Thus with one exception, to be referred to later, the ferment activity in the second group is greater than that in the other two and may be very excessive. 5. Total acidity.—

Atrophic dyspepsia.	Pyloric stenosis.	Acid dyspepsia.
	0.069 per cent.	
	0.087 "	
Below 0.1 per cent.	0.076 "	Above 0.1 per cent.
	0.21 "	
	0.15 "	

The pyloric stenosis cases are given in the same order in the two tables. It will therefore be seen that the ferment activity and the acidity tend to vary roughly inversely. These results may be tabulated thus :—

Class.	Quantity.	Mucin.	Ferment activity.	Total acidity.
Atrophic dyspepsia.	Small.	Absent.	Low.	Low.
Pyloric stenosis.	Large.	Present.	Usually high.	Variable (usually low).
Acid dyspepsia.	Large.	Absent.	Low.	High.

Some explanation may be offered of these changes in the pyloric stenosis cases. We look upon them as secondary in nature, not causing though tending to aggravate the condition. It may be supposed that the hypertrophy of the stomach wall is at first accompanied by an increased secretion of both ferments and acid, but that as gastritis is set up by the irritation due to the firm coagulum in the stomach the acid secretion tends to become diminished. In support of this is the fact that where the ferment activity is highest (i.e., the gastric wall is most hypertrophied) the acidity is diminished (i.e., the gastritis is most marked) and *vice versa*. In the exceptional case where the ferment activity was low (0.5 cubic centimetre), post-mortem examination showed that the hypertrophy of the stomach wall was not so marked as is usual, the lower end of the œsophagus not being hypertrophied, and that there was remarkably little gastritis. For this reason we put no value upon the absence of excessive ferment activity for diagnostic purposes.

Treatment.—Over the bearing of the results of these analyses in the treatment of atrophic dyspepsia we need not delay. In acid dyspepsia the importance of alkaline gastric lavage together with the administration of alkalies, such as sodium citrate or bicarbonate, will be obvious. A few remarks may, however, be made on the medical treatment of hypertrophic pyloric stenosis in this connexion.

If experimentally gastric fluid from these cases is added to milk the excess of rennin is sometimes so marked that the milk coagulates firmly almost immediately. Although we have no doubt that by means of regular gastric lavage the spasm of the pylorus can be relaxed sufficiently to allow of food entering the intestine, yet if such coagulation of milk takes place in the stomach as can sometimes be seen *in vitro* it is reasonable to suppose that there must be set up renewed spasm, peristalsis, and probable gastritis. It would seem, therefore, that the best diet for these cases would be one of non-coagulable nature, such as a whey and cream mixture, citrated milk, milk powders, or even a milk treated with an antirennin preparation such as might be made from the serum of goats which had been given repeated subcutaneous injections of rennin.

Finally, we may emphasise the importance of giving the smallest practicable doses of food, for to get any large amount of food through into the intestines which are too atrophied to deal with it is but to run the risk of setting up a diarrhoea which may be fatal. Another danger of giving too large feeds is seen in all cases of wasted infants but particularly in those of hypertrophic pyloric stenosis where the stomach is so much dilated. The infant, although apparently doing well as the vomiting has diminished, gets an accumulation of several feeds in the stomach, has a very large vomit, collapses, and dies. This we think to be a very real danger and one to be kept in mind.

In conclusion, our thanks are due to the members of the staff of the Hospital for Sick Children, Great Ormond-street,

for allowing us to make use of their cases, and also to Dr. Leonard G. Guthrie who very kindly sent us two specimens from a case of hypertrophic pyloric stenosis under his care at the Paddington Green Children's Hospital.

TWO CASES OF CONGENITAL DEFICIENCY OF THE MUSCLES OF THE ABDOMINAL WALL ASSOCIATED WITH PATHOLOGICAL CHANGES IN THE GENITO-URINARY ORGANS.

