bacillus in cultures in 1 part in a million. Nevertheless, animal experiments with this substance are not very promising, though clinical results, as published in Feldt's monograph in 1923, are certainly both hopeful and inspiring.

KRYSOLOGAN (?)

The last of the gold cures is that recently introduced by Moellgaard.* It is a double thiosulphate of gold and sodium, $\text{Au}(\text{S}_2\text{O}_3)_2\text{Na}_2$, which, although a well recognized substance, he calls sanocrysin. Like its predecessors it retards growth of the tubercle bacillus in glycerinated bouillon in such strengths as 1 part in a million, but how serum added to the cultures affects its action I did not discover. Nevertheless, this is a very important point, since such chemotherapeutic substances as exert an undoubted action on bacteria invariably act better in the presence of the tissue fluids. Moellgaard assumes that his gold injections destroy the tubercle bacillus *in vivo*, and states that doses which are not poisonous in themselves kill the tuberculous animal by producing a tuberculin shock. This shock begins with albuminuria and sometimes haematuria; it is followed by toxic myocarditis and pulmonary oedema. Non-tuberculous animals do not exhibit this shock.

The evidence to prove this important point is nowhere clearly set out—that is, that the shock in tuberculous animals after an injection of sanocrysin is due to the destruction of tubercle bacillus and the setting free of endotoxins, which act as tuberculin acts on tuberculous animals. To combat this shock Moellgaard recommends a previous injection of antitoxic serum, after which his gold cure may be injected with impunity. It would be interesting to know whether other metallic poisons cause a like shock.

Moellgaard made many experiments on calves, rabbits, and other animals to test his sanocrysin, and the treated calves certainly did better than the controls, and some of them recovered. His procedure was as follows: He injected a number of animals on the same occasion; after some days the animal which seemed least affected was chosen as a control. Dr. A. S. Griffith thinks that this was wrong, since the calves may have been infected before they were used for experiment. The injection of the tubercle bacillus would cause those which had acquired some increase of resisting power as the result of a spontaneous infection to react violently for perhaps a week, while the really susceptible animals would remain unaffected; one of the latter would be selected as the control. It is true Moellgaard made tuberculin tests when the animals were three weeks old, but their weights show that they were much older when injected. The experiments of Dr. L. Cobbett at Stansted under the Royal Commission showed that, in spite of ideal conditions and constant care, it was impossible to exclude spontaneous infection.

Another feature of importance in these experiments is that the cultures used were attenuated. The doses necessary to kill were enormous and many control animals failed to die or contract severe disease. The attenuation of the bacilli makes recovery much easier and the large size of the dose intensifies the severity of the reaction to injection in an animal already infected.

So far as I am able to judge of the value of the recorded experiments, they are suggestive, but certainly not conclusive. Some of the krysolgan and copper experiments on animals have been equally suggestive, and with regard to the patients it is early to draw conclusions; it is the opinion of those best able to judge that clinical records of treated patients must be kept for years before the true value of a remedy for tuberculosis can be gauged.

Sanocrysin, then, is still on its trial.

* A paper by Professor Moellgaard on the theoretical basis of the sanocrysin treatment of tuberculosis was published in this JOURNAL on April 4th (p. 643).—Editor, BRITISH MEDICAL JOURNAL.

Humeian Lectures

ON

SOME FORMS OF VOMITING IN INFANCY.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF LONDON, MARCH-APRIL, 1925,

BY

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LECTURE II.—VOMITING DUE TO OBSTRUCTION FROM NEUROMUSCULAR INCO-ORDINATION.

Etiology.

I COME now to consider the etiology of the second form of obstruction, that which depends primarily upon neuromuscular inco-ordination. The beautiful experiments of Bayliss and Starling have made clear the method by which a bolus of food is moved along the intestinal lumen. Excitation by the bolus produces a twofold reaction in the muscular tube—contraction in the segment above accompanied by relaxation in the segment below. The bolus, urged forward by the contracting muscles into the area of relaxation, thus evokes a succession of peristaltic waves which hurry it along the bowel. These peristaltic movements persist in an isolated loop of gut separated from its central nervous connexions. Nevertheless, the intestine is possessed of a double nerve supply which has the power of augmenting or of inhibiting intestinal tonus and movement. The inhibiting fibres pass in the splanchnic nerves and peripheral stimulation produces cessation of movement and relaxation of tone. Stimulation of the vagus is less uniform in its effect, but most often after a short period of inhibition there follows a long phase of unusual activity of movement, and at times the gut may be so contracted as to obliterate its lumen.

