fessor Charcot, appear to have demonstrated their existence beyond all question. For some time, he was disposed to admit, after Professor Ferrier's experiments, that such centres probably existed also in monkeys; but he considers that the experiments of Luciani and Tamburini (notwithstanding their personal inferences) tend to prove the contrary. Most physiologists, especially in Germany, he adds, have rallied to this opinion. Even Professor Hitzig admits that, in those centres, which he still calls "motor", he recognises centres of the sense of muscular attitude. Lastly, Professor Schiff's opinion has been completely confirmed by the fine experiments of Dr. Munk. According to the investigations of Drs. Goltz and Munk, if the lesion (at first affecting the cortex and producing tactual insensibility) be carried deeper into the white substance, sensibility to pressure and sensibility to pain will become affected. It follows from the foregoing that, according to the depth of cerebral lesion made, we can abolish tactual sensation alone, or simultaneously diminish sensibility to pressure (and to pain). Such changes can be made to affect the hinder or fore extremity, or the face, at the side opposed to the lesion, by following the indications given by MM. Hitzig and Fritsch for the destruction of the so-called "motor", that is, the sensory centres of these organs. Professor Schiff holds, with Professor Hitzig, that there is no restoration of function possible in a centre which has been really destroyed. Apparent restoration occurs only where the destruction has been imperfect, or where the function had been temporarily abolished through the influence of an injury to a part in the vicinity.

Making use of the means indicated, anaesthesia of cerebral origin was produced in several dogs, and allowed to affect them to a greater or less extent. Some months were then permitted to elapse, in order that all transitory effects of the wound should have disappeared, after which experiments with the magnetising bobbin (to use Ampère's expression) were begun. The animal, having been placed on the table in an attitude of repose, the fore-limb (when that was experimented on) was passed through the cavity of the solenoid, which thus included the lower part of the humerus, whilst the protruding paw was free in order that the state of sensibility might be determined by irritating it. The mode of irritation adopted was of the mildest, consisting as it did in lightly stroking the hair with the finger-tips, avoiding pressure with the nail. Repeated tests showed that the anaesthesia remained unaltered whilst the animal was in this position, so long as the circuit was open. The effect of completing the circuit may best be understood by a short description of an experiment. A long-haired spaniel, which had been operated on some time before, had to all appearance completely recovered his normal condition, with the exception that, in walking over rough ground or going upstairs, he frequently leant his weight on the dorsal part of his toes, which Professor Schiff regards as an infallible sign of tactual insensibility. The presence of anaesthesia was carefully determined by other tests. When the animal was placed in position as described, if the dorsal hairs of the left (*i.e.*, the sound) paw were slightly stirred by a quick downward touch, the paw was at once withdrawn, a thrill passed through the body, the hinder legs were momentarily extended, and that which was uppermost or free was kicked out. The muscles of the neck stiffened. The effect, like the irritation, was momentary. When the palmar surface was lightly and quickly touched, the result was less general, but the paw was drawn back, and the hinder leg kicked out. When the right (or anaesthetic) limb was irritated in the same manner, there was no movement whatever, neither in the paw nor elsewhere. Neither was there any movement when the hand was lightly closed and drawn over the included paw, though this had caused movement in the left limb and left side.

The circuit was closed. Five minutes afterwards, when the last-mentioned test was repeated, there resulted movements of extension and flexion of the articulation of the foot, and occasionally the toes spread out. Twenty minutes later, the excitability was found to have increased, for if the palmar surface of the paw were tickled by stirring the hairs, the foot was flexed; extension and flexion after contact had become more marked. The left extremity retained its sensibility. The acquired sensibility persisted during four or five hours, and even longer; when it disappeared, it could be reproduced in the same manner, sometimes in the course of the same day; but, generally speaking, on the following day. The result was subject to some modification when the experiment was performed on dogs of different temperaments; for instance, in the case of a large dog, of less sensibility than the spaniel, recovery of tactual sensibility was only shown after pressure on the interdigital web, but here the posterior extremity showed signs of recovery as well, though the fore-limb alone had been placed within the solenoid.

