treatment is now complete since slight traction on the stalk of the bag or on the leg of the foetus effectively stays further hæmorrhage by causing an inverted cone to press on the lower uterine segment; delivery can now be awaited with equanimity, the strength of the patient being supported as much as is necessary. It matters little whether the bag be pushed through the placenta or membranes or between the uterus and placenta. Ergot was given in several cases with no bad result. In a case with a very rigid cervix ergot might possibly do harm in the first stage of labour by tiring out the uterus in overcoming the resistant cervix and so lead to atony and post-partum hæmorrhage later; for this reason ergot was not given early in Case 2. Moreover, tension on the stalk of the bag is enough to put a stop to flooding. After the bag is delivered, unless the head or breech is driven down by strong pains, internal version is the best course, since flooding occurred in several cases after the bag was expelled. In these cases the insertion of the arm for version acts as a plug till the foetus is turned and can in its turn act as a plug. The alternative of applying the forceps may be rendered difficult by the distance the head is up and by the placenta baulking the operator. Although nothing untoward happened in the seven cases thus dealt with in this collection yet the forceps is of bad repute in the treatment of placenta prævia, for King mentions ten cases with a mortality of 30 per cent. and Auvard 21 cases in which the forceps was used at the "moment of election" with a mortality of 45 per cent.

If the os be considerably dilated when the case is first seen internal version or bringing down a leg in pelvic presentations seems to be the indication; inserting a bag would only increase the amount of manipulation without corresponding advantage. As has been already pointed out, the high mortality of cases suitable for this plan of treatment is due to the fact that so many of them are in a very dangerous condition when first seen. Having met the first indication—namely, checking the hæmorrhage—the next point is to keep up the patient's strength. In severe cases the quickest method is by auto-transfusion, or, in other words, raising the foot of the bed on chairs. Next in rapidity of action is intravenous infusion of saline solution. The latter, though a simple proceeding in a hospital or decent house, where some assistance and clean water can be obtained, may yet in the hovels of the poor prove a very different matter; the water may be so full of extraneous matter as to block completely fine tubes and cannulæ. It is here that normal saline solution given per rectum is of the greatest service; it is rapidly absorbed and can be quickly given with a Higginson's syringe. The rectum of freshly delivered women is surprisingly enduring of liquid refreshment. From four to six pints can be given in this way in from one to two hours without any discomfort or return. The weak point about this method is that it cannot be used till labour is complete, whereas intravenous or subcutaneous infusion can be used as soon as hæmorrhage is controlled. With regard to the third stage of labour, it is well to remember the comparative frequency of adherence of a placenta prævia, and consequently there is reason to remove the placenta manually more often than in normal cases.

CASE A.

Recorded for the difficulty in diagnosis; probable rupture of varicose veins of the vagina; gross hæmorrhage; plugging; recovery.—An English multipara with a badly ruptured perineum and slightly contracted pelvis was advised to have labour induced at eight and a half months. There were very large varicose veins of the legs extending on to the vulva and up the vagina. The patient was on an inclinable table and I had just got so far as to begin inserting a bougie for the purpose of inducing labour when the patient strained two or three times. In a moment there came a gush of hæmorrhage as if a large jug had been upset. Swabbing away blood to look for a bleeding point was out of the question. A clenched fist thrust into the vagina and raising the pelvis also failed. Owing to the ruptured perineum blood poured out all round. The patient was already blanched and nearly pulseless. I hurriedly rolled a sterilised towel into a ball and pushed it up the vagina, the flooding ceased, though how much from the plug and how much from loss of blood is difficult to say. Not having much confidence in the plugging and thinking it possible that passing the bougie might have detached a portion of a low-lying placenta I thought it advisable to make pressure on any bleeding point which there might be from the

upper end of the lower uterine segment down to the vulva. I therefore, having removed the towel quickly, inserted a de Ribes bag, the os being sufficiently patulous. Having filled the bag with water, I then stuffed the vagina with gauze, at the same time pulling down the bag and so exerting firm pressure on the whole lower part of the uterus and vagina. There was no further sign of hæmorrhage. Although the whole episode had not taken more than ten minutes the patient seemed to be on the point of dying from acute anæmia. Five pints of intravenous saline infusion, however, made her rally and in 12 hours she was delivered of bag, plug, and dead child without further bleeding. She made a symptomless recovery. The exact source of bleeding remained undiscovered.

