Conclusion

I imagine it may have been the lot of more than one Harveian Orator, as he comes to the end of his discourse, to fear that he has disappointed expectation and fallen short in the performance of an honourable duty. I have taken the name of Thomas Willis for commemoration as one of the Benefactors of the College. He gave well of the fruits of his labours preserved in those many pages which even Anthony Wood (1813-20) praised for the "natural smoothness, pure elegancy, [and] delightful, unaffected neatness" of their Latin style. I have occupied some part of your time with speculation about the cause of symptoms resulting from occlusion of the internal carotid artery. If there be anything in this of the least value let me exhort you, as I have the right, to search and study out these secrets of nature by way of experiment. Willis would have approved this exhortation and perhaps have thought us laggards. He foresaw great works. The ancients, he said, bound to false suppositions, might be excused the faulty structure of their reasoning, but "those who come after should take care for the Re-edifying of the Building even from the ground, on which our most famous Harvey hath laid the Circulation of the Blood as a new Foundation in Medicine."

REFERENCES

- Anonymous (1830). *Lives of British Physicians*, p. 58. London.
 Browne, T. (1646). *Pseudodoxia Epidemica*, preface. London.
 Foster, M. (1901). *Lectures on the History of Physiology*, p. 181. Cambridge.
 Franklin, K. J. (1931). *Proc. roy. Soc. Med.*, 25, Sect. Hist. Med., 8.
 Fulton, J. F. (1935). *Bibliography of Two Oxford Physiologists*, p. 13. Oxford.
 McDonald, D. A., and Potter, J. M. (1951). *J. Physiol. (Lond.)*, 114, 356.
 Nicolson, M. H. (1930). *The Correspondence of Anne, Viscountess Conway*. London.
 Schurr, P. Personal communication.
 Stubbe, H. (1670). *Legends no Histories; or a Specimen of Some Animadversions upon the History of the Royal Society*, Pt. 2. London.
 Willis, R. (1664). *Cerebri Anatome: cui Accessit Nervorum Descriptio et Usum*. London.
 — (1684a). *Anatomy of the Brain*, p. 68. Published in his *Practice of Physick*. London.
 — (1684b). *Introduction to Pharmaceutice Rationalis*, Pt. 2. Published in his *Practice of Physick*. London.
 Wood, A. (1813-20). *Athenae Oxonienses*, 4 vols. London.
 — (1891-1900). *Life and Times of Anthony Wood*, 5 vols. Oxford.

PANCREATITIS FOLLOWING PREGNANCY

BY

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The relation between pregnancy and pancreatitis is little discussed in the literature, although many reports of pregnancy complicated by pancreatitis have been published (Kingsley, 1934; Cassel and Malewitz, 1950; Burger, 1952) since the cases of Schmitt (1818) and of Lawrence (1831) early last century. Langmade and Edmondson (1951) collected 53 cases from the literature and reported nine more of their own.

The earliest report of pancreatitis occurring in the post-partum period seems to be that of Haidlen (1884). Watts (1918) and Fallis and Plain (1939) have each reported two cases, and others have been described or mentioned by Deaver (1918), Paxton and Payne (1948), Roberts *et al.* (1950), Rose (1951), and Comfort and Steinberg (1952). McWhorter (1932) reported 64 cases of pancreatitis in two of which "the onset of pancreatitis occurred shortly after childbirth." Langmade and Edmondson (1951) noted that, in pancreatitis compli-

cating pregnancy, liability to an exacerbation during the puerperium was an outstanding characteristic.

Nevertheless, most recent writers do not stress pregnancy as an aetiological factor in pancreatitis (Paxton and Payne, 1948; Warren, 1948; Macnab, 1949; Fallis, 1951; Saint and Weiden, 1953). There is, however, increasing evidence to support the idea of Whipple (1907) that "acute" and "chronic" pancreatitis are differing phases of the same disease process (Comfort *et al.*, 1946), while Saint has rightly emphasized that "pancreatitis" is a general term covering several diseases which have in common only the feature of localization to the pancreas (Saint and Weiden, 1953).

