There appear to be no figures to indicate the incidence of carcinoma, in general, in relation to pregnancy, but clinical experience suggests that it is, fortunately, low. On general principles it would appear that growth and spread are more rapid than the average when the neoplasm is associated with a cyesis, and in the case of carcinoma of the cervix there is some evidence to prove that this is so.

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VOMITING IN THE NEW BORN

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I see vomiting in the newly-born from rather a specialised angle. Most of the children met with have been seen by a physician first, and such conditions as are purely medical have usually been treated. I propose to describe Hypertrophic Pyloric Stenosis, and then to recall some of the patients suffering from other conditions for whom my assistance has been sought.

Hypertrophic pyloric stenosis has become a widely recognised disease. Vomiting, the first symptom, usually occurs about the second to the fourth week, and it occurs in a previously healthy infant. At first, it is projectile. It occurs usually towards the end of a feed, or after the whole feed has been taken. The absence of the projectile nature of the vomit is of no great significance. If the food has remained in the stomach any length of time before it is vomited, the vomit is acid in re-action. The vomited material never contains bile. In an unsuccessfully treated or untreated child the vomited material changes in character, and may become dark coffee ground in appearance, and contain altered blood.

The first vomit may occur earlier in life than the second week, and two children stand out in my memory in this respect, one a seven-month premature infant, who vomited soon after birth, and upon whom I operated about fourteen days after birth to find a definite tumour. The second child lived in the North of England and was the third in its family. I had operated on its two elder brothers for hypertrophic pyloric stenosis, and on the third day after the birth of the third child the mother said it had hypertrophic pyloric stenosis as it behaved as its brothers had done. I opened its abdomen in spite of the fact that there was no palpable tumour. The stomach and pylorus appeared normal. I did nothing at all and closed the belly. The child gained half a pound, and then began to lose weight and vomit again. After three weeks, when the child had dropped one pound in weight, I re-opened it and found a definite pyloric tumour which was split in the usual way. The child then made an uninterrupted recovery.

These two observations suggest, firstly, that hypertrophy of the pylorus can occur *in utero*, and secondly, that there is a stage in the development of the disease before hypertrophy occurs, when a palpable tumour is not present.

The vomiting is usually associated with increasing constipation. The typical stool is hard and dry, but normal in colour. If the vomiting is accompanied by frequent green stools the prognosis is adversely affected. During the early stages of the disease the child is bright, alert and hungry, but as the disease progresses the child becomes loose-skinned, sunken-eyed; its colour changes to a dull bluish grey, and it becomes lethargic and dull, having vomited much of its fluid content, and the acid in its stomach. It becomes alkalosed and suffers from uraemia. The blood urea in one child, Master D., had risen to 90 milligrams per 100 c.c. of blood, and the infant was being treated by the medical man in charge for nephritis! This child survived operation, and is now a boy of seven years of age. This baby illustrates the dictum that it is never too late to operate.

Hypertrophic pyloric stenosis is one of those fortunate conditions which has an infallible sign, when the condition has become well established, viz., the presence of a palpable tumour. If this tumour can be felt, it is diagnostic. It is usually to be palpated a little to the right of the mid-line midway between the ensiform and the umbilicus. The tumour can be felt best when it is in systolic. Hence it is necessary to encourage the child to suck. A finger covered by a sterile finger stall may be placed in the infant's mouth, or it may be given a feed. It is not possible to palpate the abdomen of a crying infant and make a positive diagnosis. The examiner's hands should be warm, the room should be warm, and ample time must be expended. A segmented rectus muscle and a Reidal's lobe of the liver are two factors to be excluded. If a tumour is not felt at the first examination it should be repeated.

Visible peristalsis is observable in a number of normal infants. In pyloric stenosis it may be peculiarly violent, and is usually easily observable.

It is quite a simple matter to pass a stomach tube on an infant. There should be little gastric residue in the stomach of a normal infant four hours after its feed, that is, immediately preceding its next feed. In pyloric stenosis especially, four hours after a feed that has not been vomited there is nearly always a gastric residue. If a persistent gastric residue is found, a diagnosis of pyloric stenosis is probable.

The X-ray findings are not uniformly consistent, and as an experienced observer can feel a tumour if one be present, the barium meal has little place in the diagnosis. The X-ray should show a large stomach with active peristalsis and little barium leaving the pylorus after one, two or three hours. Sometimes, however, the barium does go through, although there is hypertrophy of the pyloric sphincter.

A congenital short oesophagus is a lesion which must be present from birth. In adults, this lesion is becoming increasingly widely recognised. On one occasion, there were three such patients in hospital at the same time. Yet I have visited large hospitals and there have been no records of any such condition being diagnosed. All depends on whether the radiologist screens the lower end of the oesophagus during the barium swallow examination. The reason the condition is more usually diagnosed in adults is because in them it is often complicated by ulceration and consequent spasm. This leads to pain and to the signs of oesophageal stricture; often a mistaken diagnosis of oesophageal growth is made. The X-ray appearance of the lesion in the adult is shown in Fig. 1. I have been fortunate in being able to recognise this lesion in two infants, and I think that it will be increasingly recognised as a cause of vomiting in the newborn.

