rubber bag is suspended at a good height the infusion can be completed within ten minutes. The fluid is absorbed very rapidly, so that it is unnecessary to use such an elaborate and comparatively dangerous method as intravenous infusion, still used by those who do not know or who do not appreciate the advantages of the simpler method. In bad cases the initial axillary injection is followed by the continuous rectal saline infusion during the first twenty-four hours. In milder cases I prefer to have a saline enema given regularly every three hours. If carefully given it is generally retained.

To procure sleep the first night after the operation a dose of morphine may be injected subcutaneously, or 30 grains of aspirin may be added to a saline enema.

In the treatment of the shock strychnine is avoided, because clinical experience confirms the statement of Crile that strychnine is the surest method of producing shock. Alcohol is not much better. The effect of adrenalin is very transient, and the same is true to a lesser degree of the various preparations of ergot. Lately I have used subcutaneous injections of pituitary extract, which raises the blood pressure for several hours. Shock is rarely severe when the operation is completed, as it should be, within twenty or thirty minutes. The collapse, which is due to deficiency of fluid, is best treated by giving plenty of water by the mouth, by the rectum, or subcutaneously. It is particularly important not to give any purgative during the first two days after the operation; there is nothing more harmful. There is no need to get the bowels open for the first day or two, because they are generally fairly empty as a result of the vomiting and enforced starvation which usually precede the operation. Moreover, it is of vital importance that the small intestine should be kept at rest, for intestinal movements tend to disseminate the peritonitis. The purgative often fails to act, especially in the worst cases. Under these circumstances the drug is an irritant poison, and generally induces vomiting. The bowels generally act naturally on the third or fourth day, and often there is diarrhoea about this time. An enema given after the third day does no harm, and is useful to empty the lower bowel. If flatulence be troublesome, minim doses of oil of peppermint may be given by the mouth or a rubber tube with several perforations may be passed into the rectum and left *in situ* for an hour or more at a time. It is unfortunate that many people still believe that it is necessary to purge these patients. Some have an idea that if the bowels are not opened soon the patient will die. Others think that an action of the bowel is necessary for the excretion of toxins. There is no foundation for either belief. It is true enough that the bowels are paralysed in the worst cases in which the operation has been too long delayed, but no purgative will make the paralysed bowel act. My experience is that it increases the distension, probably by forcing the contents of the upper and comparatively healthy part of the intestine into the ileum, which is more or less paralysed.

The treatment of paralytic distension of the intestine is a difficult subject. Pituitary extract may be found useful, as suggested by Blair Bell, but in bad cases temporary enterostomy is the only thing likely to succeed. In several cases treated in this way I have been able, three or four days later, to close the fistula under a local anaesthetic. Three or four days after the operation laxatives and purgatives are not so dangerous, and it is often good to give small doses of nux vomica and belladonna, with the idea of keeping the intestines active at this stage to prevent them getting too adherent. The half-sitting position is very valuable to ease the breathing and to lessen the risks of subdiaphragmatic and pulmonary complications.

In the after-treatment of the wound, when drainage has to be adopted, the drainage tube is generally removed about the third day, and is replaced by a smaller one or by a wisp of gauze. In many cases when there is only a little discharge the stitches previously inserted are tied, almost completely closing the wound. When there is much discharge, drainage has to be continued for a longer time. When the discharge has the offensive smell characteristic of the *Bacillus coli*, the wound may be bathed and the dressing soaked with a weak solution of sodium hypochlorite or hydrogen peroxide. Healing is sometimes hastened by the use of *Bacillus coli communis* vaccine.

When drainage at the vagina has been adopted the tube is removed on the third day. If the temperature goes up again, the wound at the posterior fornix can be easily dilated with the finger to let out any collection of pus. A vaginal douche is used in these cases.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, January 29th, 1910, p. 245.

THE EFFECT OF ABDOMINAL COMPRESSION IN A CASE OF VERY RAPID HEART,

WITH INSPIRATORY OBLITERATION OF THE
RADIAL PULSE, FOLLOWING INJURY:
RECOVERY.

