afferent ganglionic system (cranio-spinal), yet I do happen to be the first neurologist to have grasped its wide distribution and its clinical importance in man.

The experimental indications of this subject are clear. By a combination of the retrograde chromatolysis and the degeneration methods the experimental physiologist can settle the points at issue, among which I may enumerate (1) whether all or some of the ventral spinal roots contain afferent fibres; (2) whether any are crossed; (3) whether they rise in the smaller cells of Clarke's column, or the dorsal horn, or the "middle cells," or even in the nucleus cuneatus. Naturally the cervical extension of Clarke's column (C. 3 and 4) and its sacral extension (S. 2 and 3) would be examined histologically in certain cases. There are many other points which I reserve for my full subsequent work.

In concluding this necessarily very incomplete note I wish to say in the clearest fashion that I deny that any motor cells exist in (1) tectum mesencephali, (2) locus coeruleus, (3) Deiter's nucleus, (4) dorsal vago-glossopharyngeal nucleus (ganglion), or (5) in any of the four spinal dorsal-zone cell groups which I have just mentioned. Since all these parts are developed in the dorsal zone of the embryonic brain or cord (the alar lamina of His), and since, further, there is no instance known in any vertebrate of motor fibres rising in cells derived from the alar lamina, I hold that it is nothing less than a morphological outrage to teach that any of these cell groups give origin to motor fibres. I have already explained (*loc. cit.*, February, 1911) that if any experimentalist can demonstrate degenerated fibres of the mesencephalic fifth root (tract) entering the motor-end plates of the muscles supplied by V⁵ a drastic revision of our conceptions of neuro-morphology will be necessary, and we shall then have to teach that the roof and the dorso-lateral walls of brain (and cord) do give origin to motor cells—a view I, for one, resolutely decline to entertain without overwhelming proof of its accuracy. It is, unhappily, true that some of the greatest workers in neuro-physiology have adopted the fundamentally erroneous view that the cells of the tectum mesencephali, dorsal vago-glossopharyngeal ganglion, Clarke's column, and dorsal horn, are, in part, motor. But I can promise that my future complete work will win over all of them to my side. One of them—no less a neurological pioneer than Dr. W. H. Gaskell—at one time (1886) taught that the cells of Clarke's column formed a splanchnic afferent centre, though it is quite true, I think, that he did not even then grasp that it was, in part, a splanchnic endo-neural afferent ganglionic cell-group.

REFERENCES.

- ¹ Abbe, *Boston Med. and Surg. Journ.*, vol. cxxxv, 1896, p. 329.
² Jacoby, *New York Med. Journ.*, vol. xxxvii, 1907, p. 192, ³ Groves, *Lancet*, July 8th, 1911, p. 81. ⁴ Chavaz, *Gaz. hebdom. d. sci. méd.*, vol. xxvii, 1906, p. 369. ⁵ Trendelenburg, *Arch. f. Anat. u. Physiol.*, 1906 (*Physiol. Abtheil.*), S. 1-126, 34 figs. ⁶ Merzbacher, *Arch. f. d. gesammte Physiol.*, 92, 1902, S. 585. ⁷ *Review of Neurology and Psychiatry*, October, November, December, 1910, pp. 594, 673, 749; and also for February, 1911, p. 62.

PANCREATIC CYST: RUPTURE: RECOVERY.

BY

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THE following case presents some points of interest, more especially with regard to diagnosis and treatment, and from a legal point of view:

History.

J. O., aged 34 years, a labourer in iron works, on January 18th, 1909, was pulling on the jib of a crane, to swing it round, when the jib stuck; instead of getting assistance, he persisted in trying to overcome the resistance, an effort generally necessitating the combined action of two or three strong men, and suddenly experienced a very sharp severe pain in the centre of the epigastrium, causing him to sit down, feel faint, and make the remark, "I have pulled my inside out." Shortly afterwards he partly walked, partly crawled, home. The pain was continuous and very intense, and strictly localized to the one spot. He vomited, was very pale, and obviously suffered from shock.

About an hour after his "accident" he was seen by Dr. Falconer, who found him in the condition described, and made a tentative diagnosis of perforation, probably of the stomach.

He was admitted into the North Ormesby Hospital, and I saw him about an hour later. At this time he had acute abdominal

pain, limited to the epigastrium, which was tender to the touch. Both recti muscles above the umbilicus were as hard as boards—below the umbilicus quite soft—pressure did not relieve him. He had vomited several times, and the bowels had not acted. The temperature was 97° and pulse 60. His face was anxious, and he was obviously not exaggerating his symptoms. There was no history whatever of any previous "indigestion," and his past health had always been very satisfactory.

My first thought was in favour of perforation of stomach or duodenum, but the condition of the pulse and temperature more than two hours after the onset of symptoms (although not negating this diagnosis) stayed my hand from immediate operation, and I decided to watch him for another hour or so.