Bibliography.—Auvard: *Treatment*, Thèse d'Agrégation, 1866. Barnes: *The LANCET*, Lettsomian Lectures, July 4th (p. 1) and 18th (p. 51), August 15th (p. 159) and 29th (p. 211), Sept. 12th (p. 261), and Oct. 10th (p. 357), 1857; *Edinburgh Medical Journal*, 1862; and *Lectures on Obstetrical Operations*, 1876. Behm: *Treatment by Bipolar Version*, *Zeitschrift für Geburtshilfe und Gynäkologie*, 1883, Band ix. Blacker: *Treatment with de Ribes bag*, *Transactions of the Obstetrical Society of London*, 1897. Braxton Hicks: *Bipolar Version*, *THE LANCET*, July 14th (p. 28) and 21st (p. 55), 1860; *Transactions of the Obstetrical Society of London*, 1864. Champetier de Ribes: *Description of Bag*, *Annales de Gynécologie*, December, 1888, p. 401. Charpentier: *Traité des Accouchements*, 1889. Conroy: *Treatment by Casarean Section*, *Boston Medical and Surgical Journal*, 1902. Donoghue: *Treatment by Casarean Section*, *Boston Medical and Surgical Journal*, 1900, p. 571. Dudley: *Treatment by Casarean Section*, *New York Medical Journal*, Nov. 3rd, 1900. Dührssen: *Vaginal Casarean Section as Treatment*, *Centralblatt für Gynäkologie*, 1904, No. xiii. Godwin: *Hydramnios and Placenta Prævia*, *THE LANCET*, Sept. 27th, 1902, p. 869. Herman: *de Ribes Bag*, *THE LANCET*, Jan. 7th, 1893. Higgins: *Treatment by Casarean Section*, *Boston Medical and Surgical Journal*, 1902, p. 6. Hofmeier: *Bipolar Version as Treatment*, *Zeitschrift für Geburtshilfe und Gynäkologie*, 1882, Band viii., p. 89. Jungbluth: *Sponge Tents as Treatment*, *Centralblatt für Gynäkologie*, 1884, Band viii., p. 38. King: *Statistics of 240 Cases*, *American Journal of Obstetrics*, 1880. Legroux: *Source of Hæmorrhage*, *Archives Générales de Médecine*, 1855, vol. ii., p. 641. Macdonald: *Rupture of the Uterus in Placenta Prævia*, *THE LANCET*, Oct. 6th, 1880, p. 601. Maxwell: *Rupture of the Uterus in Placenta Prævia*, *Transactions of the Obstetrical Society*, 1901. Murphy: *Treatment by Separation of the Placenta*, *THE LANCET*, July 23rd, 1887, p. 200. Obermann: *Treatment by Bipolar Version*, *Zeitschrift für Geburtshilfe und Gynäkologie*, Band xxxii., p. 122. Palmer: *Treatment*, *American Journal of Obstetrics*, 1903, vol. xlvii., p. 294. Pinard: *Use of de Ribes Bag*, *Annales de Gynécologie*, 1891. Sequeira: *Treatment with de Ribes Bag*, *Brit. Med. Jour.*, 1893, vol. ii., p. 519. Shaw: *Treatment*, *British Gynecological Journal*, 1895, vol. x., p. 542. Sinnetamby: *Treatment by de Ribes Bag*, *Journal of Obstetrics and Gynecology of the British Empire*, 1906, p. 220. Sligh: *Casarean Section as Treatment*, *American Journal of Obstetrics*, 1892, p. 221. Smyly: *Treatment*, *Brit. Med. Jour.*, 1895, vol. i., p. 62. Spencer: *Diagnosis*, *Transactions of Obstetrical Society*, 1889. Tait: *Casarean Section as Treatment*, *THE LANCET*, Feb. 11th, 1899, p. 364. Welti: *Treatment with de Ribes Bag*, *British Gynecological Journal*, 1897, vol. xiii., p. 236. Wyder: *Treatment by Bipolar Version*, *Archiv für Gynäkologie*, 1887, Band xxix.

