This adds to the problem, because if latent animal viruses exist in this state they would not be detected by ordinary isolation procedures, because virus material would not be in a form which could infect new cells. Some inducing agent would be needed which would upset the equilibrium and allow the virus to mature to a fully infectious form, as in the induction of lysogenic bacteria by ultra-violet light. There is no clear evidence at present, however, that latent virus infection of human or animal cells resembles that of bacterial cells. Further progress awaits a suitable model of latent infection in tissue culture which could be studied by the quantitative techniques which have been so fruitful with lysogenic bacteria.

A more or less undetectable latent virus which persists indefinitely without damaging the cell in the ordinary sense may anyway seem rather an academic nicety. Absence of recognizable histological change, however, does not mean that the continued presence of viral material in a cell will not alter some of its functions. For example, the tumour viruses change the potentiality of the cells they infect without necessarily causing necrosis. Latent virus infection of a bacterium may confer entirely new characters, such as the development of flagella or toxin production, on the otherwise normal host. We have yet to see whether viruses may cause long-standing alterations of cell function in man.

Conclusion

We have now indulged in a good deal of speculation and have come some way from practical medical problems, but, judging by the numbers of latent viruses found in animals, our own tissues may harbour more unobtrusive guests than we imagine. For the survival of a virus or any other parasite it is, of course, the most satisfactory arrangement. No damage to the house—no trouble with the landlord.

When symbiosis is complete, especially if early infection leads to immune tolerance, we may expect few medical problems. These will arise only when artificial procedures, like blood transfusion, carry the virus to someone who has escaped early infection. Nevertheless, this risk will call for great care if virus vaccines are ever made from human cell cultures, which are more likely than blood to carry these hidden agents.

With other infections, like herpes and Brill's disease, the balance may occasionally be upset with a sudden acceleration of virus production and cell damage, causing illness in the carrier. Some sporadic afflictions of tonsils and adenoids, or even other lymphoid tissue, may eventually prove to be due to recrudescences of infection with adenoviruses. Such conditions lack many of the usual attributes of an infectious disease, and their recognition as latent virus infections will involve subtle problems for the virologist as well as the clinician. It is to be hoped that investigation of the more fundamental problems of the mechanism of latency, as outlined, may ultimately be of practical value in the elucidation of these diseases.

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ADULT HYPERTROPHY OF THE PYLORUS

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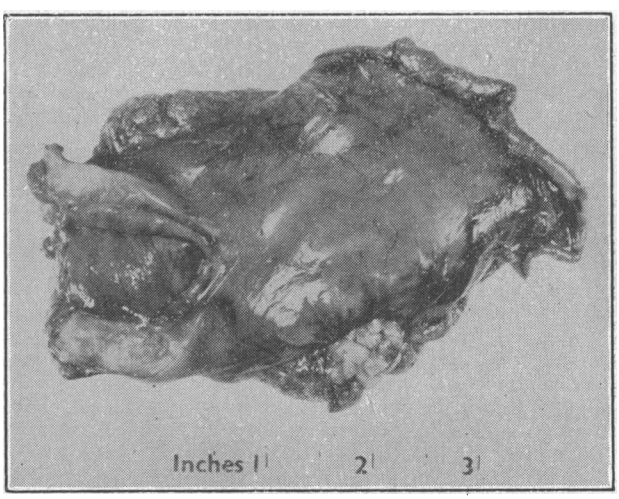
[WITH SPECIAL PLATE]

Pyloric hypertrophy in adults has in the past ten years been found 25 times during laparotomy at St. James's Hospital. Fewer than 200 cases have been recorded in the literature since the condition was first described by Cruveilhier over 100 years ago, and many of these were discovered only at necropsy. Horwitz *et al.* (1929) found that the normal thickness of the pyloric muscle varied from 3.8 mm. to 8.5 mm.; in all our cases it has been 1 cm. or more (see photograph). Furthermore, in all cases the hypertrophy was mainly confined to the circular muscle.

Only once has a confident pre-operative diagnosis of the condition been made, but increasing experience has enabled us to form a strong suspicion of its existence from the radiographs after a barium meal in a few



Resected stomach from Case 6, showing incised thickened pylorus (1.3 cm.).

other cases. The majority of patients have had co-existent duodenal or gastric ulcers, and it is the symptoms of these which have brought them under our care.