Little is known of the localization of the cerebro-spinal centres of these two antagonistic sets of fibres. The centre for the inhibitory fibres is generally thought to lie in the spinal cord, in the upper dorsal and lower cervical regions. Yet Ott obtained inhibition by stimulating the optic thalami and cerebral peduncles. Budge and Valentin showed that intestinal movements could be excited and amplified by electrical stimulation of the corpora quadrigemina and corpora striata. Bochart in Vulpian's laboratory obtained movements of the intestines of dogs by stimulating certain parts of the motor cerebral cortex. Our knowledge of the paths by which the higher centres influence the intestinal movements remains, however, doubtful, fragmentary, and contradictory. That emotional states are habitually accompanied by changes in the amplitude and frequency of intestinal movements is everyday experience. Cannon found that motor activity in the stomach and intestines of his cats lessened when the animals became excited or angry. Barclay, in studying the gastric tone and movement in normal individuals, describes the striking alterations produced by an unexpected stimulus such as a loud sound.

In considering the etiology of this group of disorders in infancy, characterized by a temporary failure of the inhibitory apparatus of the bowel, I put forward the hypothesis that in development the inhibitory apparatus is apt to lag behind the motor and augmentary apparatus. That at this early age we have to deal with an organism in which the inhibitory activities of the splanchnic nerves are functionally weak is suggested by a variety of considerations. It is perhaps to be expected that the supreme function of inhibition shall be that which is last acquired. In the case of the skeletal musculature a similar functional retardation of inhibition is the rule. The newborn infant, before the myelination of the pyramidal tracts, is rigid and spastic in all its movements. Until the cortex awakens and assumes control the infant is in a state of what might be called physiological diplegia. Only gradually does the power develop to relax antagonistic groups of muscles and to permit precise and effective voluntary movement.

It is interesting in this connexion that many observers have noted that hypertonus of the skeletal musculature is commonly more than usually marked in infants with congenital pyloric stenosis. Putzig has called attention to opisthotonos as an occasional symptom in hypertrophic pyloric stenosis. Feer has emphasized the characteristic set look of the facial muscles, showing especially in the wrinkling of the forehead. Certainly, in my experience, infants with hypertrophic pyloric stenosis, at least in the earlier stages, are more than usually tense, alert, and eager, with rigid limbs, exaggerated tendon reflexes, and clenched fists.

In certain degenerative disorders of the cerebrum we again meet with an increase of tonus in the skeletal muscles, with rigid limbs and stiff postures, and in such cases spastic states of the bowel musculature seem also unusually common. Here, as in many other conditions, there is a certain similarity between the results of functional weakness in the immature nervous system and disturbances directly due to degeneration and disease. In ascent and in descent, in development and in degeneration, the same country is traversed.

Thus I have at the present time a boy under my care in Guy's Hospital, 2 years of age. He belongs to a family more than one member of which has died of some obscure nervous disease in early childhood. In at least one of these it is known that optic atrophy was present. He is suffering from an unusual form of diffuse cerebral sclerosis, probably familial. Ophthalmoscopic examination shows the presence of optic atrophy, but no cherry-red spot such as is characteristic of amaurotic family idiocy. There is extreme rigidity of the limbs with pronounced opisthotonos. Through the rigid abdominal walls great waves of intestinal peristalsis are visible. There is extreme constipation. No motion is passed spontaneously, but by washing out the rectum hard scybalous masses are removed from time to time. The colon is hypertrophied and can easily be felt through the abdominal wall. I show its outline as seen after an opaque meal, and after an opaque enema.

In this case it is clear that in an older child with cerebral degeneration hypertonus of the skeletal muscles and hypertonus of the intestinal muscles have developed together. In both the power of inhibition has been lost. I suggest that in the newborn infant, while a large part of the cerebrum is as yet functionless, there may at times be added to the lack of inhibition of skeletal musculature a similar weakness of the power to inhibit the splanchnic musculature. In the newly born, no doubt, the functional disturbance is often trivial, shading off by all gradations into the normal, and transient. In the similar conditions of later life the disturbance is often both severe and persistent. Of infantile achalasia at the cardia and pylorus it may be said that the time limits of the obstruction appear definite. In the case of the pylorus, certainly, if the infant does not succumb to intercurrent infection, recovery may be confidently predicted in the fourth or at latest the fifth month. The high mortality of this disease in the past has been due, not to the severity of the obstruction as such, but to the peculiar inability of the infant to withstand inanition and to the great fall in immunity which the inanition causes. Once the functional weakness at the pylorus has been overcome relapse never occurs. This certainty and this permanency of recovery are in keeping with the view that the symptoms are due to a retardation of functional development.