This paper reports six patients with pancreatitis, in all of whom the disease was first manifest a few weeks or months after completion of pregnancy, in whom the clinical and laboratory findings were similar, and none of whom presented any evidence of the known causes of pancreatitis. It is suggested that these patients may be representative of one type of pancreatitis which forms a distinctive syndrome, to be separated from the large group of patients with "pancreatitis of unknown aetiology." For convenience, some of the features of these cases are given in Table I.

Case 1

A woman aged 20 was admitted on May 29, 1954. One year before admission she had been delivered of a stillborn child. Six weeks after this she developed flatulent dyspepsia and intermittent epigastric distress, later followed by severe constant epigastric pain radiating through to the back on the left side. She vomited occasionally. The pain was worse after fatty foods. Two weeks before admission she had more severe pain than previously, and this was followed by persistent vomiting and then jaundice with dark urine and pale stools.

On examination she was jaundiced. Her temperature was 99.8° F. (37.7° C.), pulse 80, blood pressure 120/90 mm. Hg. There was rigidity and guarding over her abdomen, but she was more tender in the epigastrium. The liver and gall-bladder were impalpable. The diagnosis of obstructive jaundice due to pancreatitis was made. The urinary diastase level was 400 units.

Laparotomy was performed on June 1. The gall-bladder was normal and contained no calculi, while the pancreas was irregularly enlarged and thickened. The common bile duct was drained and a pancreatic biopsy was performed; this showed pancreatitis (Fig. 1).

FIG. 1.—Pancreatic specimen taken for biopsy at operation in Case 1. At this stage the patient was still jaundiced and the urinary diastase elevated. The specimen shows increased fibrous tissue and cellular infiltration without necrosis of the parenchymal cells; the pancreatic ducts are slightly dilated. (Van Gieson. $\times 144$.)

*Working with the aid of a grant from the National Health and Medical Research Council of Australia.

TABLE 1.—*Some Clinical Features of Six Patients with Post-partum Pancreatitis*

Case No.	Family History	Parity	Pregnancy	Child	Time of Onset of Symptoms after Pregnancy	Time of Onset of Acute Pancreatitis	Abdominal Pain	Vomiting	Jaundice	Gall-stones	Result
1	Aunt had cholelithiasis	1	Normal	Stillborn	6 weeks	12 months	+	+	+	0	Well
2	Nil	1	"	Living	3 months	3 "	+	+	+	+	Recurrent pain two years later. Again pregnant Well.
3	"	1	Hypertension induction of labour	"	2 weeks	6 weeks	+	+	+	+	
4	"	1	Normal	"	9 months	11 months	+	+	0	+	
5	"	1	Some flatulence and vomiting	"	A few days post partum	2 years	+	+	Slight	+	Well
6	Father had cholelithiasis	2	Normal	"	11 weeks	14 weeks	+	+	0	?	?

Her convalescence was uneventful. A cholangiogram through the tube in the common bile duct on June 28 showed the anatomy of the biliary tree to be normal, and the contrast medium entered the duodenum freely (Fig. 4). The tube was removed on June 30. She has remained well since.

Case 2

This patient, aged 23, was first admitted on February 3, 1954. Three months before admission she had a normal delivery after a normal pregnancy. Two days before admission she had a sudden onset of severe colicky pain in the epigastrium radiating through to the back and associated with nausea and persistent vomiting. This was followed next day by jaundice with dark urine and pale faeces.

On examination she was icteric. Her temperature was 99° F. (37.2° C.), pulse 100, blood pressure 125/80 mm. Hg. There was tenderness in the epigastrium. The diagnosis of pancreatitis and cholelithiasis was made, although no urinary diastase estimation was done at this stage. She responded to conservative treatment with pethidine and antibiotics.