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DURING the last few years there has been described a number of cases in which have been associated a defect in the abdominal musculature and an abnormality of the genito-urinary apparatus. This curious grouping of defects, which occurs in greater or less degree in every case, renders the condition extremely interesting and is the excuse for the noting of the present cases.

The two cases to be described below are published by the kind permission of Dr. Charles Bolton, under whose care they were first admitted at the North-Eastern Hospital for Children. The first case was shown during life at the Clinical Society of London by Dr. Bolton and an account of it may be found in the Transactions of that society for 1905. The cases will first be reported and afterwards a *résumé* of the hypotheses which have been advanced to explain the condition, with any comment or suggestion which may appear practicable in the light of the present cases, will be given.

CASE 1.—The patient was first seen when three weeks old. The mother, who had had one miscarriage, had suffered from lack of food during the pregnancy, but otherwise this and the labour were uneventful. The patient was the youngest of a family of 11, of whom two died at three and eight months respectively from "wasting." The child was brought to the hospital because of the condition of the abdominal wall, seeming in other respects quite natural.

On admission the abdomen was somewhat distended and bulged out at each side. Distended coils could be seen, apparently under the skin; in the bulging lateral portions these coils were thought during life to be intestine, but subsequently examination after death showed that they were the distended ureters. Vertical folds of skin were present in the region of the umbilicus, and it was considered that these were due to the pressure of the binder which had to be applied constantly. The umbilicus was imperfectly formed; no discharge was present and no aperture could be detected. On palpation there was resistance in the middle line, and it was considered that the rectus was present, but at the sides where the bulging coils were visible the abdominal wall seemed to be formed of skin only, and the fingers could be pushed into the abdominal cavity and all the organs could be palpated with ease. A hard pyriform tumour was felt in the hypogastrium reaching right up to the umbilicus and becoming much smaller after the child had passed urine. The rectus could be definitely felt to contract when he cried, but the lateral muscles appeared to be absent. The left kidney appeared to be larger than the right; the liver and spleen were palpated with ease. The diaphragm moved normally. The muscles of the back and body generally were fairly developed. The testicles were not felt in the scrotum though one was doubtfully felt at the right external abdominal ring; the child had been circumcised, not because of a tight prepuce but because it was long. He was kept under observation for seven months and gained weight; at the end of this period he began to suffer from furunculosis which subsequently became generalised. This lasted over a period of two more months when there arose the complication of suppurative pericarditis to which he succumbed.

Necropsy.—At the post-mortem examination no other suppurative conditions beyond those mentioned were found, all the pathological findings being limited to the abdominal wall and to the genito-urinary apparatus. The musculature in

the abdominal region was found to be markedly defective. The rectus was represented by thinned muscle in the region above the umbilicus only, while, below, its place was taken by aponeurotic tissue. Both oblique muscles were to be found only at their costal and postero-lateral attachments; the transversalis was only barely represented by a few fibres in the upper portion. (Fig. 1.)

FIG. 1.

Drawing of abdominal wall of Case 1 with kidneys and ureters attached (to bladder). The shaded portions are very thin muscle fibres. The rectus is thicker. RA, Rectus abdominis. U, Ureter. K, Kidney with DP, Dilated pelvis. S, Suprarenal body.

Microscopical examination.—Sections were made from the rectus region, both above and below the umbilicus, and stained variously, but mostly with hæmatoxylin followed by van Gieson's stain. In sections taken from below the level of the umbilicus, practically all through the zone usually occupied by muscular tissue, was a narrow zone of loose fibrous tissue containing rounded and oval nuclei, which stained rather darkly and stood out in marked contrast to the elongated and irregular nuclei of the connective tissue proper. Some of these nuclei were surrounded by a more or less definite zone of protoplasm, the whole cell being of considerable size; a few of the cells had horseshoe or kidney-shaped nuclei but their general appearance did not suggest a vascular origin. Their exact nature was very doubtful but they did not appear to resemble the cells of muscle tissue which had undergone a regressive change. Scattered through the loose tissue above mentioned were little masses of fat. Examination of the abdominal wall in the rectus region above the umbilicus where the muscular tissue was visible to the naked eye, though thinner than usual, showed that the muscle fibres were unnaturally rounded in shape and not so closely packed together as in the normal. The size of the fibres varied in size from 12μ to 25μ in cross section, the average size being slightly greater

