the food or other substances introduced into the mouth. Thus introduction of sand into a dog's mouth evokes profuse secretion of watery saliva; meat provokes secretion of thick, viscid saliva; bread of thin saliva, chiefly from the parotid gland; and so on. The same results can be evoked by showing the dog these substances—i.e., by the psychic reproduction of previous actual stimulation of peripheral sense organs.

In the stomach the psychic secretion, which is so important in inaugurating the digestion of the food, is apparently unaffected by alterations in diet, any variations which may be caused being sufficiently explained by the different digestibilities and therefore different length of stay of the foodstuffs in the stomach. In the case of the pancreas, however, according to Walther and Vasilieff, there is an accurate adaptation of the composition of the juice to the nature of the food, the dog that has been on a bread diet secreting more amyllopsin, while the dog on a proteid diet secretes a larger amount of trypsin or trypsinogen. These results have been controverted by Popielski, according to whom the composition of pancreatic juice is determined solely by the strength of the stimulus which the pancreas receives and further experimental work is still required on the subject. If Walther's contentions are supported it will be interesting to determine whether the adaptation of the pancreatic activity to the nature of the food is nervous in character as imagined by Pawlow or whether the mechanism in this case also is chemical. A very interesting example of adaptation of the pancreatic activity to the nature of the food was described in 1901 by Weinland, who stated that, whereas the pancreatic juice of an ordinary adult dog contains no lactase and is therefore without effect on milk sugar, it is only necessary to feed the dog for some time, a week or more, with milk or with milk sugar in order to determine the appearance of lactase in the pancreatic juice. These experiments of Weinland have been confirmed and amplified more recently in my laboratory by Bainbridge. It is easy to judge of the presence of lactase in pancreatic juice by testing the action of this fluid on solutions of milk sugar and Bainbridge therefore took advantage of this adaptation to determine the mechanism by which it was carried out, and especially to decide the question whether the adaptation is of a nervous or of a chemical nature.

What are the limiting conditions? In the first place the reaction is absolutely specific. Unless the animal is taking lactose in its diet no lactase is ever found in the pancreas or in its secretion. The pancreas of newly born animals, for instance, is quite free from lactase, which, however, makes its appearance two or three days after birth as a result of the milk diet. The production of lactase is not a direct reaction of the pancreas to the presence of lactose in the blood, since subcutaneous or intravenous injection of lactose does not cause the appearance of lactase in the pancreas. The intestinal mucous membrane of all animals, whether on a milk diet or not, contains lactase and has an inverting action on lactose. It might be thought, therefore, that the production of lactase by the pancreas was a reaction to the presence of the products of inversion of lactose in the blood. This was found not to be the case. Subcutaneous injection of galactose for several days was not followed by any appearance of lactase in the pancreas or its juice. Nor was the appearance of lactase due to the increased production of this ferment in the mucous membrane and its escape into the blood. Injection of an extract of mucous membrane rich in lactase, repeated several days in succession, was not followed by any appearance of lactase in the pancreas. Injection of lactose into the duodenum and the subsequent injection of secretin after an interval of one hour were inefficacious in causing the appearance of lactase in the pancreatic juice. For the production of lactase in the pancreas or its juice it is therefore necessary that lactose should act on the intestinal mucous membrane for some time. The reaction is a slow one, like the adaptation in Vasilieff's experiments, and is certainly not due to the stimulation of certain nerve endings in the mucous membrane by the lactose.

Yet it was evident from the results already obtained that lactose must act on the pancreas through the mucous membrane of the intestine. An extract was therefore made from the mucous membrane of the whole small intestine of a milk-fed dog. This was filtered through muslin and about ten cubic centimetres were injected subcutaneously into a biscuit-fed dog once a day for three days. The dog was then

anæsthetised, a cannula was placed in its pancreatic duct, and ordinary secretin was injected. A flow of pancreatic juice was obtained and this juice was found to contain lactase. This experiment was performed eight times and in each case the juice obtained from a biscuit-fed dog which had been injected with an extract of the mucous membrane of a milk-fed dog contained lactase.