A cholecystogram on March 1 showed multiple non-opaque calculi in the gall-bladder. At laparotomy on March 5 the pancreas was enlarged, firm, and nodular. The common bile duct was normal. The gall-bladder was not thickened, but it contained small stones. Cholecystectomy was performed uneventfully. The patient has since remained well.

Case 3

A young woman, aged 19, was first admitted on December 31, 1951. Eight weeks previously she had been successfully delivered of her first child after a pregnancy complicated by hypertension and terminated by medical induction of labour. Two weeks later she had developed sudden severe pain in the back, followed by two milder attacks. Four days before admission she had a fourth attack, more severe than the earlier ones, felt both in the back and in the epigastrium and associated with vomiting. Her urine was dark and her faeces were pale.

On examination she was icteric; temperature 99° F. (37.2° C.), pulse 96, blood pressure 140/80 mm. Hg. She was thought to have cholelithiasis, and a cholecystogram on January 14, 1952, showed multiple radiolucent calculi. At operation on January 17 the gall-bladder was thickened and contained calculi. Cholecystectomy was performed. The pancreas was not examined. Convalescence was delayed by a wound infection producing erythema nodosum and necessitating secondary suture. She was discharged on March 1.

When seen again on January 28, 1954, she complained of flatulent dyspepsia and recurrent severe abdominal pain radiating through to the back and with occasional vomiting. The attacks had occurred every two or three weeks for some months. She had noted no jaundice. The clinical diagnosis of relapsing pancreatitis was confirmed by a urinary diastase of 160 units on January 28. She has been treated with a low-fat diet and atropine and ephedrine orally, with decrease in the frequency and severity of her pain.

Case 4

A woman aged 22 was admitted on March 22, 1954. Two months before admission, and nine months after a normal pregnancy, she developed flatulent dyspepsia and recurrent epigastric pain radiating through to the back, associated with vomiting and a dark coloration of her urine. Three days before admission she had severe persistent pain in the left upper quadrant of her abdomen radiating to the back, and frequent vomiting.

On examination she was not jaundiced. Her temperature was 101° F. (38.3° C.), pulse 100, blood pressure 140/90 mm. Hg. There was tenderness and rigidity in the upper abdomen, more marked on the left side. A clinical diagnosis of pancreatitis was confirmed by a urinary diastase estimation of over 2,000 units. She responded to conservative treatment with gastric aspiration, intravenous fluid replacement, pethidine, and antibiotics.

Oral cholecystography on April 7 failed to outline the gall-bladder, but an intravenous cholelithogram on May 20 showed a large gall-bladder containing multiple stones up to 1 cm. in diameter.

Laparotomy was performed on June 4. An operative cholangiogram showed the dye medium to enter the duodenum freely. There was no evidence of residual pancreatitis. The common bile duct was normal. The gall-bladder contained multiple stones and was removed, and a pancreatic biopsy was done (see below). The patient has remained well since operation, although she is again pregnant.

Case 5

A woman aged 26 was first admitted on March 22, 1949. Two years previously, a few days after childbirth, she developed epigastric pain and flatulence lasting a few days and recurring thereafter at intervals of weeks. Six days before admission she had sudden severe epigastric pain radiating through to the back and associated with nausea and vomiting.

On examination she was acutely ill and slightly icteric. Her temperature was 100.6° F. (38.1° C.), pulse 120, blood pressure 150/100 mm. Hg. Epigastric tenderness and rigidity were present. The diagnosis of acute pancreatitis was confirmed by a urinary diastase level of over 1,000 units. She was treated conservatively with morphine and antibiotics, and gradually improved over the following week.

A cholecystogram on April 8 showed multiple small calculi in the gall-bladder, and laparotomy was performed on May 4. There was fat necrosis in the omentum and mesocolon; the pancreas was thickened and irregular; the gall-bladder was thickened and contained calculi, but the common bile duct was normal. Cholecystectomy was performed uneventfully.

