sion results in the protrusion of bowel along the inguinal canals, especially on the right side, and in the forcing of fluid along the processus vaginalis, thus forming the several varieties of hydrocele seen at this period of life. In some of the cases that are classified as hernial protrusions, the sac appears to contain fluid only, and in others fluid and bowel. It is obvious, therefore, that the sole cause of the development of both these inguinal protrusions, whether hernial or hydrocele in character, is a simple mechanical one, as in the case of the corresponding stretching of the umbilical cicatrix.

My experience of the usual treatment of these conditions is that the surgeon who sees them is quite satisfied with ordering a truss, or, in the umbilical variety, if a truss is not ordered, an attempt to intrude the protrusion into the abdomen is made by strapping with or without a pad. The results of such treatment, as seen in the out-patient department of a children's hospital, shows that it is almost useless. With very few exceptions, I have found that both umbilical and inguinal hydroceles and hernias in young infants disappear spontaneously after the child has been fed properly for a time.

I do not wish to suggest that in many cases the constant application of pressure by means of a suitable truss will not very often hasten the disappearance of the hernia or hydrocele, but the reduction of the abnormally excessive intra-abdominal tension resulting from indigestion must be the chief consideration, while the local pressure which is exerted on the aperture in the abdominal wall with the object of retaining its protruding contents must be regarded as only an adjuvant. The reason this treatment occasionally fails in out-patient practice is that it is hopeless to make some parents understand the importance of feeding their children properly, or they may be so circumstanced as to be unable to give the infant the attention it requires. I think I have now shown that it is unwise to rest satisfied with treating such cases with a truss alone, whether applied constantly in the form of a washable truss or, as is almost always the case, the mother is directed to remove the truss at night time and when the child is being washed.

I have heard of surgeons stating that they have never seen a hernia in a child, however young, which could not be treated by means of a truss. I fancy that they meant that a skilled surgeon can in any case apply a properly made truss of suitable size and material to a child in such a manner that the instrument will retain the contents of the hernial sac within the abdomen during the few minutes the child is under the surgeon's observation in the room, and one would readily allow that the slighter forms of hernia will recover under this treatment alone, since there exists a very strong tendency to their spontaneous disappearance.

My almost invariable experience of cases in which the hernial contents are retained with difficulty is that unless the child's feeding is attended to, in a very short time, even assuming that the truss does keep the hernia up, the irritation produced by the pad or some other portion of the truss soaked and rendered rough and irritating, produces an eczema by its pressure on the very sensitive skin of the lowly-vitalised infant. We all know how very readily thin, badly-fed children develop inflammation of the skin from any local irritation. As an illustration of this I would point to the almost constant presence of phimosis which exists among them and which is the product of a chronic inflammation of the end of the prepuce due to its being constantly moistened with urine (which itself possibly is irritating in character as the result of bad feeding) and to subsequent contraction of the inflamed orifice. Some surgeons have been so far misled by the frequent association of phimosis with hernia as to suggest that the hernia is produced by the phimosis on the supposition that the child strains forcibly to pass its urine through a constricted orifice. I would, however, willingly admit that a phimosis developing in the manner above described may be so extreme that the straining necessary to drive urine through it will assist the exaggerated intra-abdominal tension in developing a hernia, or in increasing one already developed, or may oppose the recovery of one even when the child is properly fed. It serves as an interesting example of how ready we are to mistake one effect as the cause of another—a very common fallacy in surgery as well as pathology.

HEART DISEASE IN CHILDREN.

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INTRODUCTION.

I THINK it will be of more practical utility if I can lead this discussion in a direction tending to throw light upon conditions which are yet imperfectly understood, though common, rather than to refer to cases chiefly interesting on account of their rarity. Partly on this account and partly because of the little scope which such cases offer for practical therapeutics, I do not intend to make any reference to congenital heart disease.

In considering the subject of heart disease in children, the first broad fact that strikes us is the extreme frequency with which evidence of cardiac lesions is found, especially of regurgitation through the mitral valve. Mitral regurgitation causing overt, and even urgent, symptoms, is by no means rare in children, that is, before the age of 12 or 13. But the cases in which, without obvious heart symptoms, *bruits* are found indicating mitral incompetence, are incalculably more common. I have not actual statistics before me, but I believe I am justified in stating that nearly, if not quite, half of the cases treated in the medical wards of the Newcastle Children's Hospital present *bruits* indicating some degree of imperfect closure of the mitral valve. I need scarcely add that the proportion of cases which are admitted on account of obvious heart symptoms is nothing like so large. For fear of misconception, I will remark at once that when I speak of *bruits* indicating imperfect closure of the mitral valve, I do not wish it to be understood that I regard every systolic *bruit* audible at the apex of the heart as proof of deformity of the mitral valve. As I shall have to insist further on, there is no fact in connection with this subject which more needs to be emphasised than this—that there may be, and often is, mitral incompetence without any deformity of the valve curtains themselves.