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WE know so little of the causes of the conditions at present described together under the common name of "paroxysmal tachycardia" that I may be excused for giving a fairly full description of the following case. Some additional interest attaches to it because, in its course, an inspiratory obliteration of the radial pulse (the so-called "pulsus paradoxus") was observed. I do not think that this inspiratory obliteration has been previously recorded in association with cases of this sort. Abdominal compression is a recognized method of treatment in "paroxysmal tachycardia," and in this instance it appeared to have been the chief means of saving life.

A gentleman aged 68, well built and healthy, was on April 3rd, 1909, thrown down by a large motor car which backed on to him. No bones were broken (as an x-ray examination proved), nor was there any cerebral concussion, but his right hip and thigh were bruised, as well as the left side of his face, and probably a certain amount of general shock was sustained. He was attended by Mr. Russell Coombe, removed to Miss Johnstone's Home Hospital, and placed under the best possible conditions. It is not unimportant to state that he seemed in no sense neurotic, being, in fact, very courageous, and, under the circumstances, most philosophic.

His pulse on the day of the accident was counted at 100, and between April 3rd and 10th it varied between 100 and 80. The accompanying chart shows this as well as the subsequent changes in the pulse until after his last attack of tachycardia. The pain in the bruised parts gradually subsided, but on April 5th, two days after the accident, he complained of severe abdominal pain and was sick three times; he had also hiccough and a great deal of flatulence. On April 7th and 8th he was more comfortable, but on April 9th he began to complain of an aching pain over the heart, with a good deal of flatulence and abdominal distension, and his pulse became rather weak, intermittent and irregular. On April 10th, just a week from his accident, Mr. Coombe and I saw him together. We found him in the condition described, with considerable gastric and intestinal distension. The heart was apparently of normal size, free from murmur, but with rather feeble sounds. There was slight pulmonary emphysema but no other lung trouble. The urine was normal. A mixture containing bismuth, soda, carbolic acid, and nux vomica was ordered and a simple aperient.

During the day (April 10th) the pulse became very unsatisfactory, and Mr. Coombe asked me to see him again in the afternoon. The pulse was then very small, irregular, and intermittent, running about 100, and the patient was pale and perspiring. There was no dyspnoea, no distension of the veins in the neck, no sign of cardiac dilatation. There was no murmur or friction to be heard, only rather feeble sounds. He still complained of aching over the heart. He was ordered $\frac{1}{4}$ gr. of strychnine hypodermically every four hours with this four-hourly mixture: R Spt. aetheris nitrosi, spt. am. aromat., tinct. lavand. co., $\bar{a}\bar{a}$ m xv, aquam ad $\bar{z}$; also a tablespoonful of whisky every six hours. Under this treatment the pulse improved somewhat, remaining, however, very intermittent. Next day (April 11th) it rose further in frequency, and at 2 p.m. was running at 144. Small doses of digitalis were now given, and these reduced the rate as counted at the wrist. But this apparent reduction was only due to the digitalis increasing the number of beats which did not reach the radial artery, so on April 13th strophanthus was substituted, with small doses of ammonium bromide.

On the evening of April 13th the right saphenous vein became thrombosed. The pulse was then counted at 180. Next morning it was counted at 74. Cheyne-Stokes breathing was very marked on April 14th, and occurred occasionally for several days.

From this time until April 21st a very serious condition was maintained, the rate of the pulse varying greatly from time to time, as the chart shows, but being usually excessive, and often very rapid. At no time was there dyspnoea, cardiac dilatation,

PULSE CHART.—A, Accident. B, Binder applied. C, (?) if real rate so intermittent. D, Real rate. E, Pulse intermittent. F, Pulse feeble, very irregular, and intermittent. G, Pulse regular. H, Pulse irregular and intermittent. I, Pulse feeble, irregular, and intermittent.

or murmurs, distension of veins, or oedema, except in the thrombosed leg.

Nutrient enemata, with limited liquid food by the mouth, were begun on April 11th, and continued until April 18th, the liquid diet being kept up until April 19th, when solids were again cautiously begun. On April 21st he was extremely ill, and then for the first time I observed that during full inspiration the feeble, rapid pulse became absolutely obliterated. Mr. Russell Coombe confirmed this observation. I then decided to try the effect of abdominal compression, and a stout flannel binder was tightly applied all over the abdomen at 11.40 a.m. No other change was made in the treatment. No immediate improvement was noted in the condition; at 2 p.m. the pulse was 150; at 6 p.m., 146; at 10 p.m., 146. But at midnight it had fallen to 88, and was more regular; at 3 a.m. it was much stronger (64), but intermittent; at 5.45 it was again rapid (about 140), and at 10 a.m. it had dropped to 72.