When last seen twelve months after operation she was well and free of pain and dyspepsia.

Case 6

A woman aged 22 was admitted on March 17, 1954. Fourteen weeks previously she had completed a normal pregnancy. Three weeks before admission she had a mild attack of epigastric pain lasting two days. Two days before admission she had sudden onset of severe constant epigastric pain radiating to both flanks, with nausea and vomiting.

On examination she appeared flushed and drawn, but was not jaundiced. Her temperature was 100.6° F. (38.1° C.), pulse 136, blood pressure 130/90 mm. Hg. Her abdomen was slightly distended, with generalized tenderness, and there was guarding in the epigastrium.

A urinary diastase estimation of 2,000 units confirmed the clinical diagnosis of pancreatitis. Conservative treatment with intravenous therapy, pethidine, and antibiotics resulted in rapid improvement in her condition, and she was discharged on March 31. No follow-up studies have yet been possible on this patient.

Laboratory Investigations

The laboratory investigations in all patients followed a similar pattern, and may conveniently be considered together (Tables II and III).

TABLE II.—Salient Laboratory Findings in Six Cases of Post-partum Pancreatitis. In Cases 2 and 3 the Urinary Diastase Estimations were Made After recovery from the Acute Stage of the Disease

Case No.	Urine				Blood		Total Serum Lipides (mg./100 ml.)	Serum Cholesterol (mg./100 ml.)
	Proteinuria	Glucosuria	Bile	Diastase	Hb (g.%)	Leucocytes (per c.mm.)		
1	+	+	+	400	13.3	6,000	480	160
2	0	0	+	30	12.1	7,000	520	210
3	0	0	+	160	11.3	11,200	500	195
4	0	+	+	2,000	9.9	16,000	500	—
5	+	0	+	1,000	14.0	28,800	—	—
6	0	0	+	2,000	7.8	15,000	—	—

TABLE III.—Liver-function Tests in Six Cases of Post-partum Pancreatitis. The Significant Abnormalities are slight Elevation of the Serum Bilirubin and Alkaline Phosphatase in Cases 1, 3, 4, and 5

Case No.	Serum Bilirubin (mg./100 ml.)	Cephalin Flocculation	Serum Albumin (g.%)	Serum Globulin (g.%)	Serum Gamma Globulin (g.%)	Serum Alkaline Phosphatase (K-A Units)
1	4.0	Weak positive	4.7	2.0	0.8	23
2	0.2	Negative	4.5	2.3	0.9	7
3	0.2	Negative	5.6	2.1	1.3	29
4	2.5	Weak positive	4.3	1.9	0.9	12
5	6.0	Negative	4.8	2.3	—	25
6	0.5	Weak positive	3.7	2.0	—	—

Urine Examination.—Two patients (Cases 1 and 5) showed transient proteinuria, two (Cases 1 and 4) transient glucosuria, and all cases had bile in the urine during the acute stage of the disease (Table II). In all cases examination of the urine was normal on discharge from hospital.

Urinary Diastase.—In four patients estimations of the urinary diastase level on admission to hospital with severe symptoms revealed diagnostic levels confirming the clinical diagnosis of pancreatitis (Table II). In Case 3 a less marked elevation to 160 units was recorded when the patient presented with post-cholecystectomy dyspepsia three years after the attack of "acute cholecystitis." This reading is regarded as diagnostic in this laboratory. In Case 2 no significant elevation of the urinary diastase was found, but it was not possible to examine the urine during an exacerbation of symptoms. In this patient the diagnosis of pancreatitis was confirmed at laparotomy. Serum amylase levels were not investigated.

Blood Examination.—The results of blood examinations were variable. Two patients had a moderate hypochromic anaemia. Acute exacerbations were accompanied by a polymorphonuclear leucocytosis, which in Case 5 reached 28,800 per c.mm. (Table II).