We start, then, from this point, that mitral regurgitation is extremely common in children, much more common, I am sure, than would be supposed by any who are not accustomed to observe and record physical signs with more than ordinary minuteness, and very much more common than is found to be the case in adults, at least, if we leave out of calculation cases occurring in young women and due to chlorosis. We have, then, two problems to consider; first, the etiology of heart disease, and especially mitral regurgitation, in children; and, secondly, the later history.

ETIOLOGY.

Without doubt many cases of heart disease in children are really rheumatic in origin, even where no history can be obtained of an attack of rheumatism of the joints. It is a clinical commonplace that rheumatism in children affects the heart more frequently than in adults, and that the joint affection is usually milder in children. But this is only a small part of the truth. Cases certainly do occur in which acute endocarditis or pericarditis may be the only local manifestation of an attack of acute rheumatism, there being no joint affection at all. It is admitted that this may occur in adults; but it happens much more frequently in children. I may mention, as a typical instance of this form of disease, a little girl who was admitted into hospital under my care for chorea. The chorea followed a fright, and was of the ordinary type, moderate in severity. A systolic *bruit* at the apex was audible when the child was admitted. When the child was almost entirely recovered from the chorea, she suddenly developed a high degree of pyrexia, with severe cardiac distress. There was no joint affection. After a time the acute disease subsided, but it was evident that the heart was left in a worse condition than before. Here was a case of severe acute endocarditis, which, by its occurrence during convalescence from chorea, is stamped as almost certainly rheumatic. But suppose a milder case to occur, without the coincidence of chorea as a guide, it is very likely that the nature of the disease will be misunderstood, or even that it may be entirely overlooked, though quite severe enough to leave behind permanent injury in the valves of the heart. Children often appear tolerant of a considerable degree of

pyrexia, so that in hospitals, where the temperature is regularly taken as a matter of routine, it is not at all uncommon to find that the thermometer reveals a feverish state of which the appearance and demeanour of the patient gave no hint. Even when *malaise* is sufficient to lead to the discovery of pyrexia, there may be little to draw attention to the heart as the seat of the disease, and no examination may be made; and even if it is otherwise and some *bruit* is discovered, unless the practitioner has happened to have examined the heart shortly before the appearance of the acute disease, any morbid signs which are observed may not be recognised as recent; they may be taken to be the result of some older mischief. In all probability many cases of rheumatic endocarditis without joint affection do not produce sufficient obvious disturbance to bring them under medical treatment at all, although they may be quite sufficient to leave permanent injury to the valves.

Pericarditis is not quite so likely to escape notice, because it is more likely to be accompanied by local pain. I believe that these considerations give the key to the origin of many *bruits* in children where no history of rheumatism can be elicited by the closest inquiry. While I am on the subject of acute heart disease, I may mention that the so-called "malignant" or septic endocarditis sometimes occurs in children, and that even very severe examples of this grave disease may recover. A case which apparently belonged to this class was recently under my care in the Children's Hospital. A little girl, aged 12, who had suffered from the symptoms and presented the physical signs of phthisis for more than a year, showed also the evidence of mitral stenosis. This combination of conditions, so very rarely found associated, is in itself sufficient to make the case one of unusual interest. But its after-history was still more remarkable. While the child was in the hospital under treatment for the phthisis her temperature rose rapidly and took on a septic type, with very wide excursions. At the same time there was very severe cardiac dyspnoea and distress, and *bruits* which varied greatly in character from time to time were heard. The spleen became enlarged and tender to pressure, and a hæmorrhage took place from the bowel which was very nearly fatal. After about five weeks the acute febrile disturbance gradually subsided. The child left the hospital with her heart apparently in no worse condition than before the acute attack. She has since presented herself occasionally amongst my out-patients, complaining more of her phthisis than of symptoms referable to the heart.

