain, after a few increasingly distressful days marked by headache, vomiting, and double vision, is pushed to its last extremity of violent but useless, exhausting convulsions, and soon all is quiet in a fatal coma.

Finally, there is the unconsciousness of Adams-Stokes disease, which, beginning suddenly, and often preceded by an epileptiform convulsion, comes from the *anæmia* of the brain when the ventricle of the heart stops beating in consequence of some grave disease of the auriculo-ventricular bundle. The diagnosis in these cases is made with the finger on the pulse, when, with a beat of only thirty or even fewer, the nature of the seizure will be apparent.

Then, for a last word, let me add that, whatever our difficulties in recognizing these several states, we have always, in addition, to beware the cunning of the malingerer and the delusions of the hysteric. It is true that neither of these is ever really comatose, or even can assume the essential signs of coma. Your misgiving, if any, will lie rather in the thought that perhaps the patient, before your arrival, was indeed comatose, and is now mending. Coins of this stamp, however, usually ring false as soon as you come to test them. Yet, if you feel any uncertainty, wait; for, whatever happens, you must not mistake a genuine case of coma for one of these. With them, as indeed with every dubious case, *err—and err we all must at times*—by inclining to the side of caution and prudence.

GALVANIZATION OF THE BRAIN.

BY

R. T. CAESAR, M.R.C.S.ENG., L.R.C.P.LOND.,

MAIDSTONE.

ALTHOUGH the value of electricity is so well known, and its action on the circulation is so marked, yet it is difficult to find records of its application in brain lesions.

The following case is interesting:

D. L. J., aged 33, a mining surveyor and assayer, was at the bottom of a mine shaft in South America, when a stone or "key" fell on his head, rendering him unconscious. The skull on the left side above and in front of the ear was fractured. He was for three months unconscious—from July 5th, 1909, till about the beginning of October. He then made a gradual return to consciousness and a tedious return to partial recovery.

For an account of this period of his illness I am indebted to his Spanish doctor, who made copious notes of his condition during his stay in hospital. At that time his right arm was useless. Speech was normal, but he very easily lost all power to talk, any slight excitement or sudden noise rendering him incoherent. He suffered from convulsions which were very easily induced. He returned to England, went into a hospital, and several pieces of bone were removed, but when he left the hospital he was not much better.

When I saw him, at the beginning of July, 1911, two years after the accident, he had great difficulty in speaking, and it was often impossible to understand what he said. He was in a very nervous state, was quite unable to go about by himself, could not dress himself, as he could not use his right arm. He was unable to express his thoughts in word. His eyesight was "blurred and foggy," and he "saw double." He suffered from convulsions, very slight causes being sufficient to excite them. He was quite unable to raise his arm from his side, though he could pronate slightly; his hand was numb; he could grasp an object in his hand, but could not let go again. The electrical reactions of the various muscles were curiously varied, but there was no reaction of degeneration.

I had no hesitation in promising him considerable benefit, not complete cure, of course, but that he would, after electrical treatment, be able to speak correctly, lose his convulsions, regain his sight, and be able to use his arm. He then told me that he had been advised not to have any electrical treatment, as it would make his arm more "rigid." As time had gone on without any improvement he had made up his mind to have electrical treatment, in spite of that advice, and had undergone a three months' daily course, including Sundays, without any

benefit whatever; on the whole, he thought he was rather worse after, than before, treatment.

I commenced treatment on July 13th, 1911. On this occasion the current was passed through the brain and right arm, the latter being immersed in a porcelain arm bath. The amount of current was only 2 milliampères, this being sufficient. I afterwards increased the current considerably, being guided entirely by symptoms. On two occasions I passed as much as 30 milliampères. The application of the current was always followed by an increased sense of well-being; it acted as a tonic to him; it made him somewhat drowsy and he slept well from the commencement of his treatment. It was so pleasant in its action that he was always asking me to increase the strength. The current was passed on each occasion for fifteen minutes. This was followed by stimulation of the affected muscles by means of the interrupted-continuous current, 100 interruptions to the second. No medicine was given.

This combined treatment was carried out twice weekly till August 24th (six weeks), when I stopped all treatment.

He can now talk quite easily and plainly, having only a slight stammer, and can convert his thoughts into words without any trouble whatever. The convulsions have stopped. His eyesight is normal. He can use his right arm quite well, can dress and feed himself without any trouble, but cannot extend his thumb. The arm is much wasted, but is considerably increased in size under the treatment, and will continue to improve with use.

He goes about by himself, using train, bus, etc., according to his needs, transacts his business, and has so entirely and successfully recovered that he has obtained another post similar to the one he occupied before his accident.

It is seldom possible to obtain such direct evidence of the value of passing an electric current through the brain.

For some unknown reason this treatment is not adopted in many cases which undoubtedly would be benefited by it. There must be a large number of persons suffering from the after-effects of a "stroke," who would derive considerable benefit from this method of treatment, and who would be willing to submit to it if even they had a chance of improving their condition.

The condition of the brain and its coverings is very much the same whether the lesion is caused by an effusion of blood from a vessel damaged either by degeneration of its walls or from injury. The blood is there in either case, attended by pressure effects. In recovering cases a certain amount of absorption takes place, but there still remains an appreciable amount, which can often be directly influenced by a suitable electric current, leading to a further improvement.

The rapidity with which the effusion, due to a recent injury, disappears under the application of a properly applied current is very striking. I have reduced a 2½ in. swelling of the ankle-joint in thirty-five minutes, and that is quite an ordinary and usual experience, and the reduction is accompanied by entire loss of pain.

If, then, this can be done to the ankle-joint, why not make use of the same treatment in the case of the brain? It is, of course, not advisable to use the galvanic current too soon, time must be given for recovery from shock. This would probably begin at a month or six weeks from the onset. In cases of embolism it would do no harm to commence much earlier. Each case should be judged on its own merits, and every case, if possible, should be treated by the foregoing method. Electrification of the paralysed muscles alone is not sufficient, as this case clearly demonstrates, but what may be called the greater treatment should include the less.

We are, after all, only making use of Nature's own remedy. An electric current is set up in our bodies whenever a muscle is used or the heart beats. Whenever any part of the human body is injured a current is caused and flows in a definite direction and can be measured. This electric current must therefore be necessary, and probably acts by stimulating the various cells of the part to increased work, thus leading to repair of tissue and absorption of deposits.

The current should be turned on very gradually and carefully measured by a galvanometer. The choice of and relative position of the poles vary in any given case. The electrode should be water, the usual pads of gauze or lint not being so satisfactory. The patient must be assiduously attended to throughout the treatment.

UNDER the title of *Dunlop Motor Tyres, 1912*, the Dunlop Company has brought out a booklet which, in addition to information as to prices, contains a large number of useful hints on the subject of economical driving. There are also well-illustrated descriptions of the firm's detachable wheels.