Treatment has therefore been directed to cure of the ulcer. The accompanying table summarizes the findings in these cases.

History

In no case were we able to obtain a clear-cut history of vomiting in infancy suggestive of congenital pyloric stenosis. One or two patients stated that in childhood they had suffered from bilious attacks with vomiting, and one gave a history of migraine with vomiting dating back to childhood. It may be significant that in this case the migraine has been

Table Showing Details of 25 Cases of Adult Hypertrophy of the Pylorus Admitted for Treatment of Peptic Ulcer

Case No.	Age at Onset (Years)	Age at Operation (Years)	History of Congenital Pyloric Stenosis	Ulcer Dyspepsia	Vomiting	X-ray Findings		Gastroscopic Findings	Procedure	Operative Findings	Remarks
						Original	Retrospect				
1	50	58	Nil	+	+	Prepyloric ulcer	—	—	Pyloroplasty	Pyloric hypertrophy	
2	46	48	,,	+	−	Duodenal ulcer with retention	Same as original	Inconclusive	Billroth-I gastrectomy	Pyloric hypertrophy; mucosal stenosis; gastric ulcer	No external evidence of ulceration at operation
3	19	28	,,	+	+	Duodenal ulcer	Poorly filled, irregular cap	—	Polya-type gastrectomy	Pyloric hypertrophy; duodenal ulcer	
4	46	50	,,	+	+	,, ,,	Narrowing of canal; bulbar impression	—	Billroth-I gastrectomy	Pyloric hypertrophy; gastric and duodenal ulcers	No external evidence of ulceration at operation
5	24	49	,,	+ with haemorrhage	−	Normal	Slight bulbar impression	Normal	,, ,,	Pyloric hypertrophy; duodenal ulcer	
6	38	53	,,	+	−	—	—	Gastric ulcer	,, ,,	Non-rotation of gut; pyloric hypertrophy; 2 gastric ulcers	
7	30?	53	,,	+	−	Gastric ulcer with retention	—	—	Polya-type gastrectomy	Pyloric hypertrophy; gastric ulcer	
8	39	43	,,	+	−	Hyperperistalsis	—	Gastric ulcer	,, ,,	,, ,,	
9	39	48	,,	+	++	Duodenal ulcer	—	—	,, ,,	Pyloric hypertrophy; high gastric ulcer	
10	55	60	,,	+	Self-induced	Gastric ulcer; duodenitis	—	Gastric ulcer	Billroth-I gastrectomy	,, ,,	
11	32	42	,,	+	++	Duodenal ulcer; prestenotic diverticula	—	—	Polya-type gastrectomy	Pyloric hypertrophy; duodenal ulcer	Specimen R.C.S. No external evidence of ulceration at operation
12	27	42	,,	+	+	—	—	Gastric ulcer	Billroth-I gastrectomy	Pyloric hypertrophy; gastric ulcer; mucosa prolapse	
13	28	28	,,	Vague	−	Pyloric ulcer with retention	—	—	Pyloroplasty	Pyloric hypertrophy	
14	?	59	,,	+ with haemorrhage	+	Elongated canal; duodenal ulcer with retention; ?pyloric hypertrophy	Elongation and narrowing of canal; proximal and distal impressions; pyloric hypertrophy	Blood-clot on narrowed antrum	Billroth-I gastrectomy	Pyloric hypertrophy; gastric ulcer	No external evidence of ulceration at operation. Hypertrophy diagnosed pre-operatively
15	42	47	,,	+	−	Duodenal ulcer with retention	—	—	Polya-type gastrectomy	Pyloric hypertrophy; mucosal prolapse	
16	40	40	,,	−	+	Normal	Bulbar impression; elongation and narrowing of canal	—	Ramstedt operation	Pyloric hypertrophy	Laparotomy performed for recurrent intestinal obstruction due to bands
17	20	70	,,	+	−	Duodenal ulcer with retention	Elongated pylorus; deformity of cap; retention	—	Gastro-enterostomy	,, ,,	
18	66	68	,,	+	+	Elongation and narrowing of canal surrounding tumour; proximal and distal impressions; pyloric hypertrophy	Same as original	—	Billroth-I gastrectomy	Pyloric hypertrophy; pyloric ulcer	Hypertrophy diagnosed pre-operatively. Radiologist reported palpable tumour during screening
19	34	54	,,	+	+	Duodenal ulcer with retention	—	—	Polya-type gastrectomy	Pyloric hypertrophy; gastric ulcer	
20	37	47	,,	Not typical	+	Small deformed bulb	Peaking of pylorus; very small cap	—	Pyloroplasty	Pyloric hypertrophy; mucosal stenosis	Relief of symptoms
21	20	50	,,	Atypical		Duodenal ulcer	Same as original	Small scar; gastric ulcer	Billroth-I gastrectomy	Pyloric hypertrophy; duodenal ulcer	
22	68	70	,,			Duodenal ulcer; 24-hour retention	,, ,,	—	,, ,,	Pyloric hypertrophy; gastric ulcer; mucosal stenosis	
23	20	43	,,			Deformity of cap	,, ,	Gastric scar	Polya-type gastrectomy	Pyloric hypertrophy; gastric ulcer	
24	19	40	,,			Duodenal ulcer with retention	,, ,,	—	,, ,,	Pyloric hypertrophy; duodenal and gastric ulcers	
25	28	35	,,			Duodenal ulcer with retention	,, ,,	—	Pyloroplasty; cholecystectomy	Pyloric hypertrophy; gallstones	Patient a pure negro

