thesis. One thing is certain. Fully established fibrillation is due in majority of cases to permanently damaged muscle or other structure of the heart. It is therefore unreasonable to assume, as Sir Thomas Lewis does, that once one succeeds in interrupting the circus movement a normal rhythm will be restored. The cause which originally brought about this condition remains potent all the time the abnormal rhythm continues. The disturbance of functional continuity of the heart muscle in some instances may be due to a slight derangement, which may be effectively treated by a simple remedy. In the majority of cases, however, it is probably based on some myocardial trouble. In order to restore the normal rhythm, we would have to repair a damaged muscle, an infinitely more difficult task to perform than the mere interruption of the circus. The discovery of a remedy for this condition does not, therefore, lie in the direction suggested by Sir Thomas Lewis. Only by adopting measures which shall prevent the overstrain of the auricle, we might possibly succeed in making the condition less common than it is now. This is one instance where a great number of tables only served to obscure the issue. If the problem had been approached not only from the electrical but also from the clinical and pathological point of view, the conclusion arrived at would probably have been more in conformity with the actual mechanism of this affection of the heart.

Cardiac Asthma.

According to Sir Thomas Lewis and his co-workers, cardiac asthma³ is due to an acidosis of the blood, caused by defect in renal function. In order to prove this contention the blood and alveolar air of patients exhibiting this defect have been subjected to a very complicated analysis. The experiments, which were of a very difficult nature, have been carried out with great care and skill, by a number of investigators who are experts in their particular line of research. Within narrow limits the paper is certainly interesting. It proves that we may have dyspnoea without an increase in the CO₂ content of the blood, and that we might have an acidosis which is neither due to carbonic nor lactic acid. But it cannot be said, by the widest stretch of imagination, that the authors have succeeded in proving that which at the outset they intended doing—namely, that the acidosis is the *cause* of cardiac asthma. As we shall soon see, acidosis is probably the effect underlying the clinical condition of asthma and not the cause.

Although the paper contains numerous references to pathological and clinical findings, yet this material has not been made use of when the conclusion was promulgated. Here, too, it would be best to keep in mind the clinical and pathological facts which are associated with cardiac asthma. Sclerosis of the coronary arteries is so constant in this condition that it is hardly fair to disregard this fact altogether. Clinically, too, the only conclusion one can arrive at is that cardiac asthma is a condition of acute heart failure. The symptoms are in a great number of cases those of failure. Cases subject to this attack show a great deal of sclerosis and often a high blood pressure. The clinical symptoms and pathological findings suggest the following simple explanation in regard to the causes which gave rise to asthma. The coronary arteries are sclerotic. An increase of pressure in the coronary arteries is necessary in order that an efficient circulation should be maintained in the heart muscle. The high pressure usually associated with this condition is probably compensatory in nature.

Now, when a patient is at rest at night, for well-known reasons the pressure in the coronary arteries is lower than during the time the patient is active. It might therefore fall to a lower level than that necessary to maintain an effective circulation, with the result that heart failure sets in. Is it not a reasonable assumption which conforms with similar phenomena that acidosis is the effect of the heart failure and not the cause? Surely it is a little strange that in such a painstaking investigation it was not attempted to bring the most striking fact of all in connexion with the disease into conformity with the conclusion arrived at.

I refer to the pathological condition associated with this affection—disease of the coronary arteries. Where does this fact come into the scheme of their theory? Why does the prognosis of these cases depend on the condition of the heart muscle?

Their control experiments are quite unconvincing; they compared cases which exhibit only a slight degree of dyspnoea with patients suffering from severe attacks of acute heart failure! In a matter of this sort the degree of dyspnoea is the all-important factor, as they are fully aware, since even in exertion the kind of acidosis depends on the degree of exertion. The matter is of great practical value. If acidosis be the cause of cardiac asthma, all that is necessary is to treat for acidosis. As a matter of fact, the only treatment of value in these cases is the one which concerns itself with improving the circulation in the heart. It has certainly not been proved that cardiac asthma is due to acidosis.

But to return to my thesis. The elaborate tables published by investigators, which give one the impression of exactness and solidity, only serve to mislead one in regard to the value of the conclusions based on them. Vital phenomena are very complicated. There are many links in the chain of a pathological condition; all of them will have to be traced before a true insight into the mechanism of an abnormal function can be obtained. At the best the method of investigation in question can only deal with a fraction of the problem, with a subordinate fact, leaving great gaps unexplored, which are usually bridged over by arbitrary assumptions.