It is, however, unnecessary to enter into the details of the several experiments. The essential point is that, in all of them, recovery of tactual sensibility in the anaesthetic region was detected. In some

animals there was less recovery, in others more; but in every one of the cases the anaesthesia gave way before the influence of the solenoid, in the cavity of which an isolated member was placed. These remarkable results, therefore, supply ample evidence of the correctness of my conclusions, and distinctly corroborate the report of Professor Charcot's Commission at La Salpêtrière.

FATTY CHANGE (AND FAILURE) OF THE MUSCULAR WALL OF THE GUT, AS A DIRECT AND INDIRECT CAUSE OF INTESTINAL OBSTRUCTION AND DEATH.

By FURNEAUX JORDAN, F.R.C.S.,

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FOR several years past, I have from time to time seen cases in which, with, perhaps, no premonitory symptoms, continuous vomiting and tympany, lasting one, two, or more days, have been followed by death. While these symptoms appeared in some cases to come on spontaneously, in others, and I think more frequently, they followed some abdominal or pelvic operation. The cases, as a rule, happened in fat persons, in persons with large abdomens, in persons with signs of degeneration in various organs and with a history of habits which naturally lead to visceral changes. Examination of the bodies disclosed great internal accumulations of fat, and occasionally indications of visceral degeneration, but, curiously, no obvious or recognised cause of intestinal obstruction. In all the cases, the intestinal canal was greatly loaded with fat, and presented a strikingly yellow appearance; in some cases, indeed, it seemed to be simply a tube of fat. In one case, the microscope conclusively showed that the unstripped muscular fibres of the bowel were converted into fat. In observing and reflecting on these cases, of some of which I shall speak later, I have arrived at the following conclusions.

1. The smooth muscular fibres of the bowel are subject to fatty degeneration, which may become more or less complete; and that, consequently, they may, and do in given cases, wholly cease to contract.

2. This fatty change of the essential element of the gut-wall, when it ends in complete cessation of contractility, causes death by intestinal obstruction. Fatty failure of the intestines being in some cases extensive in area and reaching high up towards the stomach, the ensuing obstruction is acute, the vomiting incessant, and death early. In other cases, there may be less complete, or more limited, or irregularly distributed fatty change; and there will follow a slower or more fitful stream of symptoms and a later death.

3. Fatty transformation in the gut is more likely to appear (though perhaps not exclusively) in fat, especially very fat persons; in those who, from habits or natural tendency, are liable to have fatty degeneration of other organs, especially of the heart. Death in heart-cases is quick and direct; in intestinal-cases, slower and more indirect, but nevertheless very certain.

4. As premonitory syncope or exhaustion may happen from time to time before death from heart-fattiness, so "attacks" of obstruction may run before final obstruction from intestinal fattiness.

5. Failure of the bowel is helped on by continued flatulent distension, however it arises; the altered muscular fibres being so injured by overstretching that they never regain their functional contractility. Herein may be traced a likeness to atony of the bladder, where, it is well known, long-continued distension is in certain cases followed by entire loss of contractility; and it is not unlikely that fatty conversion of the muscular wall of the bladder is the basis of certain obscure cases of retention and cystitis coming on after middle age. It is conceivable that healthy gut may become the subject of fatal atony from long-continued stretching; but some, however slight, fatty change would, greatly favour such a result.

6. In a limited number of cases, death is due directly to failure of intestinal action, and may come without obvious exciting cause. The muscular fibre is now no longer muscular. In a larger number of cases, death comes more indirectly from some immediate shock to the abdominal organs. In strangulated hernia, when fatty bowel is present the, blown-out tube never again contracts. The vomiting continues, or returns, and death follows, notwithstanding that reduction has been easy and complete, and that there is no inflammation, or gangrene, or other cause of death. All injuries and operations in persons with failing gut are liable to be followed by vomiting, which ceases only with death. Especially is this so in operations on the abdominal or pelvic organs. Herniotomy and lithotomy are now and then followed by fatal vomiting, and subsequent search brings nothing to light; no injury to the peritoneum, no hæmorrhage, no inflammation, no other lesion; nothing but a hugely distended bowel.