cured, at least temporarily, by partial gastrectomy. In 15 of the 25 cases vomiting was a prominent symptom, and it was profuse in four, two of which had a duodenal ulcer and two a gastric ulcer. In 18 cases concomitant peptic ulcers were confirmed at operation, and all but one of these presented with ulcer-type dyspepsia. It is interesting that five of the remaining seven cases also gave typical ulcer histories. In another case the condition was an incidental finding at an emergency operation for obstruction due to bands; and the last case had a history of vague discomfort, waterbrash, and a sensation of choking. In short, ulcer-type dyspepsia was the main feature whether or not the presence of an ulcer was confirmed, and vomiting was frequently a symptom.

If there is an aetiological connexion between peptic ulceration and adult hypertrophy of the pylorus, it might be expected that the site of the ulcer would be fairly constant, but this does not appear to be so, since in 11 cases it was gastric, in four duodenal, in two both gastric and duodenal, and in the other pyloric.

We have been unable to discover any symptom or group of symptoms which would help in establishing a confident diagnosis. A history of profuse vomiting for many years associated with an atypical history of ulcer dyspepsia, without gross gastric retention, might lead one to suspect the condition.

Physical Signs

Here again no diagnostic facts have been elicited. Epigastric tenderness has been a constant sign in cases with or without ulcer. A tumour has never been felt on clinical examination, but in Case 18 the radiologist felt a pyloric tumour during screening. In seven cases a succussion splash was noted, and in these a large gastric residue was apparent on *x*-ray examination. Four of them had a gastric ulcer, and in three the hypertrophy was the only finding. Curiously, none of the cases associated with a duodenal ulcer had a splash or showed gross gastric retention on *x*-ray examination.

Radiological Findings

The *x*-ray findings are reported in two ways: as original reports and as reports in retrospect. Two cases were not examined radiologically, the diagnosis of gastric ulcer being made in each case by gastroscopy. In nine cases a retrospective examination of the radiographs has not been possible.

Although duodenal ulcer was found at operation in only six cases, a pyloric or duodenal ulcer was reported in 17. The reports on the remainder are in summary as follows: no lesion seen, 2; hyperperistalsis, 1; gastric ulcer with retention, 1; gastric ulcer with duodenitis, 1; probable adult hypertrophy of pylorus, 1. Thus the diagnosis was suggested with confidence in one case only, though in another two it was mentioned as a possibility.

Retrospective examination of the barium-meal radiographs has been interesting. In the two cases in which it was reported that the condition might be present it is now obvious that the report could have been given with confidence (see Special Plate, Figs. 1 and 2). In seven cases no reason could be found to modify the original report. Two of these had a duodenal or pyloric ulcer, three others a gastric ulcer with gross mucosal stenosis and in which no cap could be demonstrated, and one both gastric and duodenal ulcers. In five other cases revised reports were as follows:

Case 4.—There is narrowing of the pyloric canal with a slight duodenal bulbar impression; the canal is not elongated. Operative findings: gastric ulcer and duodenal ulcer with hypertrophy.