A nearly liquid diet was resumed. From this time forward the pulse returned to an ordinary rate, stronger and not intermittent. On April 26th, the binder was loosened. On April 27th solids were cautiously begun again. On April 29th he was so much better that he was allowed to be propped up in bed for an hour. At first this caused some intermission of the pulse, but the effect soon passed off. On the succeeding days no intermissions were observed. On May 2nd he was propped up for two hours, and on May 3rd for a little over three hours. On May 5th he sat out of bed for nearly two hours, and seemed none the worse for it. His pulse had been excellent for days. On May 6th he was carried (in bed) into a larger room. There was no jolting or indeed any disturbance of the patient, but his pulse rose soon after to 132. There had been a delay in giving him an enema that morning, and the rise of pulse was attributed to that. This rate was maintained till the afternoon, when it again fell to normal. At the same time the patient complained a good deal of "indigestion." At 3.30 a.m. the pulse rose to 146 and remained at that for almost an hour, then falling to 90. At 5 a.m. it was 84, small, and regular. I again applied the abdominal binder, in the hope of preventing further accelerations, and noticed that the pulse at once slightly increased in size. After this the rate remained natural, but the pulse was not so good as before. That day some oedema and pain were observed on the back of the right hand, and a few days later a similar swelling affected the left hand.

The binder was loosened after a few days. On May 10th some venous thrombosis took place in the left leg, below the knee, with a great deal of pain. The tachycardia returned on May 12th, having been heralded for some days by occasional intermission. He was now again extremely ill, very pale and grey looking, perspiring freely, the pulse very small and flickering, intermittent and irregular. At one time it seemed scarcely possible that he could survive. An enema of strong coffee did some service. So pale was he that I thought it worth while to examine the haemoglobin value of the blood. It was normal, as was the blood in other respects. Digitalin was tried on May 13th, and on May 14th, when he was again desperately bad, digitalis was prescribed with ether and strychnine. This undoubtedly did good, but did not control the tachycardia in the least.

On May 16th, at 5.30 a.m. the pulse was 164—very bad indeed. An enema of strong coffee seemed to pull the patient together, and the pulse gained in strength. At 9.30 a.m., the pulse being then 164, a binder was very tightly applied by Mr. Coombe. In a quarter of an hour the pulse fell to 96, regular and good, with only occasional intermissions.

This was the last attack of tachycardia. At the same time he was put on the following mixture: R. Tinct. strophanth. m v, ammon. bromid. gr. v, spt. ammon. arom. m xv, spt. aetheris nitrosi m xx, tinct. lavand. co. m xv, aq. ad $\mathfrak{z}$ j, every four hours; and from this date improvement was continuous.

On May 23rd the patient was well enough to be taken home to London. He had a troublesome attack of boils soon afterwards, but except for occasional very slight pulse acceleration, he progressed slowly and uneventfully. Dr. Harper tells me that he is now "wonderfully well, and beyond occasional slight pulse acceleration, generally connected with indigestion, there has been no attempt whatever at a tachycardia."

About the gravity of the patient's condition during the continuance of the tachycardia there could be no sort of doubt. Mr. Russell Coombe, Dr. Harper (of Rosary

Gardens, London, his usual medical attendant), my colleague Dr. Henry Davy, and Sir Clifford Allbutt, who saw him with me at various times, all agreed as to its extreme seriousness.

His attacks seemed clearly in some way consequent on his accident; also on his flatulent indigestion. I fancied that a decrease in the amount of fluid ingested tended to increase his liability to tachycardia. The temperature throughout was normal except at the time of each attack of thrombosis, when, however, it only rose very slightly.

I have refrained from calling the case one of "paroxysmal tachycardia." So many diverse conditions have been described by that name that it has ceased to have any precise significance. It was first applied, and, I venture to think, its use should again be restricted, to those cases in which, without discoverable cardiac lesion, and often without obvious reason, a normal pulse-rate suddenly or almost suddenly rises to a very high frequency, maintains it for minutes, days, or weeks, and then suddenly or almost suddenly returns to its original pace. Such cases are altogether different from the present one.