The cases which led me to believe that, in certain instances, death begins in the gut from entire cessation of action in the intestinal muscular fibre, and that the cessation was due to fatty degeneration, I now briefly cite.

Several years ago, a lady so stout that she had long been confined to her room—the staircase of her house was also narrow and awkward—without any previous complaint, began to vomit. The vomiting, at first occasional, became incessant and fecal in character, and she sank in two or three days. I examined the body. The intestinal canal was from end to end enormously distended with gas; but there was nowhere any localised obstruction of any kind. The bowel was strikingly yellow in appearance; and the amount of fat, not only on the body, but within the abdomen, could only be described in words that would savour of caricature. After the most careful examination, no other appearances could be found to account for death.

Another case, which made a vivid impression on my mind, was that of an exceedingly stout man. He got out of doors a little in a specially made phaeton with a bottom so low that it just cleared the road, and was reached with one short step. Without injury or premonitory incident of any kind, symptoms of intestinal obstruction (sometimes urgent and sometimes with intervals of ease) set in, and in a few days he died. A very yellow distended bowel was seen; indeed, I remarked in this case, as I have in others, "The bowel seems a tube of fat". The distension was not uniform, but more in some coils than in others. There was, however, no band, or twist, or stricture, or cause of obstruction of any kind. I had not yet concluded that death might be caused by fatty failure of the gut. I was merely suspicious, and afraid that it might be so, or I should have called in the aid of the microscope.

In a case of strangulated hernia in a very stout man, the bowel was reduced easily and with marked gurgling, and for a few hours he seemed better; but vomiting returned, and he died. On examination, no inflammation, or gangrene, or apparently adequate cause for death, was found. The abdominal organs were greatly loaded with fat. The heart was somewhat softer than natural. The extreme yellowness of the bowel so struck me, and my reflections and fears had now taken so clear a shape, that I determined to have a microscopical examination of the muscular fibre of the bowel. This was carefully made for me by an experienced microscopist, Dr. Wood (one of our staff), and left no doubt of the marked fatty change in the suspected structure. Dr. Wood did not content himself with the appearance of the fatty intestine; he examined portions of healthy intestine, and found a striking contrast.

Not long ago, I had two cases of lithotomy, both of which ended fatally within twenty-four hours, after several hours of incessant vomiting. The cases were singularly alike, and a description of one will serve for both. A big fat "drinking" man of sixty had enlarged prostate and a large vesical calculus. There was no tangible evidence of renal or other visceral disease. There was no peculiarity in the operative steps to account for the result. I could not to-day alter any single step in the operation for the better. He was free from hæmorrhage or marked shock. His condition for a few hours was quite comfortable; then occasional vomiting set in, and tympany of the abdomen appeared. The vomiting became frequent and was associated with great exhaustion, and ended fatally. In a subsequent examination, a description of the appearances would answer for both bodies. The internal organs were loaded with fat; the heart was somewhat pale and soft; and the kidneys were not healthy. The intestinal canal was singularly and uniformly yellow, and everywhere enormously distended. There were no signs anywhere of inflammation, or peritoneal injury, or extravasation of blood, or infiltration of urine.

I believe the operation here destroyed the vitality, so far as contractility was concerned, of the bowel. Flatulent distension followed, and irretrievably spoiled the gut. This condition, affecting all or a large portion of the canal, and affecting it even to the vicinity of the stomach, was practically a condition of acute, high-up, and complete obstruction.

