50 mg. per cent. The difference was greatest at the highest point of the curve, and diminished as the blood fell to normal at the end of 90 to 120 minutes. The venous was invariably less than the arterial blood sugar, sometimes by as much as 80 mg. This shows that the normal tissues extract sugar from the circulating blood during the post-absorptive rise in blood sugar.

Diabetics.

The results are given in the accompanying table. In column 3 is shown the difference between the arterial and venous blood sugar in milligrams per cent., and in column

Experiment.		Sugar Percentage.			
		Ear.	Vein.	Difference.	Variation.
1	R.—Fasting blood sugar ... 1½ hours after 15 units ...	0.268 0.224	0.280 0.210	+12 -14	26
2	L.—Fasting blood sugar ... 1½ hours after 25 units ...	0.177 0.146	0.179 0.140	+ 2 - 6	8
3	R.D.—Fasting blood sugar ... 1½ hours after 12 units ...	0.142 0.147	0.148 0.152	+ 6 + 5	1
4	L.—Fasting blood sugar ... 1½ hours after breakfast, no insulin 2 hours after lunch and 15 units...	0.248 0.218 0.187	0.239 0.234 0.178	-9 +16 -9	25
5	L.—Fasting blood sugar ... 1½ hours after breakfast and 15 units	0.237 0.212	0.233 0.201	- 4 -11	7
6	L.—1½ hours after breakfast and 20 units	0.205	0.186	-20	
7	R.—5½ hours after breakfast, no insulin 2½ hours later after lunch and 12 units	0.223 0.136	0.222 0.124	-1 -12	11
8	R.D.—7½ hours after 12 units ... 1 hour later after 30 grams carbohydrate 2½ hours later ...	0.140 0.170 0.221	0.140 0.163 0.222	0 -7 +1	
9	R.D.—7½ hours after 12 units ... 1 hour later after 20 units and 30 grams carbohydrate 2½ hours later ...	0.155 0.187 0.165	0.160 0.172 0.165	+5 -15	20

4 the variation caused by insulin. The cases are all severe diabetics who could not be rendered sugar-free by the Allen treatment. The results lead to the following conclusions:

1. Diabetics have a marked hyperglycaemia in venous as well as arterial blood.

2. The difference between arterial and venous blood sugar is much less in diabetics than in normal persons. As has been said, there is no difference in the fasting blood sugar in normal individuals, and the raised fasting blood levels of diabetics also show little or no arterio-venous difference even at figures of 0.14 and 0.17 per cent.—figures at which normals would show a difference of 30 or 40 mg. This we might expect, as the fasting level of a diabetic, though raised, is comparable in the metabolic cycle with the normal fasting level. But the post-absorptive levels in diabetics also show no arterio-venous difference, or much less than the normal difference. From this we may conclude that the diabetic tissues have lost the power of using or storing sugar brought to them, and the blood flows through with very little change in blood sugar concentration.

3. A venous blood sugar higher than the arterial may occasionally be obtained (experiments 1 and 4 are definite, and 3 and 9 suggestive of this). It would therefore appear that there are times when the tissues are discharging more sugar into the venous blood than they are receiving from the arterial blood; the sugar must have been temporarily retained in excess in the tissues. Some experiments of Foster¹ with galactose are interesting in this connexion. He found that, unlike other sugars, galactose induced in normal individuals both a prolonged arterial and venous hyperglycaemia and a much smaller difference value between the two than any other sugar. Indeed, after ingestion of 100 grams of galactose he got two readings on the decline of the curve in which the venous exceeded the arterial sugar by 18 and 9 mg. respectively.

However, he does not appear to have noticed these figures specially, or at least makes no remarks upon them. But he concluded that galactose, unlike glucose, cannot be absorbed or stored as glycogen by the muscles. These diabetic cases also suggest that the venous sugar may temporarily exceed the arterial where sugar cannot be stored by the tissues. So far this has been observed only in cases being treated by insulin, but at a time when they were not under the effect of insulin; it would suggest that insulin enables the tissues to store more sugar than they can retain when the insulin action has passed off, so that they excrete more sugar into the venous blood than they are receiving from the arterial.

4. In the few other cases of milder diabetes so far investigated the arterio-venous difference has been less than normal, but greater than in severe diabetes.

The Effect of Insulin.