Here, then, we have some glimpse into the mechanism of the adaptation of the pancreas to the nature of the food. As the result of injection of lactose some substance which we may call α is produced in the mucous membrane of the small intestine. This substance is carried by the blood to the pancreas and there slowly gives rise to the formation of lactase, which is turned out in the juice when secretion is excited by the entry of acid chyme into the duodenum. We have no knowledge as yet as to the nature of this substance α . All we can say is that it is destroyed at a boiling temperature, since boiled extracts of the mucous membrane of milk-fed dogs do not, when subcutaneously injected, cause the appearance of any lactase in the pancreatic juice of biscuit-fed dogs.

Whether the qualitative adaptation of the juice in respect of its trypsin, amyllopsin, and steapsin is carried out in a similar fashion we cannot as yet say. We hope that an investigation of the mechanism of this adaptation, which is now proceeding, may throw light not only on the factors involved but also on the nature of the substance which is formed in the mucous membrane and has this marked effect on the activity of the pancreatic cells. Involving, as it does, two distinct sets of cells this chemical adaptation to lactose is more complex than any yet investigated and shows the intimate relation which must exist between the chemical activities of very different organs of the body.

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS, WITH AN ACCOUNT OF A CASE SUCCESSFULLY TREATED WITHOUT OPERATION.

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AND

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THE following is a clinical history of the case. The child, a male, slightly undersized but apparently normal in other respects, was born after a tedious and instrumental labour on June 20th, 1904. Nothing in his condition occurred during the first three weeks to comment upon; he appeared to thrive and was fed upon cow's milk and water in the ordinary proportions for a child of that age. Vomiting occurred once or twice but never in any way to cause any anxiety. Unfortunately the child was not weighed at birth. About July 14th the child started vomiting some of his meals but not enough for us to think of changing his food. On the 21st, however, he was sick six times in the 24 hours and was then put on to peptonised milk, lime-water, and dill-water in small doses, from two to four drachms every two hours, and was given a simple alkaline mixture. His weight about this time (July 23rd) was 6 pounds 12 ounces. For the next few days vomiting continued more or less frequently—on one day once and on others two or three times. Nothing was detected upon carefully examining the abdomen and we thought him to be suffering from gastritis; his bowels were constipated. Benger's food was added to his diet and was carefully given but was invariably vomited at once, and after three days' trial this was discontinued. By July 29th he had gained two ounces in weight in six days and on this day as he had not been sick at all for several hours his peptonised milk was slightly increased in quantity and he was given a teaspoonful of water every two hours. Constipation was very troublesome and was relieved by oil enemata. The following day, however, sickness commenced again and continued for several days in spite of careful dieting. "Humanised" milk was substituted for peptonised milk, being given every two hours in five-drachm quantities; this, however, was most unsuccessful as he vomited 12 times within a few hours of commencing it. Valentine's meat juice

in small doses, white of egg, peptonised milk and hot water were all tried but were immediately returned. His condition by now, August 5th, was very grave; his pulse was 140, his temperature was subnormal, his skin was dried up and shrivelled, his extremities were cold and bluish, and the child was wasted to an extreme degree. The tongue was very dry and brown and the bowels were very constipated. He was much too ill to be taken out of his cot and was kept wrapped up in cotton wool. The vomit was always devoid of bile, containing curdled milk and mucus; at times it was intensely acid and sour smelling. Vomiting followed practically immediately the introduction of anything into the mouth—at times being forced out on to the bed as if from a pump. It was never large in amount, unless perhaps it had ceased for a few hours, when everything came up that he had previously taken.