than normal, some of the fibres being definitely swollen. A few atrophied fibres were visible; these were of irregular shape, the myoplasm fibrils being widely separated, so that the fibres appeared irregularly refractile and coarsely granular in cross-section. Some of the fibres contained central nuclei and in a few two central nuclei could be seen in the section of one fibre. (Fig. 2.) Many of these larger fibres appeared

FIG. 2.

Cross section of muscle from the rectus abdominis above the umbilicus showing irregularity of packing of fibres. One central nucleus is seen in the middle of the field.

perfectly homogeneous and seemed to have undergone a slight degree of vitreous degeneration; this change is what might have been expected in muscles from a patient dying from some septic intoxication and is probably to be correlated with the attack of suppurative pericarditis. Still, excluding the presence of these changes, possibly of acute origin, the general sparsity of muscle fibres in the apparently well-developed parts, the absence of any definite nuclear proliferation in the rectus region below the umbilicus, and the absence

FIG. 3.

Reproduction of photograph showing kidneys and ureters. The larger kidney is that of the left side.

of any connective tissue increase such as would probably have resulted from the disappearance of already developed fibres, all would seem to indicate that the condition is an anomaly of early developmental origin. It must, however, be remembered that the sarcolemma sheath which with the sarcoplasmic nuclei develops into fibrous or fatty tissue in retrogressive muscular changes appears only about the time of birth.

Kidneys and ureters.—The kidneys show marked inequality in size, the right being much smaller than the left, as shown in the accompanying illustration (Fig. 3). There was marked dilatation of the ureters and the larger kidney presented a great enlargement of the pelvis and the calyces; the capsules stripped with difficulty from the surfaces of the organs which showed foetal lobulation; this difficulty of stripping was the more marked in the smaller kidney, which was very firm and fibrotic. Microscopically the capsule was thickened and here and there the fibrous tissue dipped downwards into the cortex while the substance of the kidney was traversed by strands of fibrous tissue; the glomeruli were not so numerous as normally and exhibited all grades of change right up to complete fibrosis. The orifices of the ureters into the bladder were exceedingly small as compared with the dilatation in the ducts above; moreover, they were surrounded by a mass of tissue which appeared to be more dense than elsewhere in the bladder; it was difficult to say whether there was a relative stenosis. The directions of opening of the ducts appeared to be normal.

The spinal cords from this case and that immediately to be reported below were examined as regards the number of cells in the anterior horns from the lower dorsal segments, and compared with similar Nissl preparations from the cord of a normally developed child of about three months old dying from some cause not implicating the spinal cord. The numbers of large and small cells in a series of five successive sections from the segments examined were counted and the results are appended below:—

	Normal.		Case 1.		Case 2.	
	Large cells.	Small cells.	Large cells.	Small cells.	Large cells.	Small cells.
7th dorsal segment ...	45	34	45	36	51	47
8th " "	62	48	43	37	45	39
9th " "	50	48	40	22	51	42
10th " "	50	44	38	27	48	39
11th " "	48	44	44	28	47	45

From this count it will be seen that one of the cases (No. 2) shows almost exactly the same number of cells as the normal cord, while the other shows only an insignificant reduction. The actual character of the cells shows no essential differences in the three cords examined.

CASE 2.—The patient, aged two weeks, was admitted to hospital on Oct. 17th, 1905, and died on the 28th. The mother was 30 years old and had had five children by a second husband; four of these were alive, one having died from bronchitis at the age of one month. The survivors were healthy and, with the exception of the patient, showed no abnormality or defect. Two children by her first husband died from "convulsions"; she had had no miscarriages. The labour in the case of the patient was easy and quite natural. The abdomen was noticed to be big at birth and on the fifth day the discharge of a clear fluid from the navel was observed, which discharge a few days later became blood-stained.

