tance of acute haemorrhagic pancreatitis with two common acute abdominal conditions are noted.

The serum amylase and lipase levels were measured in 25 normal subjects and in 12 patients with past or present pancreatic disease. No significant serum enzyme response was obtained after pancreatic stimulation with morphine, methacholine, secretin, or pancreozymin in any of the individuals investigated, and the suggestion that the response obtained with these substances may be of value as an indication of the presence of pancreatitis was not confirmed.

The serum amylase and lipase levels were investigated in three cases of acute haemorrhagic pancreatitis and in certain other acute abdominal conditions; the highest readings were obtained in acute pancreatitis, but a two-fold or threefold increase was found to be fairly common in cases of perforated duodenal and juxtapyloric ulcers, and high levels were recorded in patients with calculi in the common bile duct.

The excretion of urinary diastase during the first twenty-four hours in hospital was measured in three cases of acute pancreatitis and in 41 other patients with acute abdominal disease: wide variations were recorded, and this investigation is not considered useful or reliable.

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PRIMARY MYXOEDEMA APPARENTLY DATING FROM POST-PARTUM SHOCK

BY

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Hypopituitarism is an important sequel of severe post-partum haemorrhage (Sheehan and Murdoch, 1938), and may lead to secondary hypothyroidism. Primary (idiopathic) myxoedema may also first become evident during the post-partum period. Frank myxoedema is seldom an early feature of hypopituitarism; yet when the onset of myxoedema appears to have been preceded by post-partum shock it is natural to suspect that the primary lesion may lie within the pituitary gland.

In practice it may be hard, on purely clinical grounds, to distinguish between primary and secondary myxoedema. We propose, therefore, to draw attention to four patients whose disabilities, though dating from a post-partum haemorrhage or shock, were nevertheless found to be due to primary and irreversible thyroid failure, and were fully corrected by thyroid hormone alone. In these patients the diagnosis was established

most clearly by a careful assessment of anterior pituitary function and by radio-iodine tests after thyrotrophin (T.S.H.) injections. The validity of this last test we will attempt to evaluate by analysing the results obtained from its use in other patients.

Case 1 (E.I. 168467)

The patient, aged 33, was admitted with obvious myxoedema in 1947. Symptoms of anaemia dated from a miscarriage in 1944 which was accompanied by a severe uterine haemorrhage requiring blood transfusion. Menstruation then returned, and