Case 5.—The canal is normal but there is a slight duodenal bulbar impression. Operative findings: duodenal ulcer and hypertrophy.

Case 16.—There is elongation and narrowing of the pyloric canal with a duodenal bulbar impression. Operative findings: pyloric hypertrophy only.

Case 17.—There is elongation but no narrowing of the pylorus with a deformity of the duodenal cap. Operative finding: pyloric hypertrophy only.

Case 20.—There is slight peaking of the pylorus with a very small cap.

Gastroscopy

This was performed in eight cases. Results in two were inconclusive and in one normal; five had gastric ulceration or scarring. In only one was narrowing of the pylorus noted, but blood-clot in the antrum prevented close examination of the mucosa.

Operative Findings

In all cases at laparotomy the diagnosis was reasonably obvious. In an early case, to exclude the possibility of carcinoma, a small incision was made in the pylorus. This manœuvre removes all doubt. The white, regular, glistening muscle cannot be confused with the grey, irregular infiltrative type of carcinoma. The serosa is normal, and the submucosa loose and lax and easily defined. At the same time a small piece can be taken for microscopical examination (Tanner, 1950).

In 11 cases there was no evidence of a concomitant lesion of the stomach or duodenum on thorough inspection and palpation. In five of these a gastrectomy was performed, and the excised specimen revealed scarring or shallow ulceration in either stomach or duodenum in all but one case. In four cases a Heineke–Mikulicz pyloroplasty was performed, in one a Ramstedt operation, and in one a gastrojejunostomy. The last patient was a man aged over 70; his general condition was not good and the operation was carried out under local analgesia. In the 14 cases where a peptic ulcer was found a suitable gastrectomy was performed to cure this.

The state of the pyloric mucosa excited interest in five cases, three of which had such a degree of mucosal stenosis that only a moderate-sized probe could be passed; two of the three also had a gastric ulcer, but in the third no ulcer was found. The other two cases presented small protrusions through the pylorus into the duodenum, about 2.3 mm. in diameter. In both instances these were thought to be polypi, but section showed normal musculo-mucosal tissue. The exact nature of this is doubtful, but we think it is an abnormal form of mucosal prolapse due to the hypertrophied pyloric muscle preventing normal reduction. The cause of mucosal stenosis is obscure, but we feel that it must be inflammatory in origin, possibly from an acute pyloric or duodenal ulcer. It may be due to some degenerative change secondary to mucosal prolapse.

An associated congenital lesion was found in Case 6—namely, non-rotation of the gut. This patient had a gastric ulcer and was subjected to a Billroth-I gastrectomy.

There were no deaths in the series, and post-operative convalescence was uneventful in all cases. Two of the earlier ones have not been contacted recently, but the remainder are all known to have satisfactory relief of their symptoms.

Discussion

Various theories concerning the aetiology of pyloric hypertrophy in adults have been advanced, of which the most probable is that it is a persistence of the infantile form. Considering the amount that has been written on the immediate results of treatment of congenital hypertrophic pyloric stenosis, there is remarkably little information on the ultimate fate of the tumour. In careful follow-up studies of babies treated medically Runström (1939) has shown that the condition tends to resolve slowly and an abnormality may be detected for many years.

There seems to be fairly general agreement that a Ramstedt operation is followed by complete disappearance of the tumour (Wollstein, 1922). The very few "failures" have all required a second operation within a few days or weeks of the first, and have usually been attributed to

incomplete division of the circular muscle fibres. In no case, so far as we have been able to ascertain, has the tumour been shown to have persisted for more than a few weeks after this type of operation. Two patients at St. James' Hospital who had Ramstedt's operation in infancy have undergone laparotomy in adult life, one for gastric ulcer and the other for recurrent appendicitis. In both the pylorus felt normal and there appeared to be no persistence of the hypertrophy.