On carefully examining his abdomen the stomach appeared to be dilated and traversing it from left to right distinct waves of peristalsis could be made out following one another in rapid succession at regular and distinct intervals. These seemed to be excited by the introduction of anything into the stomach and often started at once when the abdominal wall was exposed to the air. Sometimes they could only be obtained by palpating the abdomen or flicking it slightly with the finger. They could also be felt when the hand was laid upon the stomach moving across the abdomen. We were fortunate to obtain a photograph (Fig. 1) which

FIG. 1.

Reproduction of photograph taken in September, 1904, showing peristaltic wave.

distinctly shows this characteristic symptom. On rare occasions we saw a retrogressive wave which evidently started from the pylorus and travelled towards the cardiac end of the stomach; it was noted that it was often accompanied by vomiting. Any food that he was given was in very small quantity—from one to two drachms—and at intervals of from one and a half to two hours day and night. On August 5th he had seven or eight tonic convulsions. On the following day he had 22 and was in a state of extreme collapse, taking no nourishment by the mouth. The pulse was 118. On the 7th and 8th the convulsions were very frequent—from 22 to 24 in the 24 hours. He was now so ill that beyond keeping him warm with hot-water bottles and a teaspoonful of hot water at frequent intervals we did nothing for him. The temperature was 97° and the pulse had fallen to 67. During the next few days, as the convulsions had diminished much in frequency and severity, we again tried him upon peptonised milk and white of egg. Vomiting, however, started at once and on August 10th we commenced to give him injections of normal saline solution, about half a pint twice daily and at first doing nothing else; he retained them and was much less restless, having no convulsions and only a little sickness. By the 14th he appeared to have rallied somewhat, but was still sick three, four, and nine times during the 24 hours. He was now taking peptonised milk and water and occasionally a few drops of Valentine's meat juice and white of egg, a portion of which he certainly retained. His weight on this date was 5 pounds 12 ounces, being a loss of 1 pound 2 ounces in 14 days. He still retained the saline injections, the constipation having to be relieved by an oil or simple enema. He was taking no medicine. On August 18th his weight had further fallen, being 5 pounds 10½ ounces. The temperature was subnormal, the pulse varying from 82 to 64 during the 24 hours; there had been no further convulsions

but the vomiting was as bad as ever, starting immediately anything was introduced into the stomach and occurring as often as seven, nine, 17, and 20 times during the 24 hours. At this time his local condition was much as before but we were able upon two or three occasions to define a small rounded swelling situated to the right of the umbilicus under the costal margins of about the size of a small walnut. Often, however, we were unsuccessful in demonstrating it. A wet nurse was obtained and the child was fed by means of a spoon with her milk but this was a complete failure. The weight now (August 24th) was 5 pounds 8 ounces, the temperature was subnormal, the pulse was 62, and the child looking as if he were about to die at any moment, more shrivelled up and emaciated than ever. He retained the saline injections and nothing else. We had tried a nutrient enema of peptonised milk on several occasions but it had started diarrhoea and was retained for a very short time only. We finally gave up everything except teaspoonfuls of hot water and the normal saline injections and the child remained thus for a day or two when we again commenced teaspoonfuls of peptonised milk and water, which in spite of vomiting two or three times in the day was partly retained. He seemed now, if anything, brighter and upon being weighed we found that he had gained three and three-quarter ounces in three days. The nurse on August 30th started massaging the abdomen twice daily with cod-liver oil, particular attention being paid to massage of the stomach wall towards the pylorus. On Sept. 2nd he had not been sick for two days and weighed 5 pounds 13½ ounces. He was still taking only small quantities of peptonised milk and water and upon again trying nutrient enemata we found that with a little difficulty they were retained. We gave nothing but peptonised milk and continued this treatment for ten days during which he commenced decidedly to improve, his weight being 6 pounds 2 ounces, and except for some slight oedema of his feet and over one eye he certainly looked better. On Sept. 12th we had to stop the nutrient enemata owing to the local irritation they caused but we were enabled still to continue the normal saline injections which, together with an increased amount of peptonised milk by the mouth, given at more frequent intervals than two hours, was the treatment for the next four weeks. The local condition had not altered much. Possibly the peristalsis was not so distinct. We certainly were unable to feel the pyloric tumour that we had previously noticed. It was now unusual for him to be sick more than once a day. On several days there was no vomiting. Abdominal massage with cod-liver oil was still continued. On Oct. 1st his weight was 6 pounds 3¼ ounces. On Oct. 5th Dr. Eustace Smith, in an interview with one of us, recommended the addition of a few grains of pulv. papain compositus to each meal which now consisted of peptonised milk and Benger's food. This was practically the treatment for the next three months. His weight gradually increased and the child began to look much fatter. The only difficulty was that of constipation