Although rheumatic endocarditis occurring without any accompanying affection of the joints may serve to explain many cases of heart disease arising in children which might otherwise be obscure, we must not conclude that this key will fit every lock. One of my recent cases has appeared to me to present unusual interest, just because it furnished a puzzle the solution of which seems well nigh impossible. A boy, about 10 years of age, was under treatment in the hospital for severe bronchitis. He had recovered from this, and was for some time allowed to get up. I was about to discharge him one day, but just for routine's sake I examined him before doing so. To my astonishment I found a loud systolic *bruit*, audible over the whole precordial region and at the angle of the scapula. To suppose that this *bruit* had existed before, but had been overlooked both by the resident medical officer and myself, would only be admissible on the understanding that we had both been stone deaf. Under no other circumstances could we have failed to detect in our numerous examinations a *bruit* so widely diffused and so loud. Any attack of acute endocarditis, such as I have described above, was entirely excluded by the ascertained absence of pyrexia; for it is the custom in our hospital to have the temperatures of all patients recorded every night and morning. I sent the child back to bed, and kept him at rest under treatment with digitalis and iron for several weeks. But the *bruit* did not alter, except that it was sometimes audible at the angle of the scapula, and sometimes not so. It would, of course, be more satisfactory if a clear account could be given of the etiology of the heart lesion in this patient. But the case is worth recording if only to show that a mitral regurgitation (promising to be permanent) may arise under circumstances apparently excluding the ordinarily recognised causes of heart disease.

AFTER-HISTORY.

The extreme frequency of signs of mitral regurgitation in children, so greatly in excess of what is observed in adults, suggests the necessity of an explanation of the discrepancy. No doubt the severer cases die early; but these are quite the exception, and the number of deaths from heart disease in children is quite insufficient to furnish the solution of the problem. Although we may admit that children having valvular defects (even though not presenting obvious heart symptoms) are likely to resist the attacks of other diseases less successfully than children who are not handicapped in the same way, yet even this would give a very insufficient account of the matter. I think the only explanation at all adequate is that in a very large proportion of cases the mitral incompetence observed in children disappears in later life. I wish it were possible to furnish statistics in support of this proposition. But that is obviously impossible. In the nature of things these mild and favourable cases are just the ones that disappear from our observation the soonest. Physicians to children's hospitals necessarily lose the opportunity of seeing them again in later life. Hence the difficulty of bringing to statistical proof the fact of which I am nevertheless strongly convinced, namely, that mitral regurgitation in children is a condition which is very frequently recovered from.

Now, it is probable that structural alteration of a valve, following endocarditis, may be more readily recovered from in childhood than in later life. But there is another condition equally capable of producing the phenomena observed, and much more readily recoverable—I mean regurgitation through the mitral valve produced by dilatation of the left ventricle. I believe that far less prominence has been given to this condition than it deserves in ordinary clinical teaching. Regurgitation through the tricuspid, arising in a similar manner, is a clinical commonplace. But when distinct evidence of mitral regurgitation is discovered, valvular lesion from old (or recent) endocarditis is usually assumed as the almost necessary corollary. Many authorities certainly speak of mitral incompetence following dilatation, but in ordinary clinical teaching the fact of its frequent occurrence is left in the background. I have shown elsewhere that it takes place in chlorosis much more commonly than is generally supposed. In 400 cases of chlorosis under my own care I found complete evidence of mitral regurgitation (a systolic *bruit* audible at the angle of the scapula, as well as at the apex) in 123. In many of these the graver physical signs were known to have appeared during the time that the chlorosis was getting worse from neglect of treatment; and in a still larger number they entirely disappeared when the anæmia was removed by the administration of iron. It was therefore evident that the mitral incompetence was due to dilatation, and not to any structural change in the valve itself. I cannot doubt that dilatation of the left ventricle plays a very important part in the production of these common mitral *bruits* in children. I am convinced that the small vegetations commonly observed on the edges of the valves in chorea do not explain the *bruit* which is so frequently heard in the course of that disease, and which is comparatively seldom permanent. For although such vegetations are rarely absent when a *post-mortem* examination is made after chorea, we must recollect that it is only in cases of altogether exceptional severity that death occurs. So that it is rather an unwarrantable assumption that endocarditis is a frequent accompaniment of the ordinary mild type of the disease which recovers. But there is a much stronger reason for doubting the causal relation of these tiny vegetations to the *bruits* in question. It is not at all likely that such vegetations would interfere with the closure of the valve. And, therefore, they would not produce a systolic *bruit*; if they produced any *bruit* at all it would be presystolic. But the *bruit* in chorea is usually, if not always, systolic. Probably these minute vegetations, when they do develop, have nothing to do with the ordinary transient *bruit* of chorea. I think that ventricular dilatation is a more satisfactory explanation than irregular—choreic—action of the muscoli papillares. I once observed, in a girl, aged 11, who had suffered from chorea, a *bruit* audible at the pulmonary area only, the first sound at the appendix being normal. The *bruit* disappeared under treatment. In another patient,

whose age was 24 when I examined her, and who had suffered from chorea following a fright when she was about 10 years old, I found a *bruit* presystolic and systolic at the apex and systolic at the angle of the scapula. Here I should be disposed to attribute the damaged condition of the mitral valve to an attack of endocarditis such as I have above described, although I obtained no history pointing in that direction. This seems rather more probable than to lay the blame of such extensive damage to the chorea directly.