PS.—After this article had been written, some further observations appeared in regard to flutter and fibrillation. They, however, do not affect the criticism as expressed in the above lines.

References.—1. Heart, vol. vii., No. 4. 2. Ibid., vol. vii., No. 4, p. 328. 3. Ibid., vol. v., 1913-14.

ADRENALIN IN THE STOKES-ADAMS SYNDROME.

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In a woman of 49 with complete heart-block, who was under observation for seven days and nights, severe Stokes-Adams attacks were recurring almost every minute. The subcutaneous injection of adrenalin was followed by complete cessation of the attacks within 15 minutes. It seems probable that only in isolated cases will such a beneficial effect be obtained as is here recorded, but a trial should be made of a subcutaneous injection of five minims (0.3 c.cm.) of adrenalin (1 in 1000), and if this fails, larger doses of 10-30 minims should be used. The case was as follows:—

History.—Mrs. H., aged 49, married, was in good health until about July, 1919, when she fainted and fell, cutting her head. Two or three months later she began to have fainting attacks at irregular intervals varying from three to 20 days; in the course of these she collapsed completely with loss of consciousness for a period varying from 30 seconds to three minutes; she has been seen to twitch during an attack, and twice passed water; she has occasionally hurt herself as a result of falling, but has never bitten her tongue or foamed at the mouth. She has felt perfectly well during the intervals. For three months previous to admission to hospital she was free from these attacks; they began to recur, however, on July 6th, 1921, and were accompanied by considerable mental distress and acute pain in the occipital region.

Condition on Admission.—On July 15th she was admitted to the Royal Free Hospital, where she was found to be suffering from frequently recurring attacks, of which the following were the main features in their order of sequence: 1. The patient becomes pale and loses consciousness; during this period, which lasts from 10 to 13 seconds, there is neither pulse perceptible at the wrist, nor impulse at the

apex. 2. In the second phase the pallor is rapidly replaced by a deep red flush with a slightly cyanotic tinge; the radial pulse reappears, and at the same time there are twitching movements involving the face-muscles, hands, and forearms; the legs are often drawn up and occasionally twitch. This period lasts about five seconds, during which consciousness is regained; but with returning consciousness the patient suffers from considerable mental confusion, failing to recognise her surroundings, and being oppressed by a vague sense of calamity. 3. Rapid recovery follows, and for a short period of one and a half to two minutes she is fully conscious and alive to her surroundings, answering questions intelligently; the colour is good, and the radial pulse slow, regular, and of fair volume. The whole cycle of events lasts two minutes, slightly more or less, and is then repeated.

The rate of the ventricular beat as timed by the apex beat and the radial pulse was 26 on admission, and varied very little during the whole stay in hospital; the average rate was 26-30; the lowest recorded was 20 and the highest 36. The pulse was of fair quality and quite regular. Over the large cervical veins a small but distinct pulsation could sometimes be observed at a rate about 70 a minute, independent of the impulse at the apex. The arteries were not thickened; the systolic blood pressure was 154. The heart was not enlarged; the apex beat was in the normal position and of moderate force: a soft systolic murmur was audible over the mitral area. There was no oedema, and none of the usual signs denoting myocardial insufficiency were in evidence. The patient was well nourished, and, except for a small amount of albumin in the urine, which was not constant, there was no evidence of disease of any other organ. The temperature was normal throughout. The Wassermann reaction was negative, and there had been no miscarriages or other evidence of syphilis.

Continuance of Periodical Attacks.—From July 15th to the 21st the periodical recurrence of ventricular standstill with its accompanying phenomena continued at almost regular intervals during day and night, the only important change being a shortening of the period of ventricular activity, which became reduced to 40 or even 30 seconds. Various drugs, including bromides and caffeine, were given without effect; chloral, however, appeared to control the twitchings and to ease the mental unrest. On two occasions atropine, gr. 1/100, was administered, the immediate effect being a definite diminution in the severity of the symptoms without any change in the frequency of the attacks; but the improvement was maintained only during two or three attacks, and was followed by a reaction, with more severe symptoms and enhanced mental distress.