Ramsgate.

SOME POINTS IN THE TREATMENT OF HEART FAILURE IN DIPHTHERIA.¹

BY CHARLES BOLTON, M.D., B.Sc. LOND.,
M.R.C.P. LOND.,

ASSISTANT PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL AND TO
THE NORTH-EASTERN HOSPITAL.

THE successful treatment of heart failure in diphtheria depends upon a knowledge of the various clinical forms of this condition, of the different periods during the disease when it may be expected to occur, and of the chief circumstances influencing its onset. These points are all quite easily grasped when the essential facts in the pathology of the disease are thoroughly understood. I will therefore, in the first place, take the opportunity of reminding you of these facts. In former times the treatment of diphtheria was directed solely against the local lesion but the recent study of the pathology of this disease has shown that its fatal character is dependent not upon the local lesion but upon the systemic intoxication which results from the absorption of certain poisons and that this poisoning cannot be prevented by any means directed against the local lesion. The latter is only of itself directly dangerous to life when it mechanically obstructs the larynx. The diphtheritic poison has a selective action and especially

¹ A paper read before the Windsor and District Medical Society on Dec. 20th, 1905.

attacks nervous and muscular tissues. In the acute stage of diphtheritic toxæmia acute degenerative changes occur in certain cells of the central nervous system and amongst these cells the nucleus of the vagus is affected and extensive fatty degeneration occurs in the muscular fibres of the heart. It is thus that acute diphtheritic poisoning proves fatal, the patient dying from a primary progressive failure of the heart as the result of acute degenerative changes in the neuromuscular mechanism of that organ. At a later stage of the disease, if the patient survives, the poison attacks the peripheral nerves and voluntary muscles; a primary parenchymatous degeneration occurs in the nerve and fatty degeneration in the muscle. The vagus nerve may be attacked along with other nerves and an infiltration of round cells may occur in the intermuscular substance of the heart. The conditions which we have to treat are therefore of a degenerative nature and they are, moreover, *acutely* produced, which renders them considerably more fatal than chronic changes of a similar nature. It must also be remembered that not only is the muscular power of the heart affected but that its regulative mechanism is also interfered with. A fact of the greatest importance in treatment is that the degenerative changes in the heart found in the *acute* stage of the disease, if a fatal dose of poison has been absorbed, are of themselves capable of bringing about a primary failure of the heart and death, but that the changes found in the *later* stages of the disease do not lead to death except through the interposition of some strain which the heart is unprepared to meet. The majority of patients, if they survive the acute stage, show some signs of heart failure either in the direction of irregular action or dilatation of that organ. Pathological investigation has further led to the discovery of a specific antitoxin to the diphtheritic poison and thus the treatment of heart failure in diphtheria would seem to be reduced to the simple measure of giving a sufficient dose of antitoxin; but although this is indeed the fundamental principle to be adopted yet it will be seen by a study of the various circumstances in which heart failure occurs that certain auxiliary measures may be adopted, and are indeed necessary, since it is comparatively rare that a sufficient dose of antitoxin has been given before degenerative changes have already occurred in the heart.