It should never be forgotten that, when signs of mitral regurgitation occur in the course of acute rheumatism, this is not necessarily due to endocarditis and its consequences. In a large number of instances a temporary dilatation of the left ventricle offers the more reasonable explanation. This suggests that all such cases should be treated by prolonged rest, with heart tonics, including iron. Even where actual valvular deformity exists, prolonged rest gives perhaps some chance of complete recovery, and certainly affords the best opportunity for the establishment of satisfactory compensation.

Dr. TOWNSEND said that cardiac disease in children was due in most cases to a rheumatic cause, either hereditary or acquired, and was generally very insidious and not indicated by symptoms. To be found it must be looked for. The localisation of a *bruit* in the mitral area, particularly if heard in the posterior scapular region, was the only symptom that pointed out regurgitation through the mitral valve. As an explanation of the occasional disappearance of the *bruit*, Dr. Townsend suggested that the *bruit* might be due to dilatation of the ventricle, and that the restoration of the ventricle to its normal condition might explain the cessation of the *bruit* rather than that an organic lesion was absorbed. The principal dangers to life in these cases were sudden over-exertion, fresh attacks of rheumatism, or degeneration of the arteries in middle age.

Dr. BYERS (Belfast) spoke of the frequency of mitral disease in female children, and pointed out the infrequency of the signs of sclerosis in young children as compared with those of regurgitation. He referred to the frequent occurrence of acute heart disease in children and of severe dilatation. He thought the great proportion of the cases of heart disease in chorea was organic, and he could corroborate Dr. Barlow's observations that when rheumatic nodules appeared in children the prognosis was bad.

Dr. DAWSON WILLIAMS observed that the difficulty of prognosis in heart disease in children was often very great, and suggested that this difficulty was in large part due to incomplete knowledge of the etiology. There was no difficulty in accepting the rheumatic theory when a distinct history of rheumatic joint affections could be obtained, but he questioned whether it was allowable to accept a vague history of pains in the limbs as a history of rheumatism. Was the cardiac disease following scarlet fever truly rheumatic? The so-called malignant endocarditis, that is to say infection of the valves by pyogenic organisms, was, probably, not fatal in all cases, and might be responsible for a certain proportion of the cases of heart disease occurring in association with chorea as well as scarlet fever. The onset of cardiac disease in chorea was sometimes exceedingly insidious and unaccompanied by any obvious signs of rheumatism.

Dr. COLQUHOUN followed Dr. Dawson Williams in thinking that it was not safe to assume that all heart lesions in children were expressions of the rheumatic diathesis. It was safer to assume that we had not definite information on the subject, and wait until we knew more definitely what the poison of rheumatism was. The subject of heredity was very important, as children might no doubt be born with weak hearts, which were easily affected by causes which would not affect the normal heart.

The PRESIDENT (Dr. Barlow), while recognising that other possible factors ought to be borne in mind, thought that it was only safe to exclude rheumatism after the history of the child had been thoroughly ascertained. When first seen there might be no evidence of rheumatism beyond a heart murmur, but later in the case affection of joints, muscular rheumatism, or erythema might be observed, and confirm the diagnosis of rheumatism. He agreed that cases of sep-

tic or malignant endocarditis might recover, at any rate from the acute attack. Dilatation was an important factor in the production of cardiac disorders in childhood, and might sometimes be observed in renal disease after scarlet fever. The difficulty of prognosis was often great in heart disease in childhood. He believed that, as a general rule, the louder the murmur the greater the implication of valves.

Dr. COLEY replied briefly.

NOTES ON CASES WITH SPECIMENS OF GASTRO-ENTEROSTOMY AND PYLORECTOMY.

Read in the Section of Surgery at the Annual Meeting of the British Medical Association, held in Newcastle, August, 1893.