Injection of Adrenalin.—At 10.50 A.M. on July 21st, 5 minims (0.3 c.cm.) of adrenalin solution (1 in 1000) were injected subcutaneously. The accompanying table shows the frequency and duration of the attacks immediately preceding and following the injection of adrenalin. As shown, 15 minutes later the attacks ceased, and there was no recurrence during the remainder of the patient's stay in hospital—i.e., four weeks—except for a slight attack a few hours after the injection. At 5 P.M. on the same day a second injection of adrenalin, 10 minims (0.6 c.cm.), was given subcutaneously. During the evening there was a severe paroxysm of dyspnoea with cyanosis which lasted for five minutes, and a similar paroxysm occurred 12 hours later; in both paroxysms consciousness was unaffected. For three weeks subsequent to the first injection the highest pulse-rate recorded on the four-hourly chart was 32 and the lowest 24 a minute. These rates do not differ from those recorded in the days preceding the injection, and by polygraph between the recurring attacks.

Table showing the Effect of Adrenalin.

10.40 A.M.		10.50 A.M.*	
Ventricle inactive.	Ventricle active.	Ventricle inactive.	Ventricle active.
20 seconds.	40 seconds	25 seconds	40 seconds
25 "	45 "	15 "	45 "
15 "	40 "	25 "	40 "
20 "	40 "	15 "	50 "
20 "	40 "	30 "	40 "
20 "	40 "	23 "	40 "
20 "	40 "	20 "	35 "
20 "	50 "	15 "	35 "
20 "	45 "	20 "	35 "
15 "	40 "	20 "	55 "
		20 "	45 "
		15 "	45 "
		20 "	40 "
		10 "	40 "
		15 "	40 "

* 10.50 A.M.—Inj. adrenalin (1 in 1000), Mv. (0.3 c.cm.), subcutaneously.

11.5 A.M.—The attacks (ventricle inactive) ceased completely.

Discharge from Hospital.—The patient was discharged on August 15th, 1921, free from attacks. During September

the average pulse-rate was 23, the lowest record being 20 and the highest 26. She experienced frequent dizzy attacks and several short faints on two occasions, but no serious attacks until November, when she ceased to attend and was treated at home, apparently for cyanotic attacks—"black fits"—without loss of consciousness. She was last examined on Jan. 12th, 1922, a fortnight after one of these paroxysms. Complete heart-block was again shown on the electrocardiogram. The blood pressure was 150 systolic and 70 diastolic. Radioscopy showed slight general enlargement of the heart and an apex beat just beyond the mid-clavicular line.

Description of Figures.

FIG. 1 reproduces a portion of the polygram obtained on July 19th, 1921. The whole of a Stokes-Adams attack is shown. After five regular radial beats at the rate of 32 a minute, with a jugular curve of complete heart-block, the pulse disappears owing to ventricular standstill. On the jugular curve a few auricular waves (a) are still seen, but they are soon lost on the curve now recording respiration. Fifteen seconds after the onset of ventricular standstill consciousness is lost, and a movement on the lower curve (*) marks the coincident relaxation of the arm. A moment later the respiration becomes stertorous, and its rate increases from 30 to 50 a minute. Then convulsions appear, shaking the hand and neck, which are still connected with the polygraph. Immediately afterwards, and 25 seconds after the onset, the first radial beat returns. The first two or three beats are at a lower rate, but the original pulse-rate is almost reached by the end of the record. Meanwhile consciousness is regained, though the neck muscles are still contracted so that the jugular curve only reappears in the last portion of the record.

FIG. 2 is a direct continuation of the polygram shown in Fig. 1. It illustrates the frequency of the recurring attacks, for the second attack follows the first after a very short interval (35 seconds) of ventricular activity. Unlike the first attack (Fig. 1), the second attack is immediately preceded by some ventricular slowing as seen in the gradual widening of the six antecedent radial beats.

FIG. 3 shows an electrocardiogram taken after the attacks had ceased. It confirms the presence of complete heart-block, which is indicated on the jugular curve of Figs. 1 and 2. The auricular complexes (P) are completely dissociated from the ventricular complexes (R-T), and they are far more frequent. A. 72. V. 25.

Discussion.