FIG. 2.

Reproduction of photograph of the same child taken in July, 1905.

which was as bad as ever, being only relieved by the oil enemata. The injections of normal saline solution were discontinued on Nov. 27th when the weight was 9 pounds 8 ounces and the child's age was just five months. During the latter part of October he had an attack of enteritis which yielded in a few days to simple treatment.

By Dec. 25th his weight was 12 pounds. He was taking now only Benger's food with a little barley water. His abdomen had much altered; the peristalsis was present only upon flicking the abdomen. There was rarely any vomiting but still obstinate constipation. On March 30th, 1905, nothing abnormal on examination could be detected. His weight was 18 pounds. On July 1st the child was as in Fig. 2, weighing 26½ pounds and being about 12 months old. He certainly appeared to have an unusual amount of adipose tissue for a child of his age and the upper part of his abdomen looked unduly prominent. Nothing abnormal, however, could be detected. His health is excellent and his appetite is good. He never has any vomiting and has cut several teeth. Constipation, however, still remains a difficulty.

By far the majority of papers and cases that have been published concerning successful cases of congenital hypertrophic stenosis of the pylorus have been written with the idea of showing the advantages of early operative interference, notably the papers by Wachenheim,¹ Cautley and Dent,² Nicoll,³ Thomson,⁴ Stiles,⁵ and others. It has occurred to us that the somewhat detailed account of the history of the above case might prove sufficiently interesting for publication in that the child's recovery was entirely due to careful nursing and dieting. The infant, who is the son of one of us, had the advantage of being continuously under medical observation and during the whole of his illness was under the care of his mother and an exceptionally capable trained nurse, to both of whom the credit of the child's recovery is due. The question of operative interference was frequently discussed during the earlier stages of the child's condition but was abandoned owing to his apparently hopeless condition. We had noticed, too, as the child improved, that two cases had been carefully reported by Batten⁶ and Gardner⁷ in which recovery took place after careful dieting, the patient in the former case, however, unfortunately dying later from an attack of bronchopneumonia. Recoveries have also been reported as having taken place without operation by Ashby,⁸ G. F. Still,⁹ and J. F. Goodhart.¹⁰

Etiology.—The causation of this condition is still uncertain. The chief theories are the following: 1. That the condition is due to a primary hyperplasia of the muscular tissue at the pyloric end of the stomach, an overgrowth due to an error in development (Dent and Cautley¹¹). 2. That the hypertrophy is secondary and results from over-exertion of the muscle, this being due to functional disturbance of the nervous mechanism of the stomach and pylorus (Thomson,¹² Ashby,¹³ G. F. Still,¹⁴ and others). 3. That it seems reasonable to combine the above views so far as to believe that there is some congenital hyperplasia of the pyloric sphincter, and that spasm supervenes on this, and is largely responsible for the symptoms manifested (Rolleston and Crofton-Atkins¹⁵).