The effect of insulin has been to increase the difference between arterial and blood sugar content, making it approximate in all cases towards the normal relation. In some cases the variation caused by insulin is slight, just outside the range of experimental error, but the variations are constantly in the same direction. Experiments 1 to 3 show effects on fasting blood sugar levels without food. Experiment 3 shows no variation, but the blood sugar also remained practically constant. Experiments 4 to 6 are all strictly comparable, being carried out on the same patient on a fixed diet within four days of each other. Without insulin the venous sugar exceeded the arterial by 16 mg. one and a half hours after a standard breakfast, while after insulin it was respectively 11 and 20 mg. less. Experiments 8 and 9 show after one hour a variation of 20 and 7 mg. respectively with and without insulin, but no variation after two and a half hours.

It is felt that a very extensive investigation will be required to discover at what time and under what conditions insulin shows its maximum effect on arterio-venous variation, as the questions of dose, rate of absorption of insulin and food, and previous diet and carbohydrate stores in the tissues all have to be taken into account. But the results so far obtained seemed to warrant this preliminary communication.

Summary.

1. The normal post-absorptive difference in arterial and venous blood sugar, the venous being usually 20 to 40 mg. less, is much reduced in diabetes. Higher venous than arterial figures may occasionally be found in severe diabetes.

2. Insulin increases the arterio-venous difference and approximates it to the normal. From this we may conclude that, whatever part the liver and other organs play, insulin acts largely in the muscles or general tissues, or both.

Further investigations are being undertaken in the arterio-venous difference to see how it varies in different grades and types of diabetes, in renal glycosurics, etc., in the hope of obtaining a test of value in the diagnosis, prognosis, or differentiation of various types of diabetes and hyperglycaemias.

REFERENCE.

¹ G. L. Foster: *Journ. Biol. Chem.*, lv, February, 1923.

MECKEL'S DIVERTICULUM STRANGULATED IN A FEMORAL HERNIA.

BY

R. M. LITTLER, F.R.C.S.,

HONORARY SURGEON, SOUTHPORT INFIRMARY.

MECKEL'S diverticulum, when present, as it is in one person in fifty, not infrequently causes danger to life by becoming adherent at its tip and forming a band under which the intestine becomes strangulated; or it may itself become acutely inflamed and perforated; or, thirdly, it may form the starting point of an intussusception, but it is most unusual to find it strangulated in a hernial opening, as in the case here recorded, which I consider to be one of great interest and rarity. I have consulted the textbooks

available to me, and in most of them can find no reference to the possibility of such an incident.

On November 16th, Mrs. P., aged 52, was admitted to the Southport Infirmary at 2 p.m., with what appeared to be an ordinary strangulated femoral hernia on the right side, the size of a small Tangerine orange, and very tense. Acute symptoms had begun the day before; a previously existing lump in the right groin had become larger and more painful and she had vomited repeatedly. At 11.30 a.m. on the day of admission she was seen by Dr. Martin, who at once ordered her to hospital.

A vertical incision was made, and the sac was found to contain a fair quantity of dark fluid with a slightly faecal odour. As soon as the tension was relieved, a black elongated mass of an

unusual shape presented, which assumed an almost vertical position and looked in size and shape exactly like a black warning little finger, curving forward at the tip.

As there was no mesentery visible even at the root, I concluded that I was dealing with a Meckel's diverticulum, and as I found it very difficult to insert the usual hernia director, the incision was carried upward and the abdominal muscles divided transversely.

On opening the peritoneal cavity I found a loop of small intestine entering the femoral opening, but it was firmly fixed there, and, as I feared that the

damaged bowel might give way, I finally decided to open up completely by dividing Poupart's ligament.

A good view was thus obtained of the parts, and an area of blackened intestine round the base of the diverticulum about 1½ inches in diameter was seen. The proximal end of intestine was not much distended and the lumen of the bowel was not obliterated. As the patient's condition was good I removed the damaged portion, including the diverticulum, and joined the ends by lateral anastomosis.

The peritoneum was stitched up, and the old sac separated off, the divided ends of Poupart's ligament carefully approximated and united with catgut, and the femoral opening closed as far as possible by suture to the pectineal fascia. The wound was then closed and a rubber drain inserted.

Recovery was uneventful, with the exception of some suppuration in the upper portion of the wound due to infection from the fluid in the sac. The bowels were freely moved the next day.

I have much pleasure in acknowledging the help given me during the operation by Dr. Robert Watson, our junior house-surgeon, and am indebted to Mrs. Cronin Lowe for making a coloured drawing from which the print has been reproduced.

The specimen was sent to Sir Arthur Keith of the Royal College of Surgeons, who pronounced it to be "a Meckel's diverticulum of moderate size strangulated as a hernial content, and the first specimen of its kind which we have got for the museum."

BIBLIOGRAPHY.