The experimental and therapeutic action of adrenalin upon the blood pressure is well known, and the effect upon the heart itself is often considered only in relation to the vaso-constrictor effect. But adrenalin influences the heart both through the sympathetic and through the vagi. In animal experiment there is acceleration of the heart due to stimulation of the accelerator nerves in the heart muscle (the myoneural junction). A secondary (or a relative) slowing of the heart may, however, occur, and this is due to excitation of the vagus centre by the heightened blood pressure. It is evident from the literature that the effect on the heart varies greatly with the dose administered and with the mode of administration, whether subcutaneous or intravenous. In any case it is agreed that slowing does not occur if both vagi are cut; the main cardiac effect is sympathetic, there is acceleration of rate, and there is sometimes an alteration in conduction from auricle to ventricle.

Kahn,⁸ in 1909, recorded with the electrocardiograph complete heart-block produced in a dog by a maximal intravenous dose (0.1 mg.). Both auricular and ventricular rates were concurrently raised, so that no bradycardia resulted. In addition, aberrant ventricular complexes and frequent premature ventricular contractions were recorded. Cullis and Tribe,⁹ in studying the distribution of nerves in the heart of cats and rabbits, found the action of small doses of adrenalin almost identical before and after section of the bundle. There was acceleration with augmentation of the beat both of auricle and ventricle, showing that the ventricle is well supplied with sympathetic nerve-endings. Egmond,⁶ in dogs with experimental complete heart-block, found that acceleration of both auricle and ventricle resulted from intravenous injection of 0.1 c.cm. suprarenin hydrochl. (Höchst). In one observation the ventricular rate increased from 30 to 45 a minute. Routier¹² produced complete heart-block in dogs by direct crushing of the A.-V. bundle, and then injected 1/20 mg. of adrenalin

intravenously. The primary effect, in about 25 seconds, was acceleration of both chambers; and the secondary effect, 20 seconds later, was entire disappearance of the heart-block (déblocage) for 25 seconds. This effect was reproduced three times on the same animal. He refers it to stimulation of the sympathetic terminations in the A.-V. bundle, and noting that clinical cases of complete heart-block are recorded where unblockings spontaneously occurs, he suggests a trial of adrenalin in human heart-block. Hardoy and Houssay⁷ were unable to obtain experimentally the unblocking which Routier recorded. After an intravenous injection of 0.2 c.cm. of adrenalin (P. D. & Co., 1 in 1000) into a dog with crushed A.-V. bundle, premature ventricular contractions appeared, and then both auricle and ventricle were accelerated but without affecting the block. Similarly, Clerc and Pezzi² failed to re-establish by adrenalin the normal rhythm in animals after section of the A.-V. bundle.

Experimental work, therefore, shows that adrenalin accelerates the ventricle as well as the auricle, even when complete heart-block is present. By a moderate dose Routier removed experimental block, and though this has not been confirmed by other workers, there is clinical evidence (as will be seen) that it can occur. In patients with heart-block there are a few recorded observations on the effect of adrenalin. In Danielopolu's and Danulescu's⁴ case of complete heart-block the ventricular rate rose from 30 to 40 within 20 minutes after a subcutaneous injection of 1½ mg. of adrenalin, and from 26 to 45 within 30 minutes after 2 mg. The auricular rate also increased, though without relation to that of the ventricle, and no unblocking was observed. In their case of partial (2:1) heart-block,⁵ 1½ mg. subcutaneously reduced the degree of block so that only an occasional ventricular beat was dropped. At the same time both auricle and ventricle accelerated, the original rate and ratio of A.-V. : 94 : 47 becoming A. and V. 127. Numerous premature contractions, chiefly ventricular, were also produced (cf. Roth¹⁰).

In Hardoy and Houssay's⁷ clinical case with complete heart-block, a subcutaneous injection of 1 c.cm. of adrenalin (P. D. & Co., 1 in 1000) had no effect either on the block or on the rate of the pulse. An *intravenous* injection of the same dose gave rise to disquieting symptoms with great acceleration of auricle and ventricle, yet without change in the block. Similarly, Lutembacher⁹ in a case of complete heart-block saw grave symptoms, including syncope, after an intravenous injection of 2 mg. of adrenalin (Clin). In another patient with complete heart-block, an intravenous injection of 1/10 mg. was followed in sequence by premature ventricular contractions, ventricular tachycardia (cf. Rothberger and Winterberg¹¹) and a ventricular pause with syncope. The pulse remained at 16 a minute for a week after this injection, one-half its original rate before any medication. Arrilaga¹ obtained acceleration of both ventricle and auricle in six cases of complete heart-block by subcutaneous injection of 1 mg. of adrenalin, but no modification in block was observed on the electrocardiograms. Strisower¹³ obtained a positive result in a man of 82 with complete heart-block. After a subcutaneous injection of 0.5 c.cm. of adrenalin (1 in 1000), the ventricular rate rose from 28 to 84 in half an hour, and next day the electrocardiogram showed a normal rhythm. A month later, when 2:1 heart-block was present, a smaller dose (0.25 c.cm. of 1 in 1000 adrenalin) doubled the ventricular rate and restored the normal rhythm, though only for a few minutes on this occasion. This author refers the effect to sympathetic stimulation, while suggesting that improved coronary circulation as a result of the adrenalin may also be a factor.