Symptoms.—The period after birth at which vomiting, the first symptom, commences varies much—the average is apparently from two to three weeks. In an interesting table compiled by Wachenheim¹⁶ of notes of 35 cases, in seven vomiting started at birth and in the others from three days up to six weeks. In de Bruyn Kop's case vomiting started at birth and the child died at seven weeks with anasarca and fluid in the abdomen.¹⁷ In Rolleston and Crofton-Atkins's case vomiting and convulsions started 14 days after birth and death occurred seven weeks after.¹⁸ The vomiting appears to commence at first once or twice a day and to begin with gives rise to no anxiety, until it is found that treatment and alteration in diet are ineffectual in

checking it. There seems to be no difference as to whether the child is brought up on the breast or is hand-fed. When fully developed the vomiting occurs after every meal, which is often ejected from the mouth as if pumped up. The act does not appear to be associated with any pain. The vomitus is usually acid; free hydrochloric acid has been found in a few cases and in the majority of cases bile has been absent. In our case mucus, curdled milk, and sometimes a little blood were found. Where there is much dilatation of the stomach the vomitus has a frothy appearance (Still¹⁹). Convulsions associated with the vomiting are the result of the absorption of toxic material from the stomach. We noticed that the convulsions shortly ceased after we stopped food by the mouth; it was rarely that any retching was noted. The child's tongue and mouth became dry and parched.

Constipation is a very troublesome symptom; in several cases it alternates with diarrhoea (Wachenheim) and occasionally blood has been found in the stools.

Marasmus.—This is usually progressive: the child rapidly wastes, the skin becomes yellowish-brown, and the tissues dry up and shrivel. In our case the weight fell from 6 pounds 14 ounces to 5 pounds 12 ounces in less than 14 days.

Visible peristalsis and dilatation of the stomach.—This varies very much; in some cases it is not detected at all. When present it is a most characteristic symptom which seems at one time to be excited by simply exposing the abdominal wall in lifting the nightgown, at another by the introduction of food into the stomach or by palpating the abdomen or flicking it with a finger. In our case when it was most distinct, at the time we photographed the child, it was easily elicited by exposure of the abdomen alone; later, as the child's condition improved it could only be obtained by applying a stimulus to the abdominal wall; soon it was often not obtained then, and finally disappeared shortly after the vomiting ceased. Occasionally a peristaltic wave has been noticed returning from the pylorus to the cardiac end of the stomach and usually followed by vomiting.

Palpable tumour in the region of the pylorus.—This is not by any means always detected. It is sometimes not possible to detect the tumour more than two or three times during the illness. Still considers that its "palpability depends on the temporary condition of the muscle at the pyloric end of the stomach and, like an intussusception, it may be felt only when there is some contraction of the muscular coat occurring."²⁰ It is usually not recognised until after several weeks of vomiting. J. Thomson²¹ noticed it as early as the twenty-third day after birth. The temperature is usually subnormal and the urine much diminished.

Treatment.—That a certain proportion of cases of congenital stenosis have recovered without any operative interference is now a generally held opinion but there is no doubt that the majority of observers are strongly in favour of the necessity of early surgical treatment. Cautley and Dent in their exhaustive paper state "that the subjects of the affection under consideration if untreated die at about the third or fourth month," and Wachenheim "considers it an error to continue medical treatment beyond two weeks unless there are very distinct signs of improvement; further than this, all treatment must be surgical as affording the only chance of life, and that not a brilliant one." It is interesting to note the method of treatment adopted in the two successful cases which were not operated upon and of which detailed accounts have been published by Gardner and Batten,²² the child in the former case being fed upon a teaspoonful of whey every 20 minutes, with later the addition of a little malt and meat juice. In Dr. Batten's case nasal feeding, cow's milk and water (2 to 1) three ounces every two and a half hours, was the treatment.

In the treatment of our patient we found, after trying cow's milk, Allen and Hanburys and Benger's food, a wet nurse, and "humanised" milk, all unsuccessfully, that we had the best results from small quantities of peptonised milk at very frequent intervals, together with injections of normal saline solution half a pint twice daily. The latter method of treatment we found of the greatest service in counteracting the great drain upon the tissues the result of the excessive vomiting. We preferred this method to that of nasal feeding as the child was so exhausted. In any future case we might be called upon to treat we should certainly wash the stomach

¹ Wachenheim: The American Journal of the Medical Sciences, April, 1905.