I shall first direct your attention to two most important points in the administration of antitoxin. The first point is that it is absolutely necessary to administer antitoxin at the earliest possible moment, since this is a means not of *treating* heart failure but of *preventing* the occurrence of changes which lead to it, and that the earlier antitoxin is given the less chance is there of heart failure subsequently supervening. The administration of antitoxin leads directly to the neutralisation of the toxin. If a fatal dose of diphtheria toxin is mixed in the test tube with sufficient antitoxin to neutralise it and the mixture is then injected into an animal no symptoms result. If less than this amount of antitoxin is used the resulting symptoms are proportional to the amount of toxin unneutralised; in such a case no acute symptoms may supervene but the animal may become subsequently paralysed. The same result is seen even if an additional amount of antitoxin is immediately injected. If the toxin is injected intravenously and if after a few minutes the antitoxin is injected the animal will die unless a very large dose of antitoxin is used. If a longer interval is allowed to elapse the animal cannot be saved. The toxin becomes very rapidly combined with the particular tissue affected by it and is then only neutralised with great difficulty by the antitoxin; after it has produced its effects on the tissues it cannot be neutralised. In human diphtheria the toxin is not suddenly poured into the blood as in an experiment but is formed slowly and therefore it is possible to neutralise it by large doses of antitoxin; it should, however, be borne in mind that extensive fatty degeneration of the heart may be found in three days. From experiment we therefore learn that if antitoxin is to be of any service it must be given at the earliest possible moment. Clinical observation also bears out this point. The statistics of the Chicago health department show that of cases first treated by antitoxin on the first day of the disease the mortality is 0.42 per cent., of those first treated on the second day 1.54 per cent., on the third day 3.59 per cent., on the fourth day 11.38 per cent., and later than the fourth day over 23 per cent. Other statistics are in entire agreement with these, only varying a little in the figures. These statistics include cases dying from laryngeal obstruction and also complications, but the vast

majority died from heart failure and therefore the immense importance of the early administration of antitoxin in preventing this is strikingly manifest. In making a prognosis also in a case of diphtheria, one of the first points to be considered is the day on which antitoxin was *first* administered. I say *first* administered, because a subsequent dose is undoubtedly useful in some cases in which the membrane has shown no signs of separating or goes on increasing in extent. It should be an invariable rule that immediately diphtheria is suspected in a patient and without waiting for a bacteriological examination, antitoxin should be at once administered as soon as it can be procured. This should be done however slight the case appears to be, because although the severity of the local lesion is, speaking generally, a measure of the severity of the systemic poisoning, an apparently mild case may be followed by marked symptoms. We have no idea of the individual susceptibility of the patient to the disease, neither have we any idea of the number and virulence of the bacilli present.

The second point with regard to the administration of antitoxin to which I wish to refer is that it should be given in large doses. I have already mentioned that if antitoxin is given subsequently to toxin experimentally a much larger dose is needed to prevent acute symptoms and death than if the two are mixed together before injection; and moreover, Ehrlich has demonstrated by partial neutralisation of toxin by antitoxin that the latter is much more efficient in averting death in the acute stage than in preventing the subsequent paralysis. Here again clinical observation is in entire agreement with experiment on these two points and it is found that the mortality is lower when large doses of antitoxin are used and also that to modify the subsequent paralysis large doses are necessary. Fixing the dose of antitoxin is difficult but most authorities agree that 4000 to 6000 units should be the minimum dose for an ordinary case. In the severe cases when larger doses are necessary the question arises whether 4000 units should be given at once and repeated at short intervals until, say, 12,000 or 24,000 units or even more have been injected, or whether the larger dose should be given at once. Personally I cannot see the advantage of small and repeated doses over a massive dose. I think that in a severe case of diphtheria 12,000 to 24,000 units should be given at once and if on the next day there is no improvement in the nasal discharge or separation of the membrane, or if the membrane has extended, the injection should be repeated. In the worst cases some clinicians inject up to 30,000 units at once and repeat the injection in 24 or 48 hours. There is no danger in the antitoxin and the complications that are liable to ensue such as rashes, joint pains, and so forth, are not much more likely to follow large injections than smaller ones. Besides the severity of the case as a guide to the dosage of antitoxin, the time at which it comes under treatment is of importance, a larger dose being necessary on the third or fourth day than is necessary on the first or second day. Time may be also saved if the case is seen late by injecting the antitoxin intravenously. In all other cases subcutaneous injection should be employed, as antitoxin is practically not absorbed at all by the stomach or rectum. The dose is the same for children as for adults.