Liver-function Tests.—The results of liver-function tests are detailed in Table III. They show slight elevation of the serum bilirubin and alkaline phosphatase, without change in plasma protein levels. When symptoms were severe a weakly positive cephalin flocculation was sometimes noted. The findings suggest that jaundice in these patients is obstructive in type, although slight hepatocellular damage may accompany peritoneal irritation in exacerbations of the disease. None of these patients was subjected to liver biopsy.

Gastric Investigations.—Because of the frequency with which the patients complained of flatulent dyspepsia, a histamine test-meal examination was made in five cases (Cases 1, 2, 3, 4, and 5), and in all it revealed a normal gastric acidity. Gastric biopsy in Cases 1 and 4 by the method of Wood *et al.* (1949) showed normal mucosa in both.

Glucose-tolerance Tests.—Oral glucose-tolerance tests after 50 g. of glucose were performed on three patients (Cases 1, 2, and 4), and none had hyperglycaemia or a diabetic type of curve.

Serum Lipide and Cholesterol Levels.—There was no elevation of the total serum lipides measured by the turbidimetric method of Kunkel *et al.* (1948) or of the serum cholesterol in those cases in which they were measured (Table II). In Cases 2, 3, and 4 fat-tolerance curves after ingestion of 50 g. of butter were performed after recovery from the acute stage. In all three cases these suggested poor absorption, consistent with impaired pancreatic function (Fig. 2).

Other Investigations.—The intravenous secretin test was not used in this study, as in our hands it has produced results too variable to be of great diagnostic value. Similar variations have been reported from other centres (Dornberger *et al.*, 1948; Hefferon and Gunter, 1951). In all patients the Wassermann reaction was negative. The erythrocyte sedimentation rate was raised during exacerbations of symptoms.

FIG. 2.—Fat-tolerance curves in Cases 2, 3, and 4. In all there is a low fasting lipid level and a rise of less than 100 mg. per 100 ml. after 50 g. of butter orally.

Radiological Studies

Plain x-ray films of the abdomen and chest were normal in all cases.

Oral cholecystography was performed in Cases 2 to 5 after recovery from the initial "acute pancreatitis," an interval of about three weeks. In Cases 2, 3, and 5 this revealed multiple small non-opaque calculi in the gall-bladder. However, in Case 4 there was no radiological demonstration of the gall-bladder, and an intravenous choledochogram was done six weeks later, eight weeks after initial admission to hospital. This showed the gall-bladder to be enlarged and to contain multiple calculi; the bile ducts were not outlined.

A cholangiogram was performed at operation in Case 4 (Hughes and Kernutt, 1954b). There was good filling of the biliary tree. The contrast medium flowed freely into the duodenum, and there was no demonstrable abnormality at the lower end of the common bile duct. In one film the medium had entered the main pancreatic duct by retrograde flow, and no obstruction to this was noted (Fig. 3).

In Case 1 a cholangiogram was performed four weeks after operation before removal of the T-tube from the common bile duct. The medium entered the duodenum freely and the common bile duct was normal, with the usual anatomical tapering at the lower end (Hughes and Kernutt, 1954a). No evidence of cholelithiasis was obtained (Fig. 4).

Pancreatic Biopsies

Biopsy of a specimen from the body of the pancreas was obtained at operation in Cases 1 and 4. There were no adverse sequelae from these procedures.

In Case 1 the biopsy was carried out a year after completion of pregnancy and three weeks after the onset of jaundice, at a time when the urinary diastase was still slightly raised. This biopsy showed slight increase in fibrous tissue, distorting the normal pancreatic architecture. There was some infiltration by fibroblasts, lymphocytes, and polymorphs. The acini were slightly irregular but without areas of necrosis of the parenchymal cells (Fig. 1).