On admission the child appeared to be wasted, with sunken eyes and short hair. The cardiac sounds were normal, the apex beat being in the normal position; the chest was drawn up at each inspiration. The abdomen was flaccid and bulged, especially in the flanks, the skin lying in vertical ridges. In the middle line of the abdomen there was much more resistance than in the flanks—in fact, it was thought that the rectus was present, at all events, in the upper part; at the sides, however, the lateral abdominal muscles were considered to be absent, as in these positions the abdominal organs appeared to be directly under the skin, and when the child cried the bulging sides contracted markedly into the centre of the abdomen. A little fluid was seen appearing through the navel. The spleen, liver, kidneys, bladder, and urachus could be easily felt through the abdominal wall. The external uro-genital organs were normal, except that the testicles had not descended. The extremities were normal but wasted. On Oct. 22nd the discharge from the navel was definitely recognised as urine, and more urine flowed from the umbilicus than from the urethra. On the 28th the child died suddenly.

Necropsy.—At the post-mortem examination the thorax and limbs were found to be normal. The abdominal wall was thinned, especially in its middle portions, while the umbilicus was represented by a depression, and an opening was seen which was found to lead into the bladder. On dissection of the abdominal muscles the costal attachments and the iliac portions of the external oblique were present but

were very thin; behind, near the latissimus, the intermediate part was present, but more internal it could not be seen; the aponeurosis passed from behind to the middle line; the external ring could be defined. The internal oblique muscle was present in the upper part and passed back from the ribs to the lumbar aponeurosis; it was absent at the attachment to the ilium and in the region where the external oblique was absent. The transversalis was represented by a few fibres in the epigastric region. The rectus was present and thinned from the usual upper limit to just below the umbilicus where it formed a very definite edge. The quadratus was found filling its normal limits but was thinned. The kidneys were normal in themselves but their pelves were somewhat dilated. There was marked dilatation of the ureters which increased in diameter as they neared the bladder, where their diameter was about two inches, and they showed convolutions. The testicles were undescended but otherwise normal. The bladder was markedly contracted and showed considerable hypertrophy of the muscular tissue. The urethra was patent and admitted a fair-sized probe. There was slight phimosis; the urachus was patent.

Remarks.—There are several views held as to the cause of these defects; these views have been summarised and the similar cases hitherto reported analysed in a paper by Garrod and Davies¹ published in the Transactions of the Royal Medical and Chirurgical Society, Vol. LXXXVIII.: 1. That the condition is due to a primary defect in the abdominal musculature followed by secondary changes in the bladder (dilatation and hypertrophy) and later changes in the ureters and kidneys. 2. That it is the result of some embryological defect occurring simultaneously in the abdominal wall and bladder, the nature of which is not understood, but it is suggested that there may have been some interference with the formation of the vascular plexus which precedes the development of the abdominal musculature, an abnormal blood-supply being also responsible for the changes in the genito-urinary system. 3. That the condition is to be explained by an obstruction to the flow of urine occurring during intra-uterine life resulting in dilatation (and later hypertrophy) of the bladder and also dilatation of the ureters and of the pelves of the kidneys. The distension of the bladder caused a stretching of the abdominal muscles, and this, associated with the direct pressure of the viscus, caused an atrophy of the lower part of the rectus muscle and of the more peripheral portions of the oblique muscles. This was further complicated by direct pressure on the umbilical vessels causing at the same time diminished blood-supply to the embryo and also inactive return of blood, this last being the cause of ascites which still further favoured stretching and thinning of the abdominal walls. The non-descent of the testicles is also to be explained on this mechanical hypothesis. Subsequently a relief of the obstruction has to be assumed and later a contraction and a folding-in of the anterior abdominal wall.