The point next in importance to the early administration of antitoxin is *rest* both physical and mental. By the term "rest" I mean the avoidance of all causes likely to throw any strain upon the degenerate heart. In the treatment of heart failure due to any cause whatever rest is an important factor because in these circumstances the heart has a diminished amount of work presented to it, but in the treatment of heart failure in diphtheria apart from the administration of antitoxin rest is *the* important factor and in the majority of cases it is the only one of any use whatever. In the acute stage of the disease when the heart is failing sudden death may occur at any time as the result of the strain of vomiting or jumping up suddenly in bed. If the acute stage is survived and the patient left with signs of heart failure due to changes not in themselves sufficient to kill him strain may again determine sudden death. On the other hand, if he have no signs whatever of heart failure the strain of getting up too early in the progress of the disease often initiates it, thus bringing into evidence the latent heart weakness. Causes leading to heart failure in the later stages of the disease are mental excitement or shock, sudden exertion, vomiting, and respiratory paralysis. The point which I wish to emphasise here is that in the vast majority of cases after the first three weeks of the disease have

passed death from heart failure will only occur as the result of some complication or strain and that therefore we have at hand a ready means of obviating this to a large extent.

From the foregoing remarks it would appear that rest is a more powerful therapeutic agent after the acute stage has passed than when it is present, because patients are naturally always kept in bed during this stage of the disease and heart failure leading to death at this time occurs independently of any external circumstances. And it is a fact that the key to the successful treatment of heart failure in diphtheria is antitoxin to prevent death in the acute stage and rest to prevent death in the later stage, after antitoxin has done what it is able to prevent cardiac degeneration. There are, however, borderland cases in which the presence or absence of strain in the acute stage may mean the death or recovery of the patient. Every case, therefore, however mild it may appear, is to be strictly confined to bed during the period of acute symptoms. A careful watch must be kept upon the heart and pulse and since the latter varies from hour to hour when heart failure has commenced it must be frequently examined during the day and if possible during the night, or else if only casually examined at the morning visit it might appear to be quite normal. In fatal cases the first signs of heart failure commence usually about the end of the first week, in some cases earlier than this, and death occurs with great regularity usually within the first 14 days. The signs to be observed are irregularity and weakness of the pulse, together with undue rapidity or slowing. A slow irregular pulse is of bad prognostic significance. The first sound of the heart is short and feeble, the pulmonary second sound accentuated, and the impulse may be diffuse and slapping. When these signs are present the patient should be kept lying with the head low and disturbed as little as possible. Great care should be exercised in syringing or making applications to the throat, as it is a common report from the nurse that the patient became "very collapsed" after syringing; in such a case as this very active treatment should be discontinued or there will be a danger of sudden syncope. Another danger lies in the strain produced by vomiting. Every medical officer who has had charge of diphtheria wards must have been an eye-witness to cases of sudden death resulting from vomiting. The first case I saw die suddenly was that of a little girl in the diphtheria ward of the old University College Hospital. She was suffering from heart failure during the acute stage and showed no symptoms whatever except a gradual failure of the heart that was progressively advancing. On the thirteenth day of the disease she asked for a little jelly which was given her by the ward sister whilst I was standing by; immediately after swallowing the nourishment she raised herself up to vomit and fell back in a faint; after three or four gasps at intervals she was dead. Great care must therefore be exercised in the giving of nourishment and medicines lest vomiting be excited; medicines as a rule are useless and should rarely be given in the acute stage of diphtheria. The early vomiting in diphtheria is usually independent of food and in my opinion the evidence is largely in favour of its being central in origin; it is extremely difficult to control and rectal feeding should be resorted to, drugs apparently having no beneficial effect on it whatever.

Dr. G. C. Garratt, late of the London Fever Hospital, is greatly in favour of washing out the stomach repeatedly. He claims success by this method combined with the administration of belladonna but it has not yet been sufficiently tried for an opinion to be definitely stated as to its value. I should think it would not be always a simple matter and might entail the risk of syncope, as many children would struggle considerably.