² Cautley and Dent: Transactions of the Royal Medical and Chirurgical Society, vol. lxxvi., p. 471.

³ Nicoll: Brit. Med. Jour., 1900, vol. ii., p. 571.

⁴ Thomson: Edinburgh Hospital Reports, 1896, vol. xxiv., p. 601.

⁵ Stiles: Transactions of the Royal Medical and Chirurgical Society, vol. lxxvi., p. 509.

⁶ Batten: THE LANCET, Dec. 2nd, 1899, p. 1511.

⁷ Gardner: THE LANCET, Jan. 10th, 1903, p. 100.

⁸ Ashby: Archives of Pediatrics, July, 1897.

⁹ Still: Encyclopædia Medica, vol. iv., p. 89.

¹⁰ Goodhart: Diseases of Children, seventh edition, p. 139.

¹¹ Loc. cit.

¹² Thomson: Brit. Med. Jour., Sept. 6th, 1902, p. 678.

¹³ Ashby: Transactions of the Royal Medical and Chirurgical Society, vol. lxxvi., p. 507.

¹⁴ Loc. cit.

¹⁵ Rolleston and Crofton-Atkins: Brit. Med. Jour., Dec. 18th, 1900.

¹⁶ Loc. cit.

¹⁷ Brit. Med. Jour., vol. i., 1897. Epitome, p. 21, paragraph 103.

¹⁸ Loc. cit.

¹⁹ Loc. cit.

²⁰ Loc. cit.

²¹ Thomson: Edinburgh Hospital Reports, vol. iv., p. 116.

²² Loc. cit.

out twice daily as a routine measure. Had we done this we should no doubt have avoided the convulsions which were at one time so frequent and so severe and were evidently the result of the absorption of toxic material from a dilated stomach. Abdominal massage with cod-liver oil was resorted to in despair but from the progress of the case we cannot help thinking that a certain amount is absorbed through the skin and that a certain proportion of the success of the treatment is due to the oil so absorbed.

SUBDIAPHRAGMATIC TRANSPERITONEAL MASSAGE OF THE HEART AS A MEANS OF RESUSCITATION.

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A FEMALE patient, aged 55 years, was admitted to hospital at 11.45 A.M. on Feb. 23rd, 1905, suffering from marked laryngeal obstruction. She had travelled nearly 30 miles by carriage and train, accompanied by her medical attendant. He was surprised that she had borne the journey so well considering that she had been suffering for about three months previously from increasing dyspnoea which became very urgent at times when the patient was excited. For the past three or four days this had been severe, compelling her to acquiesce in removal to hospital. On arrival she was deeply cyanosed, inspiration was much obstructed and accompanied by a very harsh "baa-ing" noise, and expiration was also interfered with. She was quite conscious and able to reply to questions, though with difficulty. No intralaryngeal examination was made. It was thought that the dyspnoea would subside with rest in bed and orders were given to prepare for tracheotomy immediately after 1 P.M., at which time I should have finished with a clinical lecture in the neighbouring ward. At 12.53 I was told that the patient was dying. On the way to the women's ward a nurse met me saying that she was dead. On entering the ward I heard her make a feeble choky inspiratory effort. Her face was very deeply cyanosed, the large veins standing out on her face and neck. This, however, disappeared with striking rapidity. High tracheotomy was performed at once with a penknife and the lips of the tracheal wound were retracted with bent hairpins. Very little bleeding occurred from a small vein at the lower end of the wound in the neck. Artificial respiration was immediately proceeded with but only four or five spontaneous breaths were taken by the patient. There was no pulse at the wrist or cardiac impulse from the first. No cardiac sounds could be heard on auscultation. She was pallid, all reflexes had disappeared, the pupils were widely dilated, the muscles became absolutely flaccid, her lips were pale, and her eyes rapidly assumed a "glassy" look. Various methods of resuscitation, including attempted external massage of the heart, were carried out but all to no purpose.