I was guided in this decision by a general consideration of all his symptoms—his facial expression, suggestive of the acute pain associated with colic, his normal temperature, and pulse, and the fact that his abdominal symptoms appeared to be becoming less imperative. I was inclined, therefore, towards the diagnosis of a very severe colic, or, failing this, the leakage of an old gastric ulcer into a localized area of adhesions, securely shutting it off from the general abdominal cavity.

His condition during the next few days in some way justified my decision against immediate operation. The pain steadily decreased. The sickness stopped. His bowels were made to act by means of enemata, and he felt much better. The contraction of the recti muscles gradually relaxed during the first few hours, and the abdomen generally returned to a normal condition, except for persistent tenderness in the centre of the epigastrium. The urine was normal.

Twelve days later he had so much improved, and all symptoms had so far subsided, that I came to the conclusion that my diagnosis of perforation had been quite wrong, and he was allowed light diet.

On February 11th (more than three weeks after the beginning of his symptoms) quite suddenly there was a return of the pain in the epigastrium, and on inspection a swelling about the size of an orange was seen to push forward the anterior abdominal wall in that region.

I operated at once. A central incision was made in the epigastrium over the swelling. As soon as the abdominal cavity was opened the stomach presented in the wound. There was no free fluid in the general abdominal cavity, and no adhesions between the stomach and the anterior abdominal wall. The stomach itself was perfectly normal, but was pressed forward by some soft, elastic swelling underneath. An opening was made through the great omentum into what turned out to be a large collection of fluid. This was withdrawn to the amount of about two pints, a drainage tube inserted, and dressings applied. The fluid was subsequently analysed and pronounced to be pancreatic fluid.

The subsequent history was uneventful. There was no collapse following the operation, and all tenderness and pain immediately and permanently ceased. The cyst cavity drained for twelve weeks before the sinus finally closed, and his health steadily and progressively returned to a robust normal.

A ventral hernia subsequently formed in the scar, but this has since been operated upon and completely cured by the method of overlapping of the structures of the anterior abdominal wall.

The main points of interest, I think, are these:

I. The Diagnosis.

A consideration of the history, severity of symptoms, and physical signs, pointed more towards perforation of some portion of the gastro-intestinal tract than any other condition, even in the absence of any history of previous intestinal disorder. The condition of the recti with the slow pulse and normal temperature favoured an extreme colic.

In either of these, the story of a severe strain would have to be regarded as merely coincident and in no way contributory.

In the light of after-events, when the diagnosis was positively established, the question arises, Under what condition was the pancreatic fluid existent? Had it been previously present as a circumscribed localized cyst of the pancreas, and if so, had this cyst perforated? or had there been a pre-existing cyst which had suddenly taken on characters of an inflammatory nature? If so, could these possibly have been caused by such an accident, or were they due to the spread of an inflammatory process from the duodenum, and so producing symptoms closely simulating colic of a very intense nature?

In the latter case, could this have been caused by, or aggravated by, such a form of abdominal strain, or must they be regarded as merely and purely coincident? Lastly, there was the alternative of a rupture of the capsule of the gland with a gradually increasing accumulation of fluid in its immediate neighbourhood, forming a cyst for itself by antecedent inflammatory adhesions.

The fluid was limited to the lesser peritoneal cavity, as no free fluid had escaped through the foramen of Winslow. An analysis of the fluid subsequently proved that blood

had been present. For two or three days after the onset of symptoms the temperature was raised to 100°-101°.

II. Possibility of Rupture by Pressure.

It is generally supposed that rupture of a cyst of the pancreas can only be produced "artificially" by some direct and violent blow. In this case the force was not of this nature. The man's own statement, confirmed by the opinion of other men present, make it strongly suggestive that the accident, producing indirect force, may have been the cause of the rupture. If not, the coincidence is remarkable; and why should it have occurred at such a time as that when he was subjecting himself to a strain of an unusual nature and degree?

I suggest that it is right to consider, under the very definite circumstances, whether the cause of rupture may not have been due to a severe and violent contraction of all the abdominal muscles, and especially the upper ones, increasing the tension in a previously tight cyst, probably very thin-walled. I recognize the improbability, but we are dealing with a very marked and unusual condition, which, to my mind, is very difficult to explain in any other way.

I have been unable to find any record of a pancreatic cyst being ruptured in any other way than by direct force, neither do I, personally, find it easy to come to any other conclusion, especially when we consider the mechanical conditions of fluid enclosed in a movable, more or less elastic bag. All I state is, that such force was not present in this case—that a rupture of some sort undoubtedly occurred—and that it is very unsatisfactory to dismiss the fact as a mere coincidence, in the face of the various statement of witnesses. The results of suddenly increased intra-abdominal pressure are sufficiently definite in other conditions, such as hernia.