The biopsy in Case 4 was carried out twelve months after the birth of her child and five months after she first developed dyspepsia. At the time of operation she had been free of symptoms for two months. The biopsy showed normal pancreatic tissue, without evidence of the preceding inflammation and with no residual fibrosis or fatty infiltration.

Discussion

These patients, and those reported by other authors, have many features in common. All were young women who developed flatulence and recurrent bouts of abdominal pain and vomiting within a few days or weeks of pregnancy, culminating in an attack of "acute pancreatitis." In all of them the clinical and laboratory findings were closely similar.

The aetiology of the pancreatitis is of the greatest interest. None of the six patients presented clinical or epidemiological evidence of infection with mumps or the other viruses known occasionally to cause pancreatitis. All had excellent dietetic histories, with no suggestion of alcoholism or malnutrition. In only two of the six was there a family history of cholelithiasis; none had a family history of cardiovascular disease, xanthomatosis, diabetes mellitus, or pan-

FIG. 3.—Operative cholangiogram in pancreatitis following pregnancy (Case 4). There is no biliary obstruction and the anatomy of the bile ducts appears normal. There has been some filling of the main pancreatic duct.

FIG. 4.—Post-operative cholangiogram in post-partum pancreatitis (Case 1). The arrangement and calibre of the bile ducts are normal, and the contrast medium enters the duodenum freely. There has been some retrograde filling of the main pancreatic duct.

creatitis, and in four cases estimations of serum lipide and cholesterol levels revealed normal values (Table II; Fig. 2). One patient (Case 2) has a binovular twin who has borne two children without evidence of pancreatic disease. These facts render unlikely the hereditary type of pancreatitis recently described and associated frequently with hyperlipaemia (Holt *et al.*, 1939; Collett and Kennedy, 1948; Comfort and Steinberg, 1952; Klatskin and Gordon, 1952). It should nevertheless be noted that one patient (No. II 3) in the family reported by Comfort and Steinberg (1952) first developed symptoms a month after the birth of her first child.

The relation between the pancreatic lesions and cholelithiasis must also be considered. Cholelithiasis was present in four of the five cases in which full radiological studies and laparotomy were performed, but in none were stones found in the common bile duct, and in none was any abnormality of the sphincter of Oddi demonstrated. In Cases 1 and 4 cholangiography showed the duct of Wirsung to be freely patent (Figs. 2 and 3). Langmade and Edmondson (1951) in their survey of 53 reported cases of pancreatitis complicating pregnancy found 34 in which the gall-bladder was examined at operation or after death, but calculi were noted in only 12 of these. They stated that "there was no particular difference between those with and those without [gall-stones] as far as the onset and course of their pancreatitis was concerned." There is also statistical evidence to suggest that there is little direct relation between pregnancy and cholelithiasis (Gross, 1929; Joske *et al.*, 1954). For these reasons it is thought improbable that cholelithiasis in these patients has any aetiological significance, but rather that biliary stasis due to pancreatitis may be responsible for the development of cholelithiasis, or that both cholelithiasis and pancreatitis are sequelae of the lipaemia of pregnancy.

Reflux filling of the main pancreatic duct was shown radiologically in two patients (Cases 1 and 4). The work

of Hicken and McAllister (1952) suggests strongly that reflux into the main pancreatic duct is a frequent and normal finding in man, and of no pathological significance. They found also that this reflux may occur whether or not there is a common entry of the common bile and pancreatic ducts into the duodenum. Experience with cholangiography in this hospital agrees with this (Hughes and Kernutt, 1954b), and the relation of biliary reflux to pancreatitis in man is thought to be slight.

It is therefore postulated that these patients were in fact suffering from a type of chronic relapsing pancreatitis which is in some way related to pregnancy, and that this may represent an entity within the large group of diseases considered together as "pancreatitis."