With regard to the inspiratory obliteration of the pulse, the so-called "pulsus paradoxus" (a term which surely ought to be given up), one cannot be expected to acquiesce in the present attitude of needlessness of explanation. If the phenomenon means anything in a case in which heart and lungs are free from demonstrable disease, it surely must indicate that the left side of the heart is so imperfectly supplied with blood that the intrathoracic suction action of inspiration, resisting outflow from the chest, is sufficient to greatly lessen the outgoing current. It was in this sense that I interpreted it. The pale livid face and hands suggested arteries half empty. The blood itself was normal. Where, then, was the blood? One could only suppose that it was ponded in the splanchnic area, much as most people believe it to be in surgical shock. To determine the state of the blood pressure I used a Potain's sphygmomanometer during his last attack (one recognizes its defects, but a Riva Rocci seemed undesirable in his state), and found that the pressure was only 100 mm. on May 15th.

In surgical shock the pulse-rate may rise to an uncountable point.

The effect of the binder I think can best be explained by its effect on the overcharged splanchnic circulation.

There are a considerable number of cases on record in which treatment directed to emptying the splanchnic vessels—compression of the abdomen in various ways, even inversion of the patient—has reduced the excessive rate of the heart to normal. Sir Clifford Allbutt refers to this in his interesting article on the subject in the last edition of his *System of Medicine*, and references to a number of instances will be found in Professor Hoover's article in Osler and McCrae's *System*.

How great may be the effects of a sudden opening of the splanchnic flood-gates I once had an opportunity of seeing, and, as the case has not been published and the explanation of such cases has not hitherto, so far as I am aware, been given, I may briefly mention it here.

A patient with left-sided empyema—not under my care, by the way—was having the empyema washed out, when he suddenly became faint and quickly died, with the appearance of one suffering from a rapid internal haemorrhage. At the *post-mortem* examination we found no haemorrhage. But the abdominal organs presented an appearance such as I have never seen before or since

—the solid organs swollen and purple with excess of blood content, and, when cut, running out like overfull sponges. The splanchnic nerves, it will be remembered, run down within the thorax.

If paroxysmal tachycardia can be really determined by dilatation of the splanchnic area and the consequent greatly diminished supply of blood to the left ventricle, it is easier to understand the usually sudden manner of its inception and termination.

Other remedies suggested for paroxysmal tachycardia are atropine (by Dr. Herringham) and vascular dilatants (by Sir James Barr). It is interesting to note that, although in this case the blood pressure was low, yet the patient seemed to do decidedly better when on a mixture containing spirits of nitrous ether.

A RED LINE ON THE GUMS.

BY

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DURING the last three years it has been my privilege to examine a very large number of men (about 900) employed at works in which lead ores are handled, and with such an opportunity one would not be surprised at the possibility of finding some new feature associated with the poisoning symptoms classified under the heading of "plumbism."

It is astonishing what a large number of cases have been placed in the category of plumbism since the passing of the new Compensation Act, which, under circumstances associated with fewer temptations, both to the workman and his doctor, would most probably have been put down to alcohol, syphilis, rheumatism, or some such disease.

The workmen are drawn from a stratum of society composed of unskilled persons, who have been brought up amidst surroundings that do not conduce to any mental or moral elevation, with the result that their resistance to temptations of an alcoholic nature is of the feeblest. With a diminished income due to alcoholic excess, this class of workman does not obtain the necessary quantity of proper food to enable him to resist disease, which, if it be associated with the blue line on the gums, is most likely classified as plumbism.

What is this blue line? In a large number of the older "hands" one nearly always sees a dark, dirty line, which, with the application of a suitable tooth powder on a good toothbrush would rapidly disappear; this is not the lead line. The typical lead line is seen to be just under the surface of the edge of the gum, and situated in the papillae, and under a magnifying glass presents an interrupted appearance. These papillae in some cases become enlarged almost to the extent of rupture, and the question naturally arises, What is the cause?

