food the lacteals, of the dog for instance, are to be found conspicuously injected with milky chyle. In the rabbit, when in a vigorous but not when in a poor or sickly state, fed on oats and taken at the proper time afterwards, I have seen the lacteals about as fully injected with milky chyle as in the dog after fatty food. Oats in their dry state contain about 5 per cent. of fat, which I am convinced upon full consideration of the matter is altogether inadequate to account for what is observed. The extent to which the villi under the circumstances are charged with fat globules I have represented by photo-engravings from microphotographs in my work on *The Physiology of the Carbohydrates*. It is admitted that animals are fat producers from carbohydrate, and with its production in the villi it reaches the system and subsequently passes on in precisely the same way as fat derived from without. In the one case we have to deal with fat emanating from a ready-formed supply, and in the other with fat formed by the protoplasmic agency of the cells of the villi, for it is not for a moment contended that what occurs is of the nature of a mere chemical transformation. As carbohydrate may happen to be here applied to fat production, so have we a provision in the direction wanted for preventing its reaching the general circulation as sugar. There are grounds also for believing that a further formation of fat is effected by the protoplasmic agency of the cells of the liver.

That carbohydrate can be turned to account in contributing to proteid production is demonstrated by the growth of the yeast cell in a medium containing no other source than sugar for the carbon constituent of its protoplasm. Further, carbohydrate is susceptible of being cleaved off from the proteid molecule. The proteids entering into our constitution do not enter the system in a ready-formed state. The proteid matter of food is as a first step towards its application to the purposes of life broken up by the ferment agency of digestion. The absorbed products of digestion then fall into relation with the living protoplasm of the cells of the villi. Peptone, which is recognisable previous to the occurrence of absorption now becomes lost sight of, and in view of all the circumstances existing it may be taken as reasonably permissible to conclude that through the instrumentality of protoplasmic action an extensive building up of proteid goes on in the villi.

By synthesis into proteid carbohydrate matter is placed in a position to be susceptible of transport through the system without running off with the urine. At the same time it is evident that its liberation from the locked-up state into a free form can be most easily effected in the presence of suitable conditions. The lactose of milk cannot, reconcilably with our collateral knowledge, be conveyed as such to the mammary gland, but must constitute a cleavage product resulting from the effect of—may it not be said—ferment agency existing within. In the grave form of human diabetes, as well as in the experimentally induced phloridzin and pancreatic diabetes, sugar is drawn from a source other than the food, and the large quantity that can be eliminated testifies to the abundant store of locked-up carbohydrate that must exist ready to be set free when the requisite agent for effecting the purpose is present, just as the sugar in amygdalin is set free in the presence of emulsion.

What has been said of the villi gives them a position of great importance in relation to the assimilation of food; a process that may be naturally looked for to immediately follow absorption. At a period of fasting, the amount of sugar in the portal blood practically stands in accord with that in the blood of the general circulation. After the copious ingestion of carbohydrate food, the amount of sugar in the portal blood rises, and I have known it reach as high as between 4 and 5 per 1,000. The circumstances are such as to lead to sugar being absorbed in too large a quantity to be fully assimilated or disposed of in the villi, and the portion that has failed to be assimilated reaches the portal blood and gives to it a fluctuating condition dependent upon the food. If there were no further provision existing for the purpose of assimilating this sugar and checking its passage into the general circulation, we should be thrown into a more or less pronounced glycosuric state after every meal, in precisely the same way as, in fact, occurs with the subject of alimentary diabetes. The liver, however, intervenes between the portal vein and the general circulation, and here a supplementary

action is performed, which, if complete in checking the flow of sugar onwards, protects the contents of the general circulation, and hence the urine, from being influenced in relation to sugar by the food ingested. If the supplementary action is incomplete, the blood and urine become influenced, and in proportion to the incompleteness so is the extent of glycosuria. The entry of sugar into the general circulation constitutes the unnatural and not the natural occurrence. In order that we may be kept free from glycosuria, sugar must be kept out of and not thrown into the general circulation.

(To be concluded.)

CHLOROFORM AND THE HEART.

BY

LEONARD HILL, M.B., and HAROLD L. BARNARD,
Lecturer on Physiology, London M.S., F.R.C.S., Surgical Registrar,
Hospital: Grocers' Research London Hospital.
Scholar.

IN the BRITISH MEDICAL JOURNAL of July 17th, 1897, Surgeon-Lieutenant-Colonel Lawrie made the following remark: "There is not one tittle of evidence of any kind, still less of physiological proof, of paralysis of the heart in the cases he describes." These "cases" were instances of sudden death in dogs occurring early in anaesthetisation, and often after severe struggling. In this article we propose to provide the proof Surgeon-Lieutenant-Colonel Lawrie seeks.