Four or five minutes after the tracheotomy was done the abdomen was opened in the middle line immediately under the xiphoid cartilage, again with my penknife. There was no bleeding. Inserting two fingers I could easily palpate the outline of the heart between the diaphragm and the ribs in front. It was absolutely flaccid. Massage by intermittent pressure was carried out for about four minutes. The compressions were performed about the rate of 70 to 90 per minute. At the end of every 15 to 30 seconds I abated the movements in order to feel any change in the condition of the heart. At the end of the above period I felt the heart becoming firm gradually. After remaining thus for a few seconds a slight tremor was felt. Then very slowly (from 30 to 40 to the minute) the heart began to beat. The pulsations were at first very feeble. I massaged the heart for a few seconds more and then it beat gradually more strongly and quickly until at seven minutes past one it was beating at the rate of about 90, and more vigorously than since admission. The gradual return of the heart's action was indicated to those around by keeping time with my left hand, the other being used for massage. The colour was observed to return to the face and lips, the eyes lost their "glassy" stare, the pupils contracted, and pulsation of the carotids became very visible.

During all this time artificial respiration had been kept up by compressing the chest. Very soon after the heart began to beat the patient took a rather deep spontaneous breath and from ten to 15 seconds later another, till by degrees respiration became normal though somewhat frequent. Slight hæmorrhage occurred from the abdominal wound which was then partially sutured and a gauze drain was inserted under the diaphragm. When I left the hospital an hour later the pulse was rather weaker and quicker but still wonderfully good, so that I was rather surprised to hear from the house surgeon that the patient died at 3 P.M., having never regained consciousness. The cause of the laryngeal obstruction was found to be a fairly large cancerous growth. Probably at least ten minutes elapsed between the times when the heart stopped beating and when it began again. Crile records the case of a man whose heart stopped for nine minutes and by the combined effect of artificial respiration, infusion of adrenalin, and manual compression of the heart the cardiac action was restored for 31 minutes.

In December, 1902, I had a similar, though not so striking, experience. I was then removing an ovarian cystoma from a woman, aged 60 years. The pedicle of the growth had just been severed when suddenly the patient became pallid, respiration ceased, and it was found that the heart had stopped. As artificial respiration produced no effect I thrust my hand up through the abdomen and found the heart in a similar condition to that in the foregoing case. In this case, however, only two or three squeezes were necessary to set the heart going again and the patient made an uneventful recovery.

Aberdeen.

A PRELIMINARY NOTE ON THE PRESENCE OF PIGMENT CONTAINING IRON IN THE THYROID GLAND.¹

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In the course of a histological study of the blood diseases we have on several occasions noticed the presence of pigment giving the free iron reaction in the thyroid. Unfortunately the thyroid had not been preserved in many instances, but we now propose (1) to describe the appearances in six cases; and (2) to give an account of the pigmentary changes in the thyroid gland of animals after blood destruction by hæmolytic agents.

Methods.—The gland tissue was fixed in sublimated formalin and sections were stained with eosin and methylene blue to show the general structure. To demonstrate iron we placed sections in a mixture of potassium ferrocyanide and hydrochloric acid and afterwards counter-stained them with carm-alum. In carrying out the latter method we were careful to avoid the use of metal instruments and to use glass vessels only.

1. CASE 1. *Pernicious anæmia with marked pigmentary changes in the organs.*—Each of a series of sections of the thyroid gland showed an area of about the size of a pin's head where there was a marked iron reaction. This was found to consist of a group of thyroid cells, most of which were packed with a yellow coloured pigment and were enlarged to about 20 times their normal size. Many of the neighbouring acini showed no pigmentation but here and there throughout the sections were large, irregularly shaped cells full of pigment lying in the fibrous tissue between the acini. All the pigment gave the free iron reaction.

CASE 2. *Pernicious anæmia.*—The thyroid had undergone

¹ Grants towards the expenses of this research were received from the Carnegie Trust and the Mason Fund of the laboratory.