By RUTHERFORD MORISON, M.B., F.R.C.S.,

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CASE I.—W. S., male, aged 65. History of two years' stomach trouble, chiefly attacks of vomiting. During last four months and a-half has had pain, and vomited all sorts of food, retaining very little. The patient is a thin careworn looking man, feeble and emaciated. A few years ago he was much addicted to alcoholic excess. A rounded hard nodular tumour is discovered under the umbilicus; tender on pressure, freely movable to left side, upwards and to right, but not downwards; it is about the size of a tangerine orange; otherwise nothing discovered. On washing out stomach a pint of yeasty offensive compound was removed. On March 3rd, 1892, pylorus excised, ends of stomach and duodenum closed, and gastro-enterostomy with Senn's plates and Lembert's sutures performed. The operation occupied two hours. He was sick at 6 P.M., 7 P.M., and 10 P.M., but never again. March 4th he appeared to be as well as before the operation, and was allowed nothing by the mouth until 7 P.M., when he had a little hot water. Nutrient enemata every two hours. March 5th he is in good condition. Pulse 80, temperature 98.4°. Allowed milk and soda water to drink. March 6th not so well. During the night he had a syncopal attack, with dyspnoea. (He says he had similar attacks at home.) He has taken a tumblerful of milk in divided portions without discomfort. In the afternoon, at 3.30, he died from heart failure.

Pathology.—The portion of stomach and duodenum removed—2½ inches in length—was an annular scirrhus carcinoma of the pylorus. On opening the abdomen all the wounds were found healthy and firm; no peritonitis; a little blood staining of intestine. There was no malignant disease left behind. The stomach was empty. The plates were loose, one lying in the stomach little changed; the other in the ileum, about 2 feet from the ileo-colic valve, in an advanced stage of digestion. The stomach is not dilated; its walls are thick and healthy.

CASE II.—T. S., male, aged 31, a dyspeptic man frequently before. Three months ago pain in stomach with vomiting after a full meal. Coffee-ground vomiting twelve days ago. Vomit has occasionally been in large quantity, and yeasty looking with a bad smell. For the last twelve days has not been in bed, for the pains prevent him from lying. He is a thin, nervous looking man. Cannot lie down for pain in back and stomach, and sits constantly stooping forward for ease. A large, fixed, hard, nodular tumour is felt filling up the whole epigastric region. There is pulsation in it and a *bruit*. Diagnosis, large malignant tumour of stomach involving pylorus. On November 17th, 1892, abdomen opened; tumour adherent to liver, involving pylorus; attached to parietal peritoneum at upper part, and several nodules of malignant growth found on parietal peritoneum. Gastro-enterostomy performed with Senn's plates. Some difficulty was experienced, for stomach was fixed and could not be drawn out of wound. Growth surrounded by a pad of omentum twisted and ring shaped, to prevent pressure on parietal peritoneum. The patient suffered from no shock, and on recovery from the chloroform found his old pain gone. On October 23rd, five days after operation, he sat up in a chair and felt well. On the eighth day he had a sudden syncopal attack after being shaved, and died in a few minutes. His general condition had been in no way disturbed by the operation; his temperature and pulse remaining normal throughout. At the time of his death he was able to eat and dispose of solid food with satisfaction, and was never sick after the operation.

Post-mortem Examination.—Nothing was found but the malignant growth as seen at operation. The union of the stomach and intestine was perfect; there was no peritonitis, except an amount sufficient to glue the omentum round the tumour. No trace of either plates. The stomach was not dilated, its walls were thick and healthy.

CASE III.—J. S., male, aged 53. Twelve years ago had a stomach illness, diagnosed as gastric ulcer. A similar illness has recurred every now and again. Three months ago had to take to bed, and has been bedfast since. Has lost several stones in weight (four, he thinks). Can take nothing but fluids, and even with them has very bad attacks of pain and vomiting. The patient looks as if he had been a ruddy, healthy man, but is much emaciated and weak. He feels hungry, but dare not eat. Dr. Smith, of Ryton, under whose care the patient was, had made a diagnosis of obstructed pylorus from cicatrization of a gastric ulcer, with dilated stomach. On examination of the abdomen, percussion and succussion showed the stomach to be dilated. On washing it out, 3 pints of water were held without setting up expulsive contraction. Gastro-enterostomy was performed on May 16th, 1893, without Senn's plates, but with three rows of sutures. From the time of recovering from the chloroform, though feeling and looking well, and free from pain, he was frequently sick, and on the following day, May 17th, brought up a considerable quantity of bile and pancreatic secretion. Regurgitation of a similar fluid continued more or less constantly till his death on March 19th. His temperature never rose above 99°, and he had no pain nor distension.

Post-mortem Examination.—No trace of peritonitis. Stomach enormously distended with a greenish, stinking fluid. A scar, about the size of six-