We have repeatedly opened the chests of animals dying at this early stage of anaesthetisation, and have invariably found the heart distended with blood, either motionless or more frequently making futile beats. These beats are confined to the auricles and base of the ventricles, and are quite incompetent to empty its cavities.

There is nothing so convincing to the observer as such simple and direct observation, but for the demonstration of the fact in a paper and to the public it is necessary to employ some graphic method. This fact we shall now demonstrate in the accompanying tracings.

Our method has been to open the thorax of a cat,¹ after establishing artificial respiration. The heart was then placed in a simple plethysmograph similar in principle to the cardiograph employed by Roy and Adami. Our plethysmograph was constructed out of an indiarubber tennis ball. A hole sufficiently large to admit the heart was cut in one side of the ball, and in the opposite side a glass tube was inserted. This tube was connected in its turn with a recording tambour. After the heart had been inserted into the ball all leakage was prevented by drawing the flaps of the divided pericardium tightly around the ball and tying them by threads to the tube. We made the junction quite airtight by packing thick vaseline both inside and round the outside of the pericardium. By this means all leakage was prevented and the apparatus recorded perfectly the changes of volume in the heart.

In the tracings an increase in size of the heart is represented by a rise in the base line, and the amount to which the heart emptied itself at each systole is shown by the amplitude of each downward excursion of the writer.²

A word, too, must be said as to the method of administering the drug. We injected pure chloroform into a cannula tied into the external jugular vein, and then washed it well into the circulation with a drachm or two of normal saline solution. This latter point is no unimportant detail, as unless it is done the drug remains in the cannula or vein and no effect is produced, as we have repeatedly observed.

In Fig. 1 we have a record of changes in the heart following the injection of small and large doses of chloroform. At the point A 1.5 minim of pure chloroform was injected into the vein and washed in with saline. After a brief stimulation it will be seen that even this small amount produced between A and B a diminution in the output of the systole and slight dilatation of the heart. At B 5 minims of pure chloroform were injected. Instantly profound dilatation followed, and the systole, though increased for a moment, was almost at once greatly reduced. For the next one or two minutes the heart remained dilated and feebly pulsing. During this condition we have in some cases opened the plethysmograph and have directly observed that this pulsation was restricted

Fig. 1.—Plethysmographic tracing of a cat's heart; chloroform injected into external jugular vein. A, 1.5 minim of chloroform injected; B, 5 minims of chloroform injected; C, heart recovered from distension.

Fig. 2.—Plethysmographic tracing of a cat's heart; ether and A.C.E. injected into external jugular vein. A, 5 minims of ether injected; B, 4 minims of A.C.E. injected; C, stimulation of alcohol and ether passed off; D, heart recovered from distension.

to the auricles and the bases of the ventricles. The blood merely regurgitated in and out of the great veins.

In the tracing it is seen that after a time the effect of the chloroform passed off and the systole became more and more effective, whilst the dilatation steadily diminished until once more the trace reaches its former level and proceeds as before. The space between B and C represents 5 minutes 30 seconds.

In this experiment we were fortunate in administering a maximal non-lethal dose, but in several other traces of experiments in which the heart did not recover the trace rose in an exactly similar manner, but remained at the same level until the last feeble efforts of the heart gradually died out.

In Fig. 2 is shown the action of ether and A.C.E. for contrast with Fig. 1. At the point A, 5 minims of pure ether were injected into the external jugular vein and washed in with normal saline. This produced great but transient stimulation of the heart accompanied by a moderate degree of dilatation. Slight dilatation with increased beat persisted for a minute or so. This is in marked contrast to the diminished beat which follows chloroform (Fig. 1).

At the point B, 4 minims of A.C.E. were injected. The stimulation of the heart due to the ether and the alcohol is clearly shown, and it terminates at the point C, from which point the effect of the chloroform still persisted, and the dilatation tardily passed off.

We may add that a trace of the action of alcohol taken by the same method shows that this drug dilates the heart more than ether, but it stimulates its beat a little less. Its action persists about twice as long as ether but nothing like so long as chloroform. This effect of alcohol has previously been demonstrated by Newall and Smith.

It may not be out of place to restate the conclusions to which our experimental researches on the action of chloroform have led us. Chloroform acts upon three parts of the economy—(1) the heart, (2) the respiratory centre, (3) the arterioles. By its action on the arterioles chloroform does not produce directly fatal results, but inasmuch as it dilates these, it lowers the blood pressure and produces anæmia of the respiratory centre. This is a most important factor in regard to the stoppage of respiration. Failure of arteriole constriction can be remedied by elevating the abdomen or compressing it, and by lowering the head. By these means the supply of blood to the respiratory centre is improved.

The action of chloroform upon the respiratory centre is twofold. It is partly indirect, as we have just stated, and partly a direct action of the chloroform itself upon the centre. Arrest of respiration will be most easily produced when both these conditions of anæmia and chloroform act together on the centre. If the centre be much damaged by anæmia far less chloroform will arrest its action than when it is well supplied with blood.