The abnormality in pregnancy causing pancreatitis remains unknown. Liver-function tests and biopsies on pregnant women show no suggestive pattern in this regard (Ingerslev and Teilum, 1945; Day and Hellestrand, 1947; Dieckmann *et al.*, 1951). Boyd (1934) has shown that neutral fat, phospholipids, and cholesterol are all increased in the plasma during pregnancy, while Potter (1936) states that the gall-bladder is distended and that there is hypercholesterolaemia at term in most women.

The management of this disorder is poorly understood. Treatment has been conservative, surgery being indicated only in the presence of cholelithiasis, for the relief of obstructive jaundice, and for drainage of residual abscesses after an acute exacerbation. Internal biliary drainage by sphincterotomy does not seem to be indicated, because of the apparently normal state of the ducts and sphincters found in these patients, and the recent anatomical studies of Hughes and Kernutt (1954a) and of Sterling (1954).

Where symptoms of flatulence and pain have persisted the patient has been placed on a high-protein, low-fat diet and given atropine and ephedrine by mouth. It is not yet possible to speak of the efficacy of this regime or of the possible effects of further pregnancies in these patients. The recurrence of symptoms in Case 3 after cholecystectomy suggests that this syndrome may be one cause of the "post-cholecystectomy syndrome," although the normal biopsy finding in Case 4 two months after cessation of symptoms indicates that resolution may be complete.

Summary

Six cases are reported of young women who developed pancreatitis shortly after pregnancy. In all of them the clinical and laboratory findings were similar.

The aetiology of the pancreatitis is considered to be related to the preceding pregnancy, although the mechanism of this is unknown.

It is suggested that these patients, and others in the literature, form a distinct subgroup within the general group of patients with pancreatitis.

Until the aetiological factors are better understood, it is suggested that the term "post-partum pancreatitis" be applied to this subgroup.

My thanks are due to Dr. Ian Wood, head of the Clinical Research Unit, and Mr. E. S. R. Hughes for their interest and help in this work; to my colleagues in the clinical research unit for the many investigations performed on these patients; and to the physicians and surgeons who referred these cases. The photographs were prepared by Dr. J. D. Hicks and Mr. Roy Ingles, of the Royal Melbourne Hospital.