This clinical evidence shows that it is possible for partial block to be reduced, and for even complete heart-block to be abolished, by subcutaneous injection of adrenalin; though often it fails to modify conduction, as one might expect from the nature of the pathological lesion usually present in such cases. An

FIG. 1.

A polygram showing the whole of a Stokes-Adams attack. Complete heart-block is present; ventricular standstill produces abrupt cessation of the pulse, lasting 25 seconds. After 15 seconds consciousness is lost, and after 22 seconds convulsions occur. The returning radial beats only gradually regain the original rate of 32 a minute. The auricular (a) and carotid (c) waves on the upper curve are dissociated.

FIG. 2.

A polygram, the direct continuation of Fig. 1, showing the onset of a second attack 35 seconds after the first. The radial pulse gradually becomes less frequent before it disappears (cf. Fig. 1).

FIG. 3.

An electrocardiogram, obtained after the attacks had ceased. Complete heart-block. P = Auricular systole; R-T = Ventricular systole, A., 72; V., 25.

increase in ventricular rate, however, is usually obtainable despite the block. This alone is a sufficient ground for an extended trial of adrenalin in Stokes-Adams attacks, where the immediate cause of the loss of consciousness is usually extreme ventricular slowing and standstill. In the case here recorded we are not in a position to state that any change in the complete block resulted, nor yet that the ordinary ventricular rate was accelerated. But the ventricular standstill determining the seizures was apparently abolished by its influence.

References.—1. Arrilaga: Abstr. Arch. des Mal. du Cœur, 1920, xiii., 370. 2. Clerc, A., and Pezzi, C.: Journ. de Physiol. et de Path. génér., 1919-20, xviii., 965-975. 3. Cullis, W., and Tribe, E. M.: Journ. Physiol., 1913, xlv., 141-150. 4. Danielopolu, D., and Danulescu, V.: C. R. Soc. de Biol., 1916, lxxix., 861-4. 5. Ibid., pp. 105-7. 6. Egmond, A. A. J. Van: Archiv f. d. ges. Physiol., 1913, cliv., 39-65. 7. Hardoy, P. J., and Houssay, B. A.: Journ. de Physiol. et de Pathol. génér., 1917-8, xvii., 605-611. 8. Kahn, R. H.: Archiv f. d. ges. Physiol., 1909, cxxix., 379-401. 9. Lutembacher, R.: Arch. des Mal. du Cœur, 1920, xiii., 345-353; Presse méd., 1921, 145-6. 10. Roth, O.: Deutsch med. Wochenschr., 1914, xl., 905-7. 11. Rothberger, C. J., and Winterberg, H.: Archiv f. d. ges. Physiol., 1911, cxlii., 461-522. 12. Routier, D.: C. R. Soc. de Biol., 1915, lxxviii., 375-7; Thèses de Paris, 1915, lxiii., 13. Strisower, R.: Wiener klin. Wochenschr., 1920, xxxiii., 269-271.