As regards (1) the defect may be placed in the muscles primarily or may be considered to be the result of an attack of acute anterior poliomyelitis occurring during intra-uterine life. The count and character of the cells in the anterior horns of the segments concerned are sufficient to negative such a view as this latter possibility. The microscopical appearances of the muscles examined in the present case would appear to suggest that the defect was of non-development rather than regression, and this was apparently so in the case of Garrod and Davies. Further, it is somewhat difficult to see exactly why the bladder should dilate because of the absence of the abdominal wall, unless we ascribe the failure of the abdominal muscles to develop as the cause of non-descent of the bladder, and this in turn making the ureters enter the bladder at an abnormal angle with resulting obstruction and secondary changes in the ureters and in the kidneys. In one case in the literature, in which a cystoscopic examination was undertaken, mention is made of a fold in the anterior wall of the bladder and displacement of the ureteral orifices which lay obliquely, and almost in a line; this particular relation changed after a laparotomy to set free the bladder from the umbilicus, and the ureters took up their usual position with regard to the bladder wall. Unfortunately it is not in every case that the bladder is attached to the umbilicus, though quite apart from this it is easy to conceive that the absence of the normal

¹ All the literature bearing on these cases will be found in their paper. I have not been able to find any similar case reported since their own.—G. H.

resistance in the abdominal wall would interfere with the descent of the bladder.

In the view (3) the assumption of a temporary obstruction to the flow of urine is extremely hypothetical and there are difficulties in the way. Although all the cases published have been of the male sex there is no evidence of any urethral obstruction; phimosis has been noted in some of the cases but only to a very slight degree. If the wasting of the abdominal muscles be due to stretching over a distended bladder it is difficult to see why only the lower segments should be affected while the tension must be equally distributed from ensiform to pubes. Moreover, in cases of ascites of extreme degree we do not meet with wasting in the stretched muscles. Again, if the change in the muscles be due to a pressure atrophy one would expect to find more definite results in the way of local reaction to the pressure, whereas no such are found; it is possible, of course, that the condition is so far advanced that these early changes have disappeared.

The ureteral and kidney changes need not be further discussed as they are quite clearly secondary results to the changes elsewhere.

A further consideration of the view (2) leads to the idea that it may be possible for both muscle and bladder changes to be due to a common cause of developmental origin, and that perhaps as the result of some vascular cause there arose a failure of a portion of the segmental development associated with a defect in the nature of an excess in the neighbourhood of the trigone of the bladder in that portion which arises from the hypoblast of the cloaca. Such a hypertrophy (which may have an analogue in the hypertrophy about the pylorus) might be associated with a relative stenosis of the ureteral orifices and a consequent dilatation in these ducts and in the pelves of the kidneys with still later secondary changes in those organs. Still, if this hypertrophy did occur we might reasonably expect it to occur alone without the constant accompaniment of deficiency in the abdominal walls.

On the whole the hypothesis of a defective development in the abdominal muscles which thus render no assistance to the descent of the bladder and allow an abnormal direction to the ureters at their entrance into the bladder, seems to be the most likely and to bring into line the condition of the genito-urinary organs with what appears to be fairly clearly a developmental anomaly of the abdominal muscles.

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A CASE OF "CAISSON DISEASE."

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CAISSON disease is a condition so rarely seen that no apology is necessary for publishing in detail the notes of a case. It would perhaps be not out of place if in a few preliminary remarks reference were made to the causes of the disease and the means taken to prevent it.

The term "caisson disease" is applied to a series of phenomena to which workers in caissons and divers in deep water are liable—in fact, all workers in compressed air in whatever circumstances expose themselves to the risk of this malady. A caisson is a cylinder of steel sunk in water, the water being excluded by means of compressed air. A diver in a diving-dress like the "caissonier" is working in compressed air, the condition of each, when working, being therefore almost identical. The caissonier, however, does not work at such a high atmospheric pressure, but the exposure to pressure is of longer duration. The causes of the disease are first, too long exposure to pressure and, secondly, too rapid decompression. These two causes may be combined. By decompression is meant the gradual reduction of any pressure over and above that of one atmosphere. Certain means to prevent the occurrence of these causes have been adopted and the data that are now worked upon have been arrived at by exhaustive experiments on animals and by observations made during diving operations. The result of these experiments and observations has been such that the incidence of symptoms is comparatively rare and the diver can now work at greater depths and with more assured safety than heretofore. The duration of decompression should bear a definite ratio to the duration of exposure to pressure and to the amount of pressure. The most constant