The failure of inhibition is, as might be expected, most evident at the site of the various sphincters, at the cardia, notably at the pylorus, at the ileo-caecal valve, and at the

ano-rectal sphincter. We owe to our President, I think, in the case of the cardia, the first enunciation of the conception that in a failure of relaxation of the ring muscle of the sphincter we have the primary cause of the obstruction. In 1896 Rolleston showed at a meeting of the Pathological Society a number of specimens of oesophageal obstruction, and put forward this hypothesis. More recently the radiographic studies of Hurst have afforded the proof of the correctness of the supposition, and he has coined the word "achalasia" to describe the condition. Under the *x* rays the failure of the cardia to relax is clearly evident, while *post-mortem* examination in these cases shows that, although there is hypertrophy of the walls of the dilated oesophagus above the obstruction, the ring muscle itself shows no trace of hypertrophy.

A. PYLORIC OBSTRUCTION.

The explanation of the remarkable condition found in hypertrophic pyloric stenosis has been the subject of endless controversy. Armstrong in 1777 was probably the first to describe a case. In 1888 Hirschsprung of Copenhagen called the attention of the profession to the condition, describing the clinical symptoms with great accuracy, but erroneously regarding the cases as examples occurring in infancy of that congenital pyloric stenosis of Landerer and Maier described in the last lecture. The view that the obstruction at the pylorus is due to pressure from the bulk of muscle, the subject of a congenital hypertrophy, seems to me to have little to recommend it. Indeed, no true stenosis of the pyloric canal is present. Within the grip of the rigid musculature of the pyloric cylinder there lies a normal pyloric canal, its lumen in no way narrowed, but, as it presents itself at autopsy, so folded up that its outline is star-shaped or rosette-shaped upon transverse section. During life it is clear that the obstruction is not

constant but intermittent. Hess and others have experienced little difficulty in passing a duodenal catheter even in the most severe case. During an operation, after the stomach has been opened, bougies of relatively large calibre can be readily passed from stomach to duodenum. There are several instances recorded (Strauch and others) in which foreign bodies of considerable size, accidentally swallowed during life, have been passed per rectum without difficulty.

We owe to Thomson the first enunciation of the more reasonable hypothesis of neuromuscular inco-ordination, against which the chief objection urged by Finkelstein, Marfan, and others is that it is difficult to understand why spasm and overaction of a sphincter should give rise to hypertrophy upon so great a scale. In the light of our knowledge of the corresponding neuromuscular obstruction at the cardia, which we owe chiefly to Hurst, it would seem tempting to consider the hypothesis that we have here to deal with what he has named an achalasia—that is to say, with a primary failure of the pyloric sphincter to relax. In spite of Hurst's refusal in a recently published paper to accept this view, I venture to think that no other hypothesis fits so well with the facts as we know them.

If we examine a radiogram (Figs. 1 and 2) of the stomach and duodenum—and, since unfortunately in infancy the duodenal cap is never apparent as a distinct shadow, it becomes necessary to use a radiogram from an adult—we find the component parts well marked. The cardiac part of the stomach functions as a reservoir; the pyloric portion forms the so-called contractile cylinder. The pyloric portion of the stomach is described as consisting of a first part, the pyloric vestibule, which is

FIG. 1.—Radiograph of adult stomach showing the shadow of the duodenal cap, a gap representing the pyloric ring sphincter, and the shadow of the lumen of the pyloric cylinder.

FIG. 2.—Diagrammatic representation of Fig. 1. D, Duodenal cap. P.R.S., Pyloric ring sphincter. P.C., Pyloric cylinder.

in general relaxed with a widely patent lumen, and the pyloric antrum (His) or the pyloric cylinder (Cunningham), the lumen of which in adults is also often widely patent, but which in the foetus and young infant after death is always obliterated by the rigid contraction of its thick muscular walls. The lumen of this pyloric cylinder is the pyloric canal. Radiographically between the duodenal cap and the shadow of the stomach adjoining is a gap less than $1/4$ in. in length, traversed from time to time or continuously by a thread-like shadow. This gap represents the true pylorus occupied by the pyloric ring sphincter.

Now when we speak of a condition of congenital hypertrophic pyloric stenosis it seems to me that we employ a term which perpetuates at least three misunderstandings. In the first place, as has been said, there is no true stenosis present, only an infolding of the lumen when the hypertrophied walls of the pyloric cylinder are in a state of rigid contraction. In the second place, it seems to me that the hypertrophy certainly involves a great deal more than the true pylorus—that is, the pyloric ring sphincter. Indeed, I hope to produce histological evidence which goes to show that the ring muscle itself takes no part in the hypertrophy, but remains unchanged in its dimensions. The great hypertrophy is disposed along the whole length of the pyloric cylinder (Fig. 3), and extends to, and involves, the vestibule as well, gradually diminishing as the cardiac reservoir is approached. The tumour formed by the hypertrophied walls of the pyloric cylinder in the infant measures on an average about $1\frac{1}{4}$ in. in length. If we are to assign all this length of hypertrophied muscle to the retaining mechanism of the stomach rather than to the expelling mechanism—

by the hypertrophied mass hardens and softens under the examining finger as each wave of peristalsis reaches it and fades away. This seems to me strange behaviour on the part of a sphincter, though readily understandable if the hypertrophy involves the walls of the contractile cylinder of the stomach and if the hypertrophied muscles are in intermittent contraction, endeavouring to force the contents of the stomach past an obstruction caused by the failure of the pyloric ring muscle to dilate.