III. Medico-Legal Considerations.

The man naturally claimed compensation; he was not advised to do so, but he thought he would "take his chance." The case came before Judge Templar. Owing to its unusual nature and uncommon occurrence, it attracted some interest, and medical evidence for the man was chiefly based upon the assumption that the onset and severity of symptoms was more than could be accounted for by mere independent coincidence—where a possible cause was so plainly and unmistakably present. No more than this could be said. The cause was described as possible (not probable) unless associated with the proved genuineness of the particular definite circumstances of this individual case.

Medical evidence for the defence maintained chiefly that no such accident was on record, as it was impossible to believe that force of such a nature could produce an accident of such a kind.

No explanation was attempted or offered of the nature of the condition or the cause of the rupture; no opinion as to whether the cyst was pre-existent, became suddenly inflamed, or due to a ruptured capsule of the gland.

His Honour, quite in agreement with the opinion of all fair-minded people present, prompted by his medical referee, Dr. Beatty of Stockton, gave his opinion against the man, his main reason being that no such case had previously been on record, although he did not deny that it was possible for it to occur.

Thus it remains as a coincidence, without any satisfactory surgical explanation.

REFERENCE.

Green's Encyclopaedia, Article on "Pancreas."

THE King has been graciously pleased to confer the Coronation Medal on Dr. W. Allan Jamieson, Surgeon to the Royal Company of Archers, the King's bodyguard for Scotland.

THE following office-bearers were elected at the annual meeting of the North-Western Branch of the Society of Medical Officers of Health: *President*, Dr. Sydney Marsden; *Treasurer*, Dr. H. H. I. Hitchen; *Secretary*, Dr. T. W. N. Barlow; *Librarian*, Dr. M. A. Arnold. A committee of four members was also elected, and Drs. J. J. Buchan and Meredith Young appointed representatives of the Branch on the General Council of the Society. The Branch meets in Manchester on the third Friday of each month from October to June.

ON THE OCCURRENCE OF AN INTRACELLULAR STAGE IN THE DEVELOPMENT OF TRYPANOSOMA LEWISI IN THE RAT-FLEA.

BY

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(Preliminary Note.)

IN studying the early phases of the developmental cycle through which the rat-trypanosome passes in the rat-flea, we have found that the trypanosome penetrates into the cells of the epithelium lining the stomach (mid-gut) of the flea, and there goes through a process of multiplication. Since no such phase has been observed hitherto, so far as we are aware, in the development of any trypanosome in its invertebrate host, and since, moreover, this discovery may throw some light on certain peculiar features of the development of the pathogenic trypanosomes, we consider ourselves justified in bringing forward a brief account of our observations, so far as they have gone, without further delay, hoping to give later a full and detailed account, with figures, of this and other phases of the development.

The intracellular phase of the trypanosome has been seen by us in fleas taken from the breeding-cage, not previously exposed to infection, and fed on infected rats during a known period of time, as early as twelve and as late as thirty-six hours after feeding; but our observations are not yet sufficiently extensive to enable us to place precise limits of time to the duration of this phase. We can only say at present that we find it most abundant about twenty-four hours after the flea has taken up the trypanosomes from the rat, and that it occurs in all parts of the epithelium of the stomach, from close behind the proventriculus to the pyloric region; that is to say, over the whole extent of the true mesenteron or mid-gut, lined by the embryonic endoderm or hypoblast. In this region of the digestive tract the epithelium, as is well known, does not secrete a chitinous cuticular lining to the gut, as in the regions before and behind (stomodaeum and proctodaeum).

The actual penetration of the epithelial cell by the trypanosome has not been observed by us up to the present, but trypanosomes of quite ordinary appearance, which have, apparently, effected an entrance very recently, have been seen frequently within the cells. The earliest change which such a trypanosome undergoes is that the body assumes a bulbous form which may be compared to that of a pear with a stalk, or of a tadpole with a long tail. The pear-shaped body is easily seen to be produced by the doubling on itself of the posterior two-thirds or three-fourths, approximately, of the body of the trypanosome, while the anterior portion of the body, with the flagellum and undulating membrane, forms the stalk or tail. In stained preparations it is seen that the folding-up of the body brings the kinetonucleus in front of the trophonucleus; the result is a flagellate of pseudo-crithidial form, easily distinguished from the true crithidial forms both by the fold of the cuticle running down the middle of the body, and by the fact that the flagellum does not pass straight to the kinetonucleus along the undulating membrane, but curves round the rounded posterior end of the body to run forward (apparently) to the kinetonucleus. Seen in the living state, these pear-shaped forms appear slightly flattened, as is to be expected from their mode of formation; they are in incessant movement within the cell, exposing now their flat surface, now their edge, to the view of the observer; and the flagellum and undulating membrane can be made out plainly, as a rule, giving an appearance as if the stalk of the pear tapered to a fine point, but in a few cases the stalk appears of even thickness, and evidently consists of the flagellum alone in the early stages. The body itself is far from being fixed and rigid in its contours; on the contrary, it shows continual changes of form of the kind that are termed, in the Flagellata generally, "metabolic," as distinguished from true amoeboid movements, form-changes