The next point which arises in relation to the question of rest is, How long should a case of diphtheria be kept in bed? We have seen that in fatal cases the heart symptoms commence about the end of the first week or even earlier, and that death usually occurs during the first 14 days of the disease and exceptionally a day or two later than this. In the case of patients who do not die directly from the toxæmia the majority show some signs of heart failure, but these commence rather later than in fatal cases. They commonly begin about the end of the second or during the third week of the disease, although in some cases they may appear at the end of the first week. In these cases when heart signs have supervened prolonged rest in bed is necessary. The general rule, which must be always adhered to, should be that, however mild the case appears to be, the patient should

be kept in bed for three weeks from the onset of the disease, and this is all the more necessary since the throat may have been clear of membrane for a day or two before the heart symptoms, which will lead to a fatal issue, appear. If at the end of three weeks no irregularity of the pulse or signs of dilatation of the heart have appeared the patient may be allowed to sit up for an hour or two on a couch during the day and each day the time of sitting up may be lengthened until the patient is at the end of a week able to walk. All this time the pulse and heart must be carefully watched, because often the effort of getting up is sufficient to bring into evidence a latent weakness of the heart which caused no signs so long as the patient was lying in bed. If any signs of heart failure are brought into evidence by the exertion of getting up the patient must immediately be put back to bed again and remain there until the signs have disappeared.

If the patient has survived the first 14 days and is left with an irregular pulse and probably with his cardiac impulse outside the nipple and a systolic apical murmur audible the question arises, How long is it to be expected that these signs will last? Although, of course, it is impossible correctly to answer this question yet we are not altogether without knowledge on this point. The period during which the pulse remains irregular varies within considerable limits; in the mildest cases it may be only ten days or a fortnight and in the most severe four or five months. I have recently seen two patients who were suffering from heart symptoms following diphtheria. One of them, a child, aged four years, had suffered from diphtheria five months previously and the mother complained that if he got excited or ran about he became blue and short of breath; the pulse was 132 in frequency and quite regular; the cardiac impulse was just outside the nipple in the fifth left intercostal space and the first sound was short. The other case was that of a boy, aged 11 years, who had fainted twice within six months. He had suffered from diphtheria four years previously and at the time of examination the pulse was irregular and intermittent, the heart showing no signs of dilatation. This case shows that at very late periods following the disease a latent weakness of the heart may be brought out as the result of strain. The more degeneration there is produced in the heart the longer will the signs last; therefore in all severe cases showing heart failure the patient and his friends must be warned that prolonged rest in bed will be necessary, possibly for more than eight weeks. I say more than eight weeks, because cases have been recorded of sudden death occurring when the patient has been allowed to get up after being kept in bed two months. I have never heard of death occurring suddenly after the third month, therefore, if at this time the signs still persist, the patient may be allowed to sit up and gradually regain his old habits. The general rule would then be that as long as the pulse remains irregular the patient should be strictly confined to bed but that after the third month, although the irregularity may not have quite disappeared, strict confinement to bed is not absolutely necessary. All signs of dilatation of the heart may have long disappeared before the pulse regains its regularity. It is not always an easy thing to persuade the patient that confinement to bed is necessary, because he has absolutely none of the usual symptoms accompanying a heart lesion, such as dyspnoea and so forth, and therefore will not believe that there is any danger. I have known such a patient receive a wholesome warning in the shape of a fainting attack when he attempted to disobey orders. Besides confinement to bed, sitting up and playing with toys must be forbidden in the earlier stages, the bowels must be made to act easily, the diet should be plain and nourishing, and it is all the better for most patients if the friends can be excluded.