And both these conditions are present during prolonged anæsthesia. This is the explanation of death from respiratory failure after chloroform has been administered for some time. Partly from the shock of operation, partly from the prolonged action of chloroform on the heart and arterioles, the circulation through the respiratory centre has been ebbing and ebbing, until the centre is no longer able to bear up under the degree of anæmia so produced, combined with the depressing effects of the amount of chloroform which happens at that moment to be administered. Thus the respiration ceases.

But fortunately we can reproduce artificially all the functions of respiration. The lungs can be ventilated; the chloroform thus escapes from the body; the blood is drawn into and driven from the thorax by artificial respiration. When the blood pressure rises in the respiratory centre, and the chloroform circulating in the blood disappears, then the centre recommences work. Cessation of respiration late in an operation is very seldom a fatal event. This is borne out by statistics, and must be the experience of every clinician.

But the action of chloroform upon the heart is a much more serious matter. When chloroform dilates the cavities of the heart, the power required to empty them increases as the cube of their radius, yet at the same time the power of the poisoned muscle is directly diminished. There are thus two factors acting in the arrest of the heart: (1) the distension of its cavities and (2) the direct weakening of its muscle. We have shown that when there is great distension a very

small dose of chloroform will be sufficient to arrest the heart. On the other hand, when a heavy dose of chloroform has been administered, it requires but a slight distension to produce the same result. This distension may be produced by accidental pressure on the abdomen, by efforts at artificial respiration, by inverting the patient as recommended by Nelaton. We have opened the chest of an animal and have observed a chloroformed heart to start and stop beating repeatedly, according as we placed the animal vertically feet down and emptied the right heart into the veins of the belly or vertically head down and distended it from them.

Both distension and overdose are present in typical cases of death occurring early in chloroform administration. The patient, often alcoholic, by struggling and holding his breath engorges his right heart. The anæsthetist to limit his struggles pours an excess of chloroform upon the mask. At last the patient gives in and takes deep gasping inspirations of air very highly charged with chloroform vapour. The first rush of blood through the lungs arrives in the left ventricle charged with chloroform, and is distributed by the coronary arteries to a right heart which is still labouring with distended cavities. The heart stops paralysed and dilated. The blood pressure rapidly falls to zero, and this sudden anæmia of the respiratory centre may cause a few gasping inspirations, followed by respiratory paralysis. Nor have we any efficient substitute for the cardiac systole. We can relieve the right heart by sitting the patient up. We may be able to excite the heart to contract by rhythmical compression through the chest wall. In dogs these methods sometimes succeed, but the time is very limited; and the statistics previously quoted by us that out of 41 deaths from chloroform 39 were of this early type show the extreme fatality of this accident.

This is the clinical picture of the danger of administering even moderate doses of chloroform to a distended heart.

The converse danger of distending a deeply chloroformed heart is, we believe, the explanation of those few cases of arrest of respiration late in anæsthesia which do not recover. The respiratory centre has, for the reasons we have stated, ceased to work, but the heart, though weak, is still able to carry on the circulation. But the cessation of respiration excites the anæsthetist to employ vigorous but ill-judged efforts to restore it. Perhaps he inverts the patient, or carries out a rapid and violent artificial respiration. These measures distend the right heart, which stops at once. The circulation is arrested, and this sudden and total anæmia of the respiratory centre excites those one or two solitary respirations which lead to the vain hope that the patient is about to recover.

In our opinion, as long as the heart beats efficiently there is very little risk that respiration will be permanently arrested. The horizontal position should be maintained, though the head may be lowered to encourage the medullary circulation. The tongue should be well drawn out, and an efficient artificial respiration carried out without undue haste or violence. Lastly, ether, alcohol, or strychnine may be injected hypodermically to stimulate the weakened heart.

NOTES.

¹ All operative procedures were carried out under chloroform anæsthesia, whilst observations were made during a constant condition of morphine narcosis. ² The idea of taking cardiographic tracings of the heart was first suggested to us by Mr. C. Wall, B.A., of the London Hospital, to whom we are greatly indebted both for this and other suggestions and criticisms.

A CASE OF CHLOROFORM POISONING IN WHICH NEARLY HALF A GRAIN OF STRYCHNINE WAS INJECTED: RECOVERY.

REPORTED BY SURGEON S. T. REID, R.N.

[Communicated by the DIRECTOR-GENERAL OF THE MEDICAL
DEPARTMENT OF THE NAVY.]

THE patient (an officer on board one of the merchant vessels in Malta Harbour) was found at 6.30 p.m. in his cabin by the captain and second officer of his ship, who noticed a strong smell of chloroform. He was at that time able to stand upright and answer the captain in semi-unconscious condition. He then fell forwards and vomited.