If the infant is treated by gastro-jejunostomy the tumour frequently remains unaltered (Holt, 1917), even well into adult life (Donovan, 1946; Walters, 1946; Armitage and Rhind, 1951; and others). Indeed, the cases recorded suggest that the tumour nearly always does persist after gastro-jejunostomy, though this may be an erroneous impression, as the absence of a tumour at a subsequent operation may be overlooked or not regarded as worthy of record. Be this as it may, it is difficult to imagine why a short-circuiting operation will lead to persistence of the tumour while other forms of treatment will cause its disappearance. We agree with McCann and Dean (1950) that the tumour will persist in a proportion of medically treated or subclinical cases and will be discovered only at necropsy, at operation for some other condition, or when some additional mechanical or inflammatory factor leads to increased stenosis with the production of symptoms.

The frequent finding of a peptic ulcer in association with the condition indicates that there may be some relationship between the two. It has been postulated that stasis is conducive to peptic ulceration: several papers have drawn attention to the incidence of duodenal ulceration in duodenal ileus, and recently Johnson (1955) has suggested that gastric ulceration may follow pyloric stenosis. The hypertrophy, then, may explain the gastric ulcers, but would not account for the duodenal ulcers.

Kirklin and Harris (1933), reviewing the literature and describing 81 cases seen at the Mayo Clinic, list the following radiographic appearances which may be aids to diagnosis: (1) lengthening of the pyloric canal (normal does not exceed 1 cm.); (2) a narrowing of the pyloric canal, though this is variable; (3) a narrow crevice or a longer depression about the mid-point of the lower edge of the canal; and (4) concavity in the base of the duodenal bulb—that is, a duodenal bulbar impression. They considered that the only definite pathognomonic feature was the last of these. While it is of interest, and occasionally of importance, to make a preoperative diagnosis, we would emphasize that all filling defects in the region of the pyloric canal in adults should be regarded as neoplastic until proved at operation to be benign.

It has been stated (Morley, 1949) that it is difficult to distinguish this condition from a carcinoma of the pylorus, but we have found that, when it has been met with once and diagnosed at operation, recognition of the innocent nature of the tumour is relatively easy. If in doubt, a small incision with diathermy into the tumour will render the diagnosis more obvious, as the glistening, bulging cut surface of the muscle is quite characteristic and in no way resembles the cut surface of an infiltrative type of neoplasm.

Having identified the lesion, we have dealt with it along the following lines: (1) If a peptic ulcer is present a gastrectomy is performed. The presence of hypertrophy is no contraindication to the Billroth-I type of reconstruction, as the duodenum is never involved in the hypertrophy. (2) If there is no obvious ulcer on examination of the serosal surfaces of stomach and duodenum and after careful palpation, we consider the history, and where there is a good clinical history suggestive of peptic ulcer we perform a Billroth-I gastrectomy, since on occasions ulcers have been found only after examination of the resected specimen. (3) If the clinical history is atypical and the diagnosis has been mainly based on radiological findings, we advise a pyloro-duodenotomy, biopsy, and resuture transversely to make a Heineke–Mikulicz pyloroplasty. This is most likely to prove satisfactory where there is coexistent mucosal stenosis (Tanner, 1950).

It is very inviting where no other lesion is found to perform a Ramstedt operation, and this was done in Case 16. The result so far has been most gratifying, but as the patient had no gastric symptoms before laparotomy for intestinal obstruction it is hardly a fair test.

In conclusion we would like to add that since we started to make a particular study of this condition in 1953 we have identified it with greater frequency than we had done before, and are sure that it is a far more common condition than is generally realized.

Summary

In the past ten years 25 cases of pyloric hypertrophy in adults have been seen during laparotomy at St. James's Hospital. In no case was a confident diagnosis of the condition made before operation, though in three cases the radiologist had suggested this possibility. All but two cases presented with symptoms suggesting peptic ulceration, and a peptic ulcer was in fact found in 18.

The radiological findings are reviewed and the aetiology is discussed. Where an ulcer is found or there is a history suggesting peptic ulcer a Billroth-I partial gastrectomy is advocated as the best form of treatment. In other cases, after exclusion of gastric carcinoma by biopsy, a lesser operation may suffice.