THE SUCTION DIFFICULTIES OF YOUNG INFANTS.*

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1. *The Infant as a Suction Apparatus.*

IN breast-feeding the main difficulties in the first weeks of life are mechanical. Only when the natural diet is left and unnatural substitutes are employed do we meet with primary inability to digest. The young baby has but one function. It acts as a sort of suction apparatus, the efficiency of which varies for many different reasons. I propose to discuss with you some of the causes which commonly interfere with effective suction, and to indicate how I think they may be controlled. In the later months of lactation maternal factors play a more and more important part. In the first establishment of the milk, in the all-important first weeks of lactation, the factor which decides between failure and success is the suction power of the infant. With persistent and regular suction which completely empties the breast the yield increases, with inefficient suction and incomplete emptying of the breast it declines. If two infants are nursed the amount secreted is double. If a tiny premature child is placed at the breast of a wet nurse which till then have been secreting a litre of rich milk in response to the vigorous suction of an older child, within a few days the amount and quality of the milk has adjusted itself to the lesser demand. The infant wins its own sustenance by its own efforts. In these early days the mother's part is confined to the provision of a nipple sufficiently well-formed to permit of the secure attachment of the suction apparatus. So formulated the problem sounds easy enough. That it is in reality very difficult is shown by the frequency with which failure is encountered among mothers anxious to nurse their babies.

As medical men, we can have no doubts as to the dangers of artificial feeding, but in the nature of things we cannot expect mothers to be equally convinced. The danger is, of course, a diminishing risk as the age of the child increases. Profound intolerance for all sorts of artificial food is in general found only in young infants under 3 months of age. Anyone who extols a particular method of artificial feeding must show its value in the very young infant. The tolerance of older children for all sorts of food may be very high. But even among very young infants the artificial diet will only occasion disaster in a comparatively small number. Nine out of ten will pass through the danger without distressing disturbance, one only will go in

risk of its life. The suction difficulties of breast-fed infants are so frequent that in these cases the abandonment of the breast and the substitution of the easily flowing bottle will in the great majority of instances appear to have achieved a striking success. It is not surprising that the mothers of such infants, ignorant of the risks that have been run, add themselves to those who denounce breast-feeding as a troublesome and unsatisfactory process. It is very important that we should detect the cause of the failure of suction power and, if possible, remedy it.

As a suction apparatus the faults of even a healthy new born infant are obvious enough. Suction is apt to be halting, feeble, and incoördinate. And this at a time when the breast is most stubborn and difficult. The strong, effective suction of an older child is not achieved at once. Aerophagy and distension of the stomach with wind is a very common result of these early and ill-controlled efforts. The mother's device of holding the child upright—as can be well seen on X ray examination—brings the air in the stomach immediately under the œsophageal opening and makes possible eructation without regurgitation of the milk. Wind is one of the results of incoördinate suction, though here again the tendency has been to explain it as due to some fault in the quality of the milk secreted. Older children at the breast only suffer from wind when their suction has been too eager and excited, as often happens when they are put to the breast after crying.

2. *General Management of Suction Difficulties.*

For the first month of life we must often be content to wait in the confident expectation that the child's dexterity in suction will rapidly increase. If progress in this respect is apparent a stationary weight curve need occasion no alarm. I believe that to supplement the breast by artificial food is, under these circumstances, the last resource to be adopted and not the first. If food is given from a bottle the child takes the line of least resistance and ceases to apply itself whole-heartedly to its difficult task. Mixed feeding, a device often highly successful in later lactation, seldom has any other end but complete weaning when adopted as an attempt to overcome the suction difficulties of early lactation. To be successful it should result in the re-establishment of breast-feeding. The baby must be put to both breasts at three- or four-hourly intervals. After the infant has done its best, the breast pump should be used vigorously and the milk so expressed should be given with a spoon. The position in which the child is held is of great importance. It is essential that the mother's body should be inclined forward at such an angle that the breast is pendulous and that the nipple falls directly towards the child's mouth. The infant should not be held up to the breast while its body is hitched in this direction or that by the anxious efforts of the mother to assist it to get a better hold. It is best for the child to lie on a pillow, laid on the mother's knee, as she sits on a low stool or chair, the seat of which slopes a little downwards from before backwards. With one hand the mother steadies the head, with the other she moulds the breast so as to make the nipple fall towards the child's mouth. There is generally need for extra warmth. Until the milk-supply is adequate it is a function of all mammalian mothers to approximate the unstable temperature of their young to their own, by the close contact of their bodies. Hot bottles may be necessary and clothing which lies close enough to prevent the access of cold air to the air space around the infant. Anointing with olive oil and bandaging the limbs over cotton-wool will sometimes assist in preventing a too great loss of heat and in preserving the body weight. Water should be given freely.

3. *Specific Causes of Suction Difficulties.*

It has been said that the difficulty of suction in the newly born is often merely a transitory want of precision and expertness, which in the nature of things is soon overcome if patience is exercised for a few

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