The following case is very interesting as bearing on the question:

A. B., aged 29, has been engaged at the same works for about ten years. On August 2nd, 1909, he lost the sight of his left eye, which had only troubled him for a few hours previously by becoming excessively watery. He gave a history of an attack of colic (which may have been due to lead) in May, 1909, but which did not last long.

He is rather anaemic; the left eye is damaged by a haemorrhage into the anterior chamber, which completely interferes with his sight. There is no paralysis. On testing the saliva the potassium sulpho-cyanide reaction is well marked, and to those who have had much experience of this test such a fact will be surprising. On the gum a marked blue line, with which is associated a line of very enlarged papillae, in many places enlarged to the extent of rupture.

Is this engorgement due to the presence of lead in the system? About two years ago, when examining the men at a works in the district, I was particularly struck with the fact that a large percentage of *their gums showed a distinctly red line*, in marked contrast to the men at other similar works, so much so that I drew the attention of Dr. Legge, of the Home Office, and Dr. Collis, one of the inspectors, to the matter, and the latter has corroborated my observation. This red line is, in my opinion, due to an enlargement of the papillae at the edge of the gum, and, peculiarly enough, was seen in lads who had been only fourteen to twenty-eight days at the works, as well as in the outside men, such as masons, whose contact with the fumes occurred only when the latter were diluted.

The works are situated on the bank of a river, and are nearly surrounded by works of many sorts, so that the atmosphere is most probably charged with particles of an irritating nature, and no doubt it is this source of irritation, especially in the case of boys newly arrived at the works, that causes the engorgement, to which the lead finds its way, and on which it leaves its mark.

The conclusion that one naturally arrives at from such facts is that the so-called lead line is no proof of lead in the system, but that it appears in workers whose gums have been irritated by particles other than lead.

The Lettsomian Lectures

ON

THE CEREBELLUM AND ITS AFFECTIONS.

DELIVERED BEFORE THE MEDICAL SOCIETY OF LONDON,

BY

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LECTURE III.

NON SUPPURATIVE ENCEPHALITIS.

NON-SUPPURATIVE encephalitis may occur in adults in association with infective diseases, but has been especially described in children after one or other of the acute diseases to which they are so liable. The mode of development of the acute ataxia in a child previously healthy, with the subsequent gradual improvement, after an attack of measles or some similar disease, are points that are highly suggestive of either encephalitis or acute degeneration, due to the action of some toxin on the nerve elements of the cerebellum. Such evidence cannot, however, be accepted without *post-mortem* confirmation, so that it is satisfactory to find that when some such cases have come to necropsy, although, as a rule, this has been at a remote period from the acute attack, the evidence of sclerotic changes in the cerebellum has been such as to justify the original diagnosis of encephalitis.

Prominent among those who have directed attention to this class of case in children is my colleague, Dr. F. E. Batten, who, in a paper on ataxia in childhood, brings out the main clinical features of the affection as it occurs in children. A child, previously healthy and of good physical and mental development, becomes ill of some acute febrile disease, which may or may not assume a definite recognized type, such as that of one of the exanthems. Loss of consciousness may supervene, but general convulsions are exceptional. After a few days, in which the child appears to be making a normal convalescence, it is sat up, but is found to be unable to maintain itself in this posture owing to loss of balance. The child's hands reveal marked incoordination, and, if supported on its feet, the lower limbs are found to be wildly ataxic. Moreover, some articulatory defect of speech may be noticed. The general health improves in the course of a few days, but the child continues ataxic for a time, after which recovery gradually takes place, and may be completed in a few months, or it may take two or three years before this result is reached.

Although the cases included in Batten's paper are open to the criticism that they are unsupported by any pathological data, in view of the fact that all the patients recovered, it will nevertheless be admitted that they are identical with other cases that have come to necropsy, in which evidences of encephalitis have been established.

No better example of this is to be found than in a case recorded by Clapton.

A child, aged 4 years, who had been previously healthy, was attacked by measles, after which she suddenly became unable to use her limbs or to talk. After remaining in more or less the same condition for about six months she began to improve, but even at the age of 15 her gait was still ataxic, and she could not use her hands properly. Death took place at the age of 33, when the cerebellum was found to be less than a third of the normal weight, and presented neuroglial proliferation, with