REFERENCES

- Boyd, E. M. (1934). *J. clin. Invest.*, **13**, 347.
 Burger, P. (1952). *Rev. franç. Gynec.*, **47**, 379. Abstract in *Int. Abstr. Surg.*, **97**, 372.
 Cassel, W. J., jun., and Malewitz, E. C. (1950). *J. Amer. med. Ass.*, **142**, 1139.
 Collett, R. W., and Kennedy, R. L. J. (1948). *Proc. Mayo Clin.*, **23**, 158.
 Comfort, M. W., Gambill, E. E., and Baggenstoss, A. H. (1946). *Gastroenterology*, **6**, 239, 376.
 — and Steinberg, A. G. (1952). *Ibid.*, **21**, 54.
 Day, E. M., and Hellestrand, A. L. (1947). *Med. J. Aust.*, **2**, 326.
 Deaver, J. B. (1918). *Ann. Surg.*, **68**, 281.
 Dieckmann, W. J., Smitt, R. C., and Pottinger, R. E. (1951). *Surg. Gynec. Obstet.*, **92**, 598.
 Dornberger, G. R., Comfort, M. W., Wollaeger, E. E., and Power, M. H. (1948). *Gastroenterology*, **11**, 701.
 Fallis, L. S. (1951). *J. int. Coll. Surg.*, **16**, 337.
 — and Plain, G. (1939). *Surgery*, **5**, 358.
 Gross, D. M. B. (1929). *J. Path. Bact.*, **32**, 503.
 Haidlen, R. (1884). *Zbl. Gynäk.*, **8**, 609. Cited by Roberts *et al.* (1950).
 Heffernon, E. W., and Gunter, A. R. (1951). *Gastroenterology*, **19**, 526.
 Hicken, N. F., and McAllister, A. J. (1952). *Amer. J. Surg.*, **83**, 781.
 Holt, L. E., jun., Aylward, F. X., and Timbres, H. G. (1939). *Bull. Johns. Hopk. Hosp.*, **64**, 279.
 Hughes, E. S. R., and Kernutt, R. H. (1954a). *Aust. N.Z. J. Surg.*, **23**, 223.
 — (1954b). *British Medical Journal*, **2**, 620.
 Ingerslev, M., and Teilum, G. (1945). *Acta obstet. gynec. scand.*, **25**, 352. Cited by Dieckmann *et al.* (1951).
 Joske, R. A., Saint, E. G., Hughes, E. S. R., and Bromilow, F. J. (1954). *Med. J. Aust.*, **2**, 473.
 Kingsley, C. R. (1934). *N.Y. St. J. Med.*, **34**, 889.
 Klatskin, G., and Gordon, M. (1952). *Amer. J. Med.*, **12**, 3.
 Kunkel, H. G., Ahrens, E. H., and Eisenmenger, W. J. (1948). *Gastroenterology*, **11**, 499.
 Langmade, C., and Edmondson, H. A. (1951). *Surg. Gynec. Obstet.*, **92**, 43.
 Lawrence, W. (1831). *Med.-chir. Trans.*, **16**, 367. Cited by Langmade and Edmondson (1951).
 Macnab, I. (1949). *British Medical Journal*, **1**, 568.
 McWhorter, G. L. (1932). *Arch. Surg., Chicago*, **25**, 958.
 Paxton, J. R., and Payne, J. H. (1948). *Surg. Gynec. Obstet.*, **86**, 69.
 Potter, M. G. (1936). *J. Amer. med. Ass.*, **106**, 1070.
 Roberts, N. J., Baggenstoss, A. H., and Comfort, M. W. (1950). *Amer. J. clin. Path.*, **20**, 742.
 Rose, T. F. (1951). *Med. J. Aust.*, **2**, 453.
 Saint, E. G., and Weiden, S. (1953). *British Medical Journal*, **2**, 1335.
 Schmitt, W. J. (1818). *Sammlung zweifelhafter Schwangerschaftsfaelle nebst einer kritischen Einleitung*, p. 172. Wien. Cited by Langmade and Edmondson (1951).
 Sterling, J. A. (1954). *Surg. Gynec. Obstet.*, **98**, 420.
 Warren, K. W. (1948). *Surg. clin. N. Amer.*, **28**, 741.
 Watts, S. H. (1918). *Ann. Surg.*, **67**, 278.
 Whipple, G. H. (1907). *Bull. Johns Hopk. Hosp.*, **18**, 391.
 Wood, I. J., Doig, R. K., Motteram, R., and Hughes, A. (1949). *Lancet*, **1**, 18.

TUBERCULOUS MESENTERIC ADENITIS IN CHILDREN*

BY

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The purpose of this essay is to draw attention to a cause of minor illness in children which is usually overlooked in this country. It is intended to show that tuberculous mesenteric glands may sometimes cause symptoms and continue to do so even after they have become calcified.

The condition is well recognized on the Continent. Klein in 1927 reported cases from Poland and commented: "Beaucoup de médecins, en presence de cette maladie, ne croient pas à la possibilité de son existence." In Sweden, Strömbeck (1932) made a thorough review of the published work on the subject. Alexandre (1949), in France, gave another excellent account.

The symptoms described are colicky central abdominal pain, nausea, vomiting, sweating, meteorism, diarrhoea, constipation, pallor, lassitude, and loss of weight. The physical signs are palpable tumours in the abdomen, local tenderness, fever (especially in the evenings), and calcification visible in the x-ray film.

Most physicians, when confronted with calcified glands, assume that calcification signifies a healed lesion and that they must look elsewhere to account for the symptoms. If this assumption be true then we must assume also that the calcification appears suddenly and instantaneously, and that at that moment

*Condensed from the Nathaniel Bishop Harman Prize essay, 1954.