Paralysis may come on whilst the pulse is irregular but it does not affect the prognosis or treatment materially unless the respiratory muscles are paralysed or vomiting commences. The normal effect of inspiration on the circulation of the blood is to aspirate blood into the heart from the extra-thoracic veins and thus aid in the diastolic filling of the right heart; at the same time the circulation through the pulmonary capillaries is rendered more free and hence more blood flows to the left side of the heart and the circulation is thus materially aided. If the respiratory muscles are paralysed the movements are slower and more imperfect and thus an obstacle is opposed to the diastolic filling of both right and left sides of the heart; at the same time this effect

is increased by the tendency of mucus to collect in the bronchial tubes and so prevent free entry of air and free expansion of the lung. Pneumonia may also be present as a complication. A strain is thus thrown upon the heart and at the same time it suffers from partial asphyxia owing to deficient oxygenation of the blood. It follows from this that a heart which was not sufficiently degenerated *primarily* to cause death may *secondarily* do so owing to the extra stress put upon it by respiratory paralysis. Unfortunately, very little can be done for this condition, but one point which should not be forgotten is to raise the foot of the bed so that gravity will assist in draining the lungs and at the same time in keeping the mouth free from saliva which collects when the pharyngeal muscles are paralysed and adds an extra danger by flowing into the larynx and causing asphyxial attacks. Vomiting in association with paralysis acts as a strain to the heart, as in the acute stage of the disease, and may lead to death from heart failure. The best treatment for this is to stop all food by the mouth and to feed per rectum or subcutaneously. Drugs are practically useless in this condition. In addition to paralysis coming on during the course of heart failure the latter may first appear just preceding the paralysis, especially when such paralysis has an early onset, or it may first appear during the course of paralysis. When paralysis comes on before the acute stage is ended it is naturally associated with an irregular pulse, as the latter is so common at this stage. When the heart failure first appears during paralysis, occurring at a late stage of the disease, it is the result of a latent weakness of the heart brought into evidence by the paralysis as shown above, or it is due to degeneration of the vagus occurring as a part of extensive nerve degeneration. Whether isolated degeneration of the vagus occurs has not yet been proved.

I come next to the question of the use of cardiac stimulants and tonics. Our knowledge of the exact mode of action of these drugs upon the vascular system is still imperfect, but it is sufficiently definite to guide us in the selection of particular drugs the pharmacological actions of which meet the requirements of different classes of cases of heart failure. At the outset I may remark that the utility of such drugs in treating heart failure in diphtheria is not very great and some of them are contra-indicated. The action of the majority of the heart poisons with which we are familiar is directed not only towards the heart itself but also towards the inhibitory apparatus of that organ and the toxin of diphtheria is no exception to this rule. Not only, as I mentioned above, is there to be found post mortem in patients dead from diphtheria extensive degeneration of the heart muscle, but also degeneration in certain nerve cells in the medulla. This degeneration is especially marked in the motor nucleus of the vagus about the region where Laborde located the vagus centre in the cat by the needle method of excitation. Clinically, we have evidence that the vagus centre is excited in the attacks of slowing with irregularity of the pulse which are so common in the earlier stages of diphtheria. A slow irregular pulse is one of the most fatal signs in the acute toxic stage of this disease. We have seen the dangers of presenting any extra work to the weakened heart and therefore all drugs which raise the blood pressure by excitation of the vaso-motor centre or by a direct action on the arterioles are contra-indicated and all drugs are likewise to be prohibited which act upon the inhibitory apparatus of the heart. This is precisely the action of the group of drugs of which digitalis is a member. In therapeutic doses digitalis slows the heart by stimulation of the inhibitory apparatus and the ventricles more completely empty themselves and contract to a smaller size by a direct action of the drug upon the cardiac muscle. With poisonous doses the inhibitory action may be more prominent, the rhythm of the heart being very slow and irregular and the ventricle dilating more completely in diastole; this inhibitory action may entirely conceal the muscular action. At a later stage, owing to increased irritability, the heart becomes rapid and passes into delirium. The blood pressure is raised by constriction of the arterioles by a direct action of the drug on the muscular walls of those vessels and probably also by its action upon the vaso-motor centre in the medulla. Whether digitalis invariably causes a rise in the blood pressure seems to be a doubtful point, because in the course of some clinical observations upon the blood pressure in cardiac dropsy I have found the height of the pressure to be unaffected by the administration of this drug, although the heart was slowed and the dropsy disappeared. *Theoretically,*