We acknowledge with thanks the kind co-operation of Mr. Norman Tanner in allowing us to use his cases. Our thanks are also due to Dr. R. V. Quilliam for his helpful suggestion with the radiographs.

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LOCAL ANALGESIA AND KIELLAND'S FORCEPS

BY

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[WITH SPECIAL PLATE]

The dangers of general anaesthesia in operative obstetrics have been repeatedly emphasized in recent years (Jeffcoate, 1953; Parker, 1954, 1956). Forceps delivery is by far the commonest operation performed, and is probably the most dangerous from an anaesthetic point of view. To avoid this risk some have advocated a return to operating with the patient in the lateral position, while others have advised the use of local analgesia wherever possible. Up to the present it has, however, been the general experience and opinion (Parker, 1956) that pudendal block and local infiltration of the perineum is applicable only to about 60% of all forceps deliveries—that is, those in which the head is at the outlet and favourably rotated.

This paper describes the methods and presents the results of an attempt to extend beyond this limit the use

M. G. P. STOKER: LATENT INFECTIONS WITH VIRUSES AND RICKETTSIAE

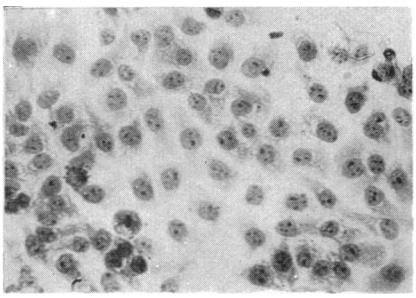


FIG. 1.—Culture of uninfected human carcinoma cells (strain HeLa). All preparations ×320, stained haematoxylin.

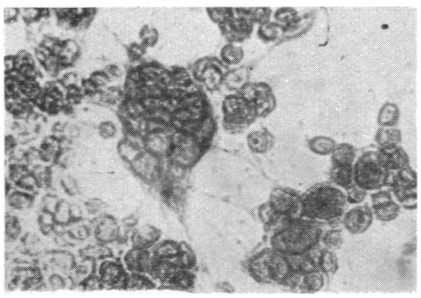


FIG. 2.—Cytopathic change five days after infection with herpes virus. Note giant cell and cytoplasmic processes between cells.

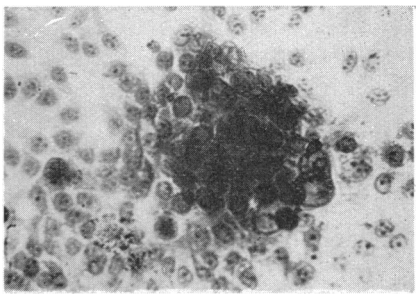


FIG. 3.—Cytopathic change five days after infection with herpes virus; herpes antibody added to medium two hours after virus. Focal distribution of affected cells surrounded by apparently normal cells.

A. M. DESMOND AND B. F. SWYNNERTON: ADULT HYPERTROPHY OF PYLORUS

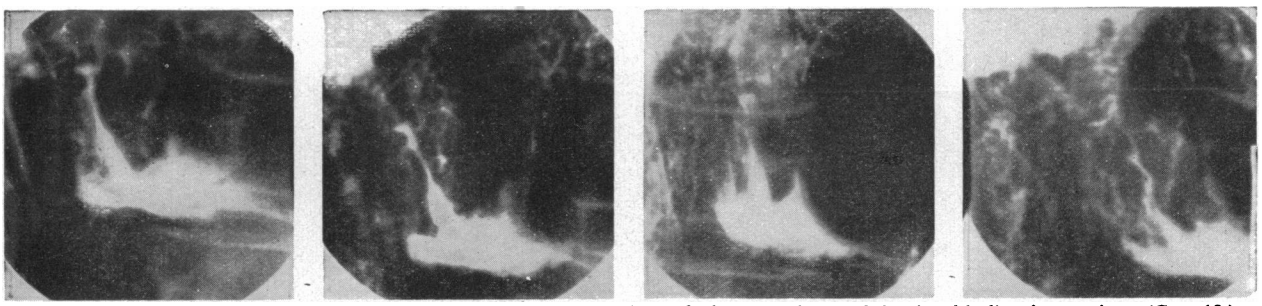


FIG. 1.—Elongation and narrowing of pyloric canal with surrounding soft-tissue shadow and duodenal bulbar impression. (Case 18.)