- Pabst (*Beitrage klin. Chirurg.*, 1910, vol. 69) summarizes all known cases (over 120) of Meckel's diverticulum in hernia down to 1910; a large number which were not strangulated are included.
 Hauf, in *Deut. med. Woch.*, 1919, reports a case.
 B. Metcalfe (*BRITISH MEDICAL JOURNAL*, 1914, ii, p. 178) reported a case of gangrenous Littre's hernia (inguinal) where the stump was invaginated.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

LABYRINTH DEAFNESS, WITH A NEW PHYSIOLOGICAL DIVISION OF THE AUDITORY ORGAN.

At the time that the discussion on labyrinth deafness was held at Portsmouth, in the Section of Laryngology and Otology, as printed in the *BRITISH MEDICAL JOURNAL* (November 10th, 1923), I presented a similar topic before the Szent István Akadémia in Budapest. The title of my paper was "A new physiological division of the auditory organ." In it attention was directed to the fact that the diagnosis of non-purulent labyrinthine diseases is very difficult, on account of the complexity in differentiating tuning-fork responses as having originated from the conducting or from the perceptive apparatus. This same uncertainty in diagnosing labyrinthine diseases seems to have been emphasized at the British Medical Association discussion held at Portsmouth.

A study of the discussion at Portsmouth on this theme made it apparent that the division into conducting and

perceptive apparatus caused such uncertainty in the diagnosis that the diagnosis of the labyrinthine diseases would be less perplexing if this division was eliminated. To further bring forth the ambiguity mentioned, attention is called to the two following factors:

1. The anatomical border-line position of the two windows causes similar questionable border-line response of the tuning-forks, which thus often leaves a question of doubt whether the responses can be interpreted as being the result of an internal (perceptive) or middle (conducting) ear pathological condition.

Mr. E. B. Waggett, the president of the Portsmouth Section, called attention to the fact that many cases of so-called internal ear deafness would be proved some day to be due to diseases in the foramen rotundum. In an issue of the *Laryngoscope* (St. Louis, July, 1920) I brought forth this last mentioned statement. I, however, limited the value of the tuning-fork tests. The following is a brief summary of this article. The shortening of the higher tones, the shortening of bone conduction (Schwabach), and the positive Rinne all can be caused by a disease of the windows as well as by the affected nerve. Hitherto we have accepted the shortening of bone conduction or the shortening of the higher tones alone as a sign of a nerve disease, although Panse called attention to the fact that the disease of the round window also causes such phenomena. We are only justified in supposing a nerve disease if we have guarantee for the integrity of the windows—that is, if the hearing of the lower tones is good. Bearing in mind this limitation we will not be compelled to say that "the tuning-fork tests are open to fallacies." We will not say that "nerve" deafness may be induced by a simple obstruction in the conducting apparatus. We will not have to suppose so often the mixed type of deafness. I am convinced that we must limit the value of the tuning-fork tests, else we will cause a general dissolution of its worth owing to the scepticism which will arise.

2. The second fact is the construction and the physiological importance of the internal ear proper. It is but necessary to call attention to these factors to realize that the labyrinth is not perceptive in function. Surely the movement of the perilymph and endolymph is conductive rather than perceptive in nature; likewise the vibration of the membrana tectoria is also of this type. Finally, Corti's organ itself, rather than an end-station perceiving organ, is a relay station in forwarding the impulse to the true perceptive factor—namely, the brain.

Mr. W. M. Mollison's effort to simplify this matter by eliminating the old "physiological division," and indexing these diseases under qualitative and quantitative groups, is but of theoretical importance, as it would be impossible to identify for practical purposes these diseases, under his classification.

I propose a new classification, a new physiological division of the auditory organ as follows:

I. The Conducting Apparatus.

A. Physical stimuli transmitted—

- (1) through the external and middle ear;
- (2) through the two windows;
- (3) through the labyrinthine fluid.

B. Physiological stimuli transmitted—

- (1) through Corti's organ;
- (2) through the eighth nerve and its tracts.

II. The Perceptive Apparatus.

Stimuli interpreted in the cortex.

Budapest.

Docent Dr. A. REJTO.

HAEMOTHORAX AFTER ACUTE PNEUMONIA.

In the following case haemothorax arose spontaneously during convalescence from acute pneumonia.

C. J. L., aged 32, an athletic man of good physique, after ten days' persistent "cold" in the head with progressive debility, suddenly, while at work, was conscious of severe stabbing pain under the right pectoral muscles, and in the right side. The pain was so severe that he had great difficulty in reaching his home in the suburbs, and when seen at 10.30 p.m. was found crouching forward in his chair afraid to move his body or take a deep breath.

The following morning at 9 o'clock all the signs of a typical and severe lobar pneumonia were present. Temperature 103° F., pulse 142, respirations 36 per minute. Pain was an extremely