Cunningham in his classical paper has made clear the disposition of the muscular coats of duodenum, pylorus, and stomach in the normal infant at birth. The sphincter is a well developed mass of muscle much thicker than the muscle of the wall of the rest of the pyloric cylinder. Into it passes the greater part of the longitudinal muscle fibres of the stomach coats spreading out within it as a fan-shaped fasciculus. It is this turning in of the longitudinal fibres which will enable us to identify the pyloric ring muscle proper in sections through the immensely hypertrophied muscles in so-called hypertrophic pyloric stenosis.

Cunningham, further, has a drawing of the muscular arrangement in the cat in which the sphincter proper is considerably more developed. This is of interest in view of Cannon's radiographical studies, which were carried out almost entirely on cats and which constantly show a widely dilated pyloric canal right up to the actual pylorus. Lastly, in a section which he shows through the muscle in a case of hypertrophic pyloric stenosis, it will be seen that the ring muscle is considerably less thick than the muscle of the pyloric cylinder behind it.

To compare with these illustrations from Cunningham's paper I show first a section through the pyloric region

FIG. 3.—Outline of specimen of hypertrophic pyloric stenosis showing the duodenum to the right, the cardiac reservoir to the left, and the thick-walled contractile cylinder between.

FIG. 4.—Section through the pyloric region of a normal infant, aged 4 weeks. D, Duodenum. P.R.S., Pyloric ring sphincter. P.C., Pyloric cylinder.

that is to say, if we are to regard it as a greatly hypertrophied sphincteric area of unusual length—the gap between stomach and duodenum would be radiographically represented by an hiatus, not $1/4$ in., but $1\frac{1}{4}$ in., in length. It is unfortunate that the absence of the duodenal cap in infants prevents this gap from being appreciable or measurable. It seems to me that a study of the *post-mortem* appearances shows clearly enough that the hypertrophy involves the whole contractile cylinder, which alone has peristaltic activity and the property of projecting the food forcibly through the narrow pylorus into the duodenum. Only the cardiac portion, which functions as a reservoir and which shows not peristalsis but peristole, escapes or almost escapes hypertrophy. In life nothing is more remarkable than the way in which the tumour formed

FIG. 5.—A similar section through the pyloric region in an infant with hypertrophic pyloric stenosis. D, Duodenum. P.R.S., Pyloric ring sphincter of unchanged dimensions. P.C., Pyloric cylinder with greatly thickened musculature.

of a normal infant, aged 4 weeks (Fig. 4). Again the ring muscle is much the thickest part and can be clearly identified by the turning inwards of the longitudinal fibres. In the next slide (Fig. 5) we see the condition in hypertrophic pyloric stenosis under the same magnification. The sphincter, identified as before by the passing inward of the dilator longitudinal fibres, which are here very considerably hypertrophied, is now very much thinner on cross-section than the immensely thick wall of the pyloric cylinder which lies immediately to the stomach side of it. The last section, also from a case of hypertrophic pyloric stenosis, shows the same appearance, although in this case the sphincter muscle is overlapped by and appears almost imbedded in the hypertrophied muscle of the wall of the pyloric cylinder.

I think it may be said that the histological evidence is not opposed to the view that in hypertrophic pyloric stenosis we have to deal with an achalasia of the pyloric ring muscle and that the hypertrophy involves, not the sphincter, but the contractile cylinder—that it is a hypertrophy of muscle devoted, not to the retaining apparatus of the stomach, but to the expelling apparatus. If so, the histological evidence is in agreement with the macroscopic appearances and with the clear clinical evidence of peristaltic activity in the palpable muscle mass.