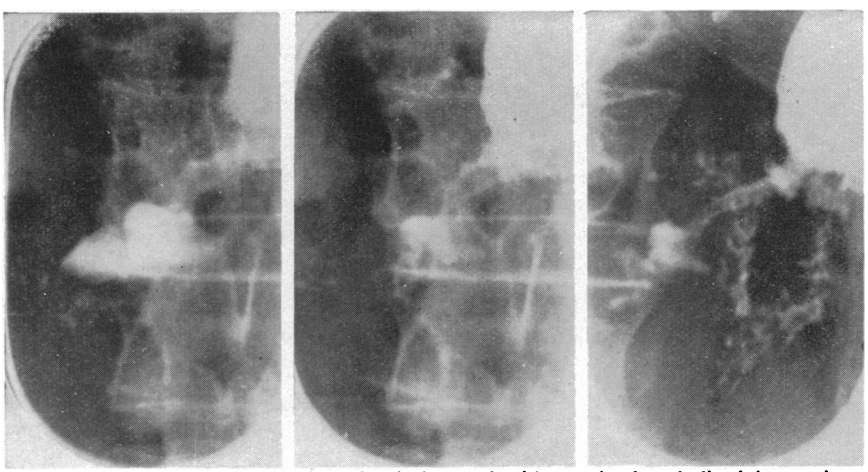
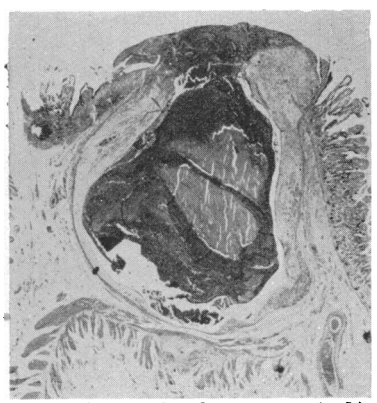


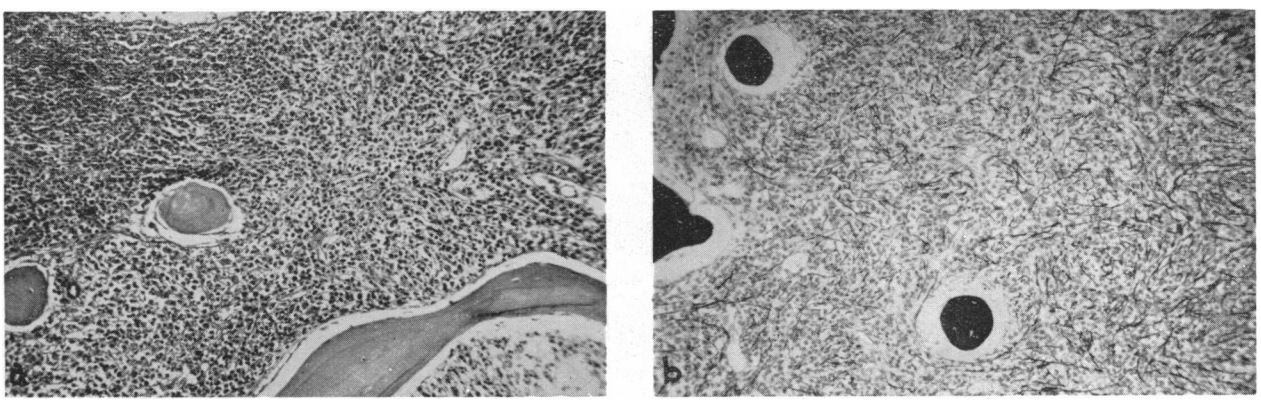
FIG. 2.—Elongation and narrowing of pyloric canal with proximal and distal impressions. (Case 14.)

A. F. ROBINSON *ET AL.*: HAMARTOMA OF BOWEL



Photomicrograph of tumour. (×5.)

R. B. THOMPSON AND S. G. M. MACKAY: ALEUKAEMIC MYELOBLASTIC LEUKAEMIA



Sections of ribs to demonstrate (*a*) fibroblastic proliferation; (*b*) reticulin fibrils. (×225.)