morbid phenomenon which has been observed at necropsies on men and animals is the presence of "bubbles" to a varying extent in the blood-vessels, heart, joints, and loose tissues. These bubbles have been demonstrated to consist of pure nitrogen. Their presence requires explanation. When a gas is in contact with a liquid under pressure the tendency is for the liquid to receive the gas in simple solution. Inspired air is practically in contact with the blood in the lungs and therefore when a diver is exposed to pressure the blood takes up an increased proportion of oxygen and nitrogen. So long as this pressure is maintained no harm accrues, neither does it if the pressure is gradually diminished. Should, however, the pressure be rapidly removed, or, in other words, should decompression be too rapid, the blood which was previously saturated tends to become hypersaturated. This tendency is overcome by the gases being set free in the form of bubbles—the carbon dioxide being eliminated by the lungs while the nitrogen finds its way into the circulatory system and loose tissues. To make this point clearer one may take as a simile a bottle of soda-water. Soda-water, as is well known, consists of water into which carbon dioxide has been forced under pressure. So long as the cork remains in the bottle no bubbling occurs, but as soon as it is removed, or, in other words, so soon as the pressure inside the bottle approaches to that of the atmosphere, bubbles are given off in abundance. This is what happens to a too rapidly decompressed diver or caissonier, the extent of the bubbling depending on the amount of pressure, the duration of pressure, and the rapidity of decompression. The reason these bubbles are composed of nitrogen only and do not contain any oxygen or carbon dioxide is explained by the fact that whereas oxygen enters into chemical combination with the tissues or is eliminated in the expired air as carbon dioxide, nitrogen does not combine with anything. With these few preliminary remarks I will proceed to describe the case in detail.

The patient, a man, aged 49 years, was admitted to the Torbay Hospital under the care of Dr. J. H. Gough. He had been engaged as diver in the examining of torpedo-boat No. 99 which went down in 25 fathoms (150 feet) of water four and a half miles off Berry Head. He was an experienced diver and had made two previous descents on different days. The patient was admitted at 7.30 A.M. On the previous night he left the surface to go down to the wreck at 7.12 o'clock and owing to the fact that his lines were fouled by the wreckage he was not brought to the surface until 12.15 A.M. When it was found possible to raise him he was brought up to within 50 feet of the surface and then decompressed as indicated in the following table:—

Depth in feet.	Stoppages in minutes.
50	25
40	30
30	30
20	30
10	45

On coming to the surface he was so exhausted as to be unable to get on board without assistance. When his face-piece was removed he was able to answer questions and apart from the exhaustion did not appear to be much the worse for his immersion. He was covered with blankets and given hypodermic injections of ether and brandy. There were no signs of dyspnoea and his radial pulses were good. One hour later he complained of "bends" in the right arm and knees. These "bends" are a fairly constant sign of this malady and consist of bending of the limbs accompanied by pain. His hearing was unimpaired. The pains and "bends" passed off and he slept. On arrival at the hospital his condition was as follows. His colour was good but the expression was rather anxious. There was no lividity of the face or of any part of his body. He was able to take an intelligent interest in his surroundings and to make his wants known but he was quite deaf. Inspiration was deep and prolonged, expiration being short and shallow. His hands were very cold and the surface temperature generally appeared to be below normal. The temperature taken in the axilla was 97.6° F., the pulse was 114 (carotid), and the respirations were 32. With regard to the circulatory system no cardiac impulse could be seen or felt. There was slight dilatation of the right side of the heart. The heart sounds could only occasionally be heard and were then very muffled. No bruits were heard. Neither radial pulse could be felt nor was any pulsation palpable over the femoral, popliteal, tibial, or dorsalis pedis arteries. Pulsation was