Here the question naturally arises, What are the customary explanations, now and heretofore, of the causes of death after continuous vomiting which follows the reduction of strangulated hernia, which follows also operations for uncomplicated herniæ, which follows lithotomy and other operations on the pelvis and abdomen. The very variety of the explanations testifies to their improbability. One says shock; another says shock with feeble heart; another says ether or chloroform vomiting; another says rapid septic poisoning; another says incipient peritonitis. I am far from saying that these, or some of them, are inadequate causes of death under certain circumstances; but they do not satisfactorily account for death in the cases I bring forward. In pure shock, with or without cardiac degeneration, vomiting is rare; in cases of, say crushed knee-joint, or amputation at the hip, or even in severe abdominal injury (in healthy persons), nervo-muscular action dwindles down

to death without vomiting. That ether or chloroform vomiting should recur after some hours of comfort is at least hypothetical; hypothetical also is rapid septic poisoning without rigor, or rise of temperature, or any other likeness to the known septic state. Peritonitis without the slightest sign of peritonitis is too metaphysical a pathology to grasp. In fatty change and consequent failure of the gut, we have an explanation which is based on clinical and microscopic observation, which clears up all difficulties, and which is consistent with known pathological laws.

A NEGLECTED PROXIMATE CAUSE OF DYSPEPSIA, WITH A NEW DIVISION OF THE DISEASE.*

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IN systematic works, dyspepsia is usually divided into two primary classes; namely, atonic dyspepsia, and dyspepsia caused by gastritis in its various degrees. These classes are then subdivided into many varieties, all of which may be traced to a single functional cause, namely, a defect in the reduction of food to chyme.

A person becomes conscious of stomach-disorder in its mildest form, by a feeling of uneasiness at the epigastrium, and this may or may not be succeeded by a sense of distention from flatulence. In severer cases, various other sensations, pain included, are experienced in the gastric region.

The instant a morsel reaches the healthy stomach, it is subjected to an unfeet process, by which its bulk is gradually diminished. Food is thus entirely reduced to a fluid state within a period which is found to vary somewhat when individuals are compared. But if the time of reduction natural to each be prolonged, more or less gastric uneasiness will be experienced. The inference is plain, that the energy of the nerves, upon which the stomach depends for the exercise of its functions, is excited by the ingesta, and that the organ can perform its task without sensation only within certain limits of time. If these limits be exceeded, dyspepsia will be produced.

A defect in the gastric juice may be one either of quantity or of quality; yet it sometimes happens, in severe cases of dyspepsia, when the opportunity of *post mortem* examination is afforded, that the cause is sought for in vain. There is no gastritis; the secreting follicles, and, in a word, the stomach, are apparently normal. There is, in fact, a large class of cases in which digestible food, even in moderate quantities, is not digested with ease. In many such cases, notwithstanding much daily discomfort, the general health is hardly affected. The food, although digested slowly, is digested effectually; there is no loss of flesh or of strength. The appetite is unimpaired, and the patient feels that, if he had not to eat, he would be well. The defect in such instances does not lie in the gastric juice. If it did so lie, nutrition would suffer; wasting and debility would ensue.

Experiment and observation show that a disproportion may exist between food and the quantity of gastric juice necessary to dissolve it; and it is certain, also, that this fluid is sometimes defective in solvent qualities. The form of dyspepsia which ensues in either case is of a severer type than that first considered. In most cases, there are gastric pain and anorexia, with failure of flesh and strength.

In by far the larger number of dyspeptic cases, the lesion is not one of secretion, but of the proper movements of the stomach.

The solution of any soluble substance in a liquid is greatly aided by motion. Put some crystals of sulphate of magnesia, or any similar salt, into a glass of water, and the same quantity of the salt into another glass containing the same amount of water. Let one glass remain at rest, while the other glass is gently rotated. The difference in time required for complete solution in the two cases will be very remarkable. This is clearly due to solution and removal by the water of successive layers of the salt in the glass that has been rotated.

In making experiments on artificial digestion, it is necessary to imitate the movements of the stomach. For this purpose, I have had glass cylinders made, the interiors of which are corrugated. The effect of this is that when, by means of clockwork, the cylinders are rotated, their contents are slowly shifted. (Cylinder shown.) By this means, reduction of nitrogenous substances with pepsin is much sooner accomplished, than if the experiment be conducted in bottles allowed to remain at rest, or only occasionally shaken.

The powerful muscles which compose the gizzard in birds, by the action of which hard substances are mechanically prepared for solution,

* Read before the Medical Society of London.