The third mistake which is perpetuated in the commonly accepted name is involved in the description "congenital." If it is clear that there is no stenosis and no hypertrophy of the pylorus, it is equally clear that the condition is not congenital. Only one observer, Dent, has reported this condition in a foetus. I think it possible that he

FIG. 6.—A, The normal stomach. B, In hypertrophic pyloric stenosis—an early case with slight enlargement of the contractile cylinder. C, In hypertrophic pyloric stenosis—a late case with great enlargement of the contractile cylinder.

may have mistaken the very rigid and tightly contracted pyloric cylinder normal in the foetus for the pathological condition with which he was familiar. If the disorder were truly congenital, its frequency is so considerable that it is inconceivable that examples should not be forthcoming from among infants submitted to autopsy within the first weeks of life. In general the appearance of symptoms is delayed for some weeks after birth—usually from two to four weeks. Even when vomiting has begun and is persisting with all its characteristic violence, the evidence of hypertrophy is not immediately available. It appears only after an interval and steadily increases so long as the obstruction persists unchanged. The contractile phases are variable. Periods of exaggerated activity and increased peristalsis in the hypertrophied contractile cylinder are succeeded by periods of exhaustion and calm. Visible peristalsis, a stomach which is easily palpable and which may stand out prominently under the abdominal wall, a pyloric cylinder which can be felt as a veritable tumour, hardening and relaxing under the finger, are symptoms encountered only after some weeks of more or less persistent obstruction. The typical x-ray appearances, as shown diagrammatically in Fig. 6, also develop slowly. Fig. 7 shows the appearance on the fourth day after the first act of vomiting and on the fifteenth day of life. Clinically, no pyloric tumour was then palpable, and the evidence of visible peristalsis was slight. Radiographically the contraction cylinder is slightly enlarged with active peristalsis. Operation on the same day showed a moderate degree of hypertrophy of the muscular wall. Yet for four days preceding operation vomiting had been very violent, the weight had fallen rapidly, and the stools had become dark-coloured and slimy. In contrast with this early case, Fig. 8 shows the stomach, at the end of the fourth month of life, from an infant in which severe symptoms had been continuously present since the third week. In this case I had no operation performed, because it appeared likely, as indeed soon proved to be the case, that spontaneous recovery was imminent. The contraction cylinder shadow is enormously enlarged; the rest of the

stomach of the normal size. I believe that in this photograph, which has caught the contractile cylinder in a phase of relaxation, we have outlined the enormous lumen which after death is completely obliterated, just as an autopsy may disclose a thick-walled bladder, tightly contracted, and without any considerable lumen, which during life was subject at times to enormous distension.

In discussing the etiology of hypertrophic stenosis it is necessary to consider at the same time a group of cases in which obstruction at the pylorus is present in equal degree yet without hypertrophy of the contractile cylinder. The pyloric part of the stomach, instead of forming a cylindrical tumour of hard whitish muscle, is absolutely flaccid and shows no evidence of gross hypertrophy of its walls. Such cases are described as suffering, not from pyloric hypertrophy, but from pyloric spasm. Clinicians who have submitted to operation a large number of cases of pyloric obstruction in infancy almost without exception record the same experience. In a very small percentage of the cases the laparotomy discloses, not the expected hypertrophy, but its unexpected absence. Finkelstein, a most careful and acute observer, for long contended that a condition of persistent and almost complete closure of the pylorus without hypertrophy did not exist. Lately, as a result of an experience such as I have described, he has more than once expressed his altered view. He now regards the mistake as inevitable in a small proportion of cases. It

is true that in very many, though not in all, cases with hypertrophy the pyloric tumour is readily palpable, but the separation of the two groups cannot be made upon the strength of this one point alone. Not only are there cases with hypertrophy in which the tumour is persistently impalpable—at times it is situated high under cover of the liver margin—but there is often little difficulty in feeling a normal pyloric cylinder in systole, provided that the abdominal wall of the infant is sufficiently lax.

Persistent and severe

pyloric obstruction, with visible gastric peristalsis upon a great scale, with inanition stools and collapsed and empty intestines, yet without hypertrophy, is rarely encountered. Very many of the cases described in the literature as examples of pyloric spasm are in reality instances of other types of habitual vomiting, which I shall consider in the next lecture. In my own experience, among 32 cases of pyloric obstruction submitted to laparotomy, the hypertrophy was unmistakable in 30. Twice only was its absence disclosed. The first of these cases occurred in 1912, the second quite recently.

J. S., aged 7 weeks, was admitted to Guy's Hospital in November, 1924—a thin, eager, alert infant, with a striking degree of muscular rigidity. His weight at birth was not known; on admission it was 6 lb. 12 oz. The mother thought he had lost a great deal of weight. Throughout he had been breast-fed. During the first eleven days of life he had made satisfactory progress. On the eleventh day vomiting began, and had occurred without exception after every meal. The most careful questioning of the mother failed to shake her evidence on this point. The vomiting was violent and explosive in character. It never occurred during nursing, but always within half an hour, usually within a few minutes, after its completion. The infant was very restless, constantly crying, and ravenously hungry. For many weeks constipation had been extreme. By means of glycerin suppositories a little dark-green pigmented material had been removed every few days. In the ward gastric peristalsis was clearly visible, two or three waves being present simultaneously, passing from left to right across the epigastrium. The pylorus could not be felt. The state of the child was such that I asked Mr. Hughes to operate at once, expecting to find the usual hypertrophy of the pyloric cylinder on which a Rammstedt operation might be performed.

FIG. 7.—Radiograph showing slight enlargement of the contractile cylinder. From an early case.

FIG. 8.—Radiograph showing great enlargement of contractile cylinder. From a case of long duration.

The stomach was large and distended, the pyloric end quite flaccid and free from any hypertrophy. The small and large intestines were alike collapsed, empty, and of strikingly small calibre.

Because of the clear evidence that a state of pyloric obstruction had long existed, I asked Mr. Hughes to cut through the pyloric ring musculature down to the mucous membrane but without prolonging the incision backwards on to the pyloric antrum, as in a Rammstedt operation. The true pyloric valve muscle and that only was severed. From the moment of section of the pyloric ring the child was never sick again. Feeding was begun at once, and the amounts given were rapidly increased. On the fifth day after the operation the child had three breast feeds in no way limited, and four feeds of a milk, water, and sugar mixture. The stools within twenty-four hours of the operation became normal in colour and consistency.

I do not wish to deduce too much from a single case, but the observations made upon this infant do very strikingly support the hypothesis that the symptoms were due to an achalasia of the pyloric valve, and that the distinction between pyloric hypertrophy and pyloric spasm is not fundamental, as, indeed, was always maintained by Holt in America. In both, achalasia of the pyloric ring muscle is the primary cause of the spasm and increased tonus of the gastric musculature. In all but a minority of the cases hypertrophy of the walls of the contractile cylinder of the stomach is gradually added as a secondary feature. It may be that the reason for the absence of hypertrophy in a few instances is to be found in the ease with which in these particular cases the food escapes through the cardiac orifice. In the case which I have just quoted the regularity and severity of the vomiting was quite unusual, and might have served to awaken the doubt as to whether hypertrophy was present. It is possible that hypertrophy is only produced when, as is more usual, the escape by the cardia is only achieved with difficulty, when the stomach retains its contents for many hours, and sometimes even for days, and when phases of violent and exaggerated peristaltic activity in the effort to overcome the obstruction are prolonged. After spontaneous recovery from all symptoms the pyloric tumour for long remains palpable, and the peristalsis may remain visible for months, or even years. Numerous autopsies upon children who had formerly suffered from pyloric stenosis have disclosed the persistent hypertrophy of the pyloric cylinder. Since hypertrophy often persists long after the cessation of all symptoms, and since symptoms of great severity and typical in character may occur without hypertrophy, it would appear necessary to presume the presence of a primary achalasia in all cases.

It remains to consider one further point. Will simple section of the pyloric ring muscle at once relieve the symptoms in a case with typical hypertrophy, or must the incision be prolonged backwards over the pyloric canal to sever the whole hypertrophied circular coat of the contractile cylinder?

I have hesitated to put this matter to the test in an individual case because it is clear that the vomiting is not due only to the failure of the pyloric ring muscle to open. The explosive character of the vomiting is determined, not by the pyloric obstruction alone, but also by the gastric hypertrophy and spasm. In purely organic stenosis the vomiting is never so violent, nor is there produced the picture of the spastic stomach with its outline clearly visible beneath the abdominal wall. Sometimes, indeed, the spasm extends to the pharyngeal muscles, so that during the height of the exaggerated gastric activity the infant may be unable to swallow. It is highly significant that there are a few autopsies upon record which have disclosed hypertrophy of the lower end of the oesophagus as well as of the contractile cylinder, presumably a combination of cardiac and pyloric achalasia. Thus, for example, Pritchard, in 1920, at the Royal Society of Medicine exhibited a specimen with hypertrophy of the lower end of the oesophagus, and hypertrophy of the "ileo-caecal sphincter" combined with hypertrophic pyloric stenosis. In this disorder of the innervation of the gastric musculature neither the failure of the pyloric ring muscle to open nor the spasm of the hypertrophied contractile cylinder above it is of itself sufficient to account for the symptoms. Both must be present at the same time. Both are parts of the same disturbance in the production of the syn-

chronous contraction above and relaxation below which together make up the peristaltic wave. The presence of a sphincter in any situation in the bowel does not alter this primary relationship between contraction and relaxation, although functionally it may throw a greater emphasis upon the movement of relaxation. In this disorder of peristalsis it seems a mistake to lay emphasis solely on the excessive contraction or solely on the failure of relaxation. Both phases are involved. Only if we can imagine a peripheral stimulus going out from the ring muscle itself can we conclude that it would be sufficient to divide that alone in order to do away with the spastic contracture at the higher level. If this were to prove the case, then the Rammstedt operation, simple and successful as it is in the great majority of cases, would be still further simplified, because complete incision of the mass of hypertrophied muscle would no longer be needed and the risk of haemorrhage would be reduced to a minimum.

If it is tempting to hypothecate a uniform cause for all these infantile disorders marked by inco-ordination of the gastro-intestinal musculature and to regard all as accompanied by achalasia of the several sphincters, we must not at the same time lose sight of the difference between the local conditions prevailing at each site. Behind the pyloric sphincter, but not behind the cardiac, is a segment of the gut which has a highly evolved propulsive mechanism. Above the cardia, in achalasia of that sphincter, the hypertrophy is slight, perhaps even at times inappreciable, because the pressure within the oesophagus is low and the cardia opens in response to a minimal stimulus. In the oesophagus, as in the auricle of the heart after death, dilatation rather than hypertrophy is a prominent feature; in the thick-walled contractile cylinder of the stomach, as in the main propulsive chamber of the heart, the ventricle, it is the hypertrophy which is most obvious. It is possible that simple section of the ring muscle may suffice at the cardia, yet fail at the pylorus.

The view that the primary cause of the spastic obstruction of the pylorus is due to a primary hyperaesthesia of the mucous membrane with an accompanying excessive secretion of gastric juice seems disproved by the immediate success of the muscle-splitting operation. The secretory changes would appear to be secondary to the disorder of innervation.

B. OBSTRUCTION AT THE ILEO-CAECAL VALVE.

At the ileo-caecal valve it appears likely that in early infancy a similar failure to inhibit the ring musculature is occasionally met with. In the following case an achalasia of the ileo-caecal valve was apparently combined with some degree of anal achalasia.

K. McD. was seen by me in Guy's Hospital on the ninth day of life. For the first three days after birth nothing abnormal had been noticed, although less meconium than usual had been passed. Vomiting began on the fourth day, and from the fifth day onwards had been continuous. The vomited matter was stained with bile. On that day, the fifth of life, he was admitted to hospital. An x-ray examination after a barium meal showed only a trivial delay of the barium in the duodenum. Within ten minutes it was seen in the jejunum. In the ward, between the fifth and the ninth days, when I first saw him, vomiting of bile-stained fluid had taken place on several occasions, but the most striking symptom was the obstinate refusal of the bowels to act, in spite of the prescription of various purgatives, such as hydr. cum cret., castor oil, and finally pituitary extract hypodermically. Enemata of soap and water had likewise failed of effect, until on the ninth day an olive-oil enema had produced the passage of a very small amount of yellow faecal material. Since the early passage of meconium in the first days of life nothing had been passed in the interval. On this, the ninth, day I saw the child. The abdomen was moderately distended, and there were visible peristaltic waves, apparently of the small intestine. On the twelfth day a second x-ray examination was made to ascertain what had become of the barium taken on the fifth day. I show the radiogram. The colon is considerably distended, although perhaps the distension is most apparent in that part which is empty of barium and distended only with gas. The dilated coils of small intestine distended with gas and containing a little barium are also apparent. Rectal examination showed nothing abnormal, but on exposing the abdomen the visible peristalsis in the small intestine was now very striking, the whole contour being broken into a series of ridges and furrows. The bowels were slightly open during this examination, and flatus was passed freely.

Confronted by this situation it was difficult to know how to act. Since the visible peristalsis appeared to be due to the small and

not to the large intestine, the diagnosis of Hirschsprung's disease appeared to be unlikely. On the other hand, the presence of the barium, so long stationary in the colon, appeared to exclude any obstruction in the ileum.

Since the general condition of the child had steadily deteriorated and much weight had been lost, at my request Mr. Steward performed a laparotomy. At once very greatly thickened and hypertrophied coils of small intestine presented in the wound. Much of small intestine had to be allowed to escape from the abdomen before it was possible to trace this hypertrophied gut to the neighbourhood of the ileo-caecal valve, where, at a point about one inch above the caecum, the calibre suddenly narrowed and the congestion marked above gave place to a paler colour. The caecum and first part of the ascending colon appeared considerably distended. There was no sign of obstruction by a band or by a diverticulum. With considerable difficulty the intestines were replaced and the abdominal wall sutured. The child at first seemed little the worse for the operation. Hypodermic injections of atropine sulphate gr. 1/500, at four-hourly intervals, were given without appreciable benefit. The bowels were open once after the operation, but the distension steadily increased, and the child died four days after the operation. No *post-mortem* examination was permitted.