the employment of the digitalis group of drugs is thus contra-indicated in the early stages of diphtheria when the pulse is slow. Strychnine, which has been employed, is also contra-indicated at this stage because it acts upon the nervous system, slows the heart by vagus inhibition, and raises the blood pressure by vaso motor constriction. *Practically,* I have never known a case to be saved by the use of these drugs and I know of no statistics proving that the mortality of the disease is lowered by their employment. This objection does not hold in the case of the cardiac stimulants which are considered to increase reflexly the force and frequency of the pulse and some of which cause vaso dilatation. Alcohol, ether, or camphor are examples of such drugs, but it is extremely doubtful if their employment is of any value whatever in the early stages of diphtheria. It is becoming more and more recognised that the action of alcohol on the heart is in the direction of lessened efficiency. If the patient has received a fatal dose of toxin, antitoxin if given early will save him by preventing cardiac degeneration, but no amount of cardiac stimulation will do any good whatever if antitoxin fails. Cardiac stimulants are therefore chiefly of use in the later stages of the disease when the weakened heart gives way before some strain, leading to a gradual failure or sudden syncope. A paralysed patient at a late stage of the disease who is cold and almost pulseless as the result of vomiting may be benefited by cardiac stimulation, but such a condition in the first fortnight of the disease leads inevitably to a fatal termination in my experience. I have found the application of heat and counter irritation to the præcordium to be valuable adjuncts to the cardiac stimulation in the later stage of the disease.

In conclusion, I should like to mention a drug which has not yet received a fair trial in the treatment of heart failure in diphtheria. This drug is belladonna. Of course, it is a very difficult matter to test the efficacy of a drug in such a condition as that the treatment of which we are discussing. It could only be done by testing it upon several hundred patients in various doses, carefully noting the effects upon the circulation and comparing the results with those obtained in a similar number of patients at the same stage of the disease and treated without drugs. The action of small quantities of atropine is to paralyse the terminations of the vagus in the heart and therefore to do away with the inhibitory action of that nerve. The heart is accelerated, the systole increased in extent, the diastolic expansion somewhat diminished, and the output of the heart per minute increased. It could not be expected that the severest cases would be saved by this drug but there are many borderland cases which might be considerably benefited by it, especially those associated with a slow pulse.

Lastly, it should be remembered that the mere presence of irregularity of the pulse or of the physical signs of cardiac dilatation is no indication whatever for the use of cardiac stimulants and that strict rest in bed is the chief requisite for such cases.

Devonshire-street, W.

ON EUCALYPTUS OIL AS A VERMIFUGE IN ANKYLOSTOMIASIS.

BY LLEWELLYN POWELL PHILLIPS, M.A., M.D.,
B.C. CANTAB., F.R.C.S. ENG., M.R.C.P. LOND.,

PHYSICIAN TO KASR-EL-AINI HOSPITAL, CAIRO, AND PROFESSOR OF
CLINICAL MEDICINE AT THE EGYPTIAN GOVERNMENT MEDICAL
SCHOOL.

IN the summer of 1905 four male pygmies were admitted into Kasr-el-Aini Hospital under my care for treatment as they were found to be harbouring ankylostomata. They were somewhat anæmic and three of them had enlarged spleen. I proceeded to treat them in the usual way with thymol, though not quite in the full doses on account of their small size and the age of the smallest. The result of the thymol treatment was not good as only two male ankylostomata were expelled from the four. Dr. Looss, the helminthologist to the school, then drew my attention to a method of treatment described by Dr. Hermann of Mons, in Belgium, as employed by him in the treatment of ankylostomiasis in miners, in which eucalyptus oil acted, with the assistance of chloroform, as a vermifuge. I accordingly tried it upon them with the result that the first administration