The first child, Dr. F.'s patient, was two weeks old, was bottle fed, and vomited. It had been doing this for a week, and when seen was below birth weight. On watching this child fed, it swallowed a part of its feed quite well. It then paused for a time, took two or three more mouthfuls, and then vomited; but it only vomited the last two or three mouthfuls, and appeared to us all that it filled its oesophagus and then vomited.

The other child was a second child. Its elder brother had been a vomiting baby but had recovered. This child vomited during and after its feed. This child had no pyloric tumour, and it was examined by means of a barium swallow. The lower end of the oesophagus was visualised, and it was seen to end some way above the diaphragm. An X-ray film was taken, but it is not good enough to reproduce well in this JOURNAL.

Both these children were cured by the passage of a relatively large-size tube down the oesophagus. The exact procedure was as follows:—The child was weighed and fed. If it vomited during the feeding it was still given the rest of the bottle. It was weighed after its feed if it had vomited. The amount of the vomit could thereby be ascertained. A tube was passed, and food was introduced into the stomach equivalent in quantity to the amount of

FIG. 1.—Congenital short oesophagus in adult.

the vomit. The passage of the tube appears to dilate the sphincter muscles, and the necessity of passing the tube gradually diminishes. The opportunity of re-examining these two infants has not occurred yet, but it is hoped to X-ray them both when they are two years old.

Vomiting may start from the moment the child is born. This type of obstructive vomiting may be caused by a very large number of varying factors. The most common are the malformation of the gut. The duodenum may fail to canalise in the region of the second part. If this occurs the bile duct often opens in the cephalic segment and the vomit contains bile. The small gut may exist as a series of disconnected segments like a string of sausages. The ileo-caecal valve may not be patent. The rectum may have a septum across it. It may open into the vagina by means of a small foramen, or the whole rectum may be absent, the colon end as a blind end high in the pelvis. There may be an internal obstruction caused by mal-rotation of the gut, by bands, and especially the remains of a Meckel's diverticulum. The gut may be strangulated in a hernia. In one child it was an inguinal hernia, and in another it was a diaphragmatic hernia.

I have seen and treated patients suffering from each of the conditions mentioned. Every child in this series vomited bile except those with imperforate anus. These were recognised by the gynaecologist and sent for treatment immediately. All these infants possess an extraordinary vitality and all cling to life tenaciously. Hence it is always worth while doing what is possible, although I have not found that a child with a colostomy does well, and I feel that this operation should be not performed in babies.

Children with duodenal stenosis fall into two main groups: those with absolute obstruction, and those with a partial obstruction. The malformed segment may be a fibrous cord, or it may contain a lumen. The lumen may be small, and if this be the case the stomach and proximal duodenum will hypertrophy to force the contents through the narrowed area. There is no hypertrophy of the pylorus; no palpable tumour, and the vomit contains bile. X-ray examination does help with those infants. Sometimes, the stenosed duodenum can be visualised and the enlarged first part of the duodenum seen. The contents of the stomach are not passed on, and there is a marked gastric residue. Dr. Kenneth Tallerman and I reported at the Children's Section of the Royal Society of Medicine one example of this complete duodenal stenosis on whom I performed a gastro-enterostomy, and who is now 7 years old.

Children with multiple congenital abnormalities do not respond well to treatment. If there are multiple areas of stenosis in the bowel my impression is that there are defects in the nervous mechanism of the intestinal track, and I have not had success in any instance where multiple anastomoses of the bowel were required. Children with multiple congenital small bowel structures always vomit from birth, and the vomit contains bile. The child's abdomen is very distended, and often there is visible small bowel peristalsis presenting as the old-fashioned ladder patterned abdomen. These infants with hopelessly imperforate bowels live for days after operation, and are a source of great anxiety to the parents.

Strangulation of bowel in an inguinal hernia does not often occur in an infant. One child eighteen days old, covered by infantile eczema, was vomiting faeces when it was first seen. This boy was under the care of a very capable doctor, who had called in a consulting physician to see the infantile eczema. Sulphonamide was prescribed. The vomiting caused by the hernia was ignored, as it was (quite unjustifiably) stated that operation was out of the question owing to the septic skin condition. When the infant commenced to vomit faeces the doctor decided to send for a surgeon. Operation was performed at once, despite the infantile eczema. A strangulated coil of bowel was released, and the infant made an uneventful recovery.

Obstetricians appear to have formed a habit of inspecting the anus of the children they deliver, for every child referred with an imperforate anus has come before obstructive symptoms were marked, and with a definite diagnosis. If there is but a thin septum and the anal musculature has developed the immediate and ultimate prognosis is excellent. If the septum be thick, if there are no anal muscles, or if the rectum opens into the vagina, the patient is liable to develop an uncontrolled faecal fistula in the perineum. These fistulae show a tendency to contract.

Another interesting infant commenced to vomit within one week of birth. When vomiting, this child became very blue, and held his breath. There was no palpable gastric tumour, and the condition puzzled everyone. A few days after the child had been seen, the mother developed whooping cough. The child died.