The case can, I think, only be explained on the assumption of an achalasia of the ileo-caecal valve with secondary spasm and hypertrophy of the bowel above, combined with a similar, though less marked, achalasia of the anal sphincter. In the literature few similar cases have been described, at least in early infancy. I can refer only to the paper by Wilms on persistent intestinal spasm, and to an exhibit before the Anatomical Society of Paris by Gaikler and Nau of three foetuses, each with multiple spastic strictures of the intestine. I have notes of one other similar case seen with Mr. Hughes, in which the thickened small intestine was both visible and palpable. The baby was sent to me from the West Norwood Maternity Home on the fourth day of life, with bilious vomiting, abdominal distension, and extreme constipation. From the tenth day onwards recovery gradually took place.

C. OBSTRUCTION AT THE ANUS AND IN THE RECTUM.

The etiology of Hirschsprung's disease, like that of hypertrophic stenosis, has been greatly discussed. Hale-White, Fenwick, and Hawkins in this country have upheld the view that neuromuscular inco-ordination in some particular segment of the bowel is responsible. Two groups of cases are described. In that which is most characteristic, and to which the name "Hirschsprung's disease" should perhaps be confined, the symptoms of absolute constipation and abdominal distension date from birth. There may be difficulty even in the passage of meconium. In a well marked case the symptoms are developed in a quite characteristic way before the child is more than a few days old. I show a photograph taken in the *post-mortem* room of an infant at one time under my care, who died on the tenth day of life in Guy's Hospital; also the photograph of an older child, aged 3 years. The great distension of the abdomen, through the wall of which the peristalsis of the hypertrophied colon was clearly visible, is well shown. Of the first case I was not able to get a satisfactory x-ray plate, but of the second case I show the shadow of the huge colon in its lower part outlined by means of a bismuth enema. The dilatation passes downwards to the anal sphincter itself. With such a shadow there can be no question of the dilatation and hypertrophy being a secondary result of obstruction by organic stenosis of the bowel or by kinks, acute angles, or spurs. In three other cases, which I have had the opportunity of examining in early childhood, the same picture—the extension to the anus itself—has been seen. It is clear that the cause must be found in a failure of relaxation in the anal sphincter. In other cases the achalasia may affect the other sphincters described by Keith in the rectum and sigmoid. Behring and Klercker have recently reviewed the literature and brought forward additional proof of the origin of Hirschsprung's disease in localized *Kontraktionsringen* with resulting secondary changes. In a second group of cases, which show the syndrome of Hirschsprung's disease, the origin is probably different. As a rule, the onset of symptoms is delayed until later life. Many show acute angular kinks of the colon, with adhesions between adjoining loops of the bowel or with the lumen, large as it is, partially

occluded by infolded spurs or valves. These doubtless are secondary effects resulting from many causes and perpetuating the obstruction. It is possible that some cases which in early infancy were examples of anal or rectal achalasia with secondary hypertrophy, distension, and elongation of the colon, may pass gradually into the second category, and that the symptoms may be perpetuated by secondary organic obstruction due to acute kinking of the bowel with spur formation long after the infantile lack of co-ordination has been overcome. In other adult cases achalasia may be the result, not of developmental delay as in infancy, but of degenerative changes in the central nervous system involving the higher central connexions of the inhibitory paths. In the recorded cases a disproportionately large number appear to have been encountered in idiots.

D. OBSTRUCTION AT THE CARDIA.

Beck and Göppert have both described carefully observed cases of temporary dysphagia in infants due to so-called "cardiospasm."

In Beck's case the infant was a male, aged 3 months. The regurgitation of milk in an unchanged condition, and quite unmixed with stomach contents, was the main symptom. Milk introduced directly into the stomach was never vomited. The act of swallowing was clearly involved, and there was much gurgling and choking. A sound encountered an easily overborne obstruction 15 cm. from the gums. At the height of the disturbance there was a loss of weight of 400 grams. The disturbance lasted for two months, when complete and permanent recovery took place. In a second case, also reported by Beck, the duration was shorter—four weeks.

Göppert had a similar experience. In the first of his cases, however, complete recovery did not take place until the ninth month of life. Hess, in passing his duodenal catheter in newly born or very young infants, noted that there is not infrequently some slight obstruction, easily overborne, encountered at the cardiac orifice.

It is probable that in the infant whose diet is wholly fluid obstruction at the cardiac sphincter is too intermittent and too easily overcome commonly to give rise to persistent symptoms such as Beck and Göppert have described in exceptional cases. Persistent cardiac achalasia of adult type is not infrequently found in young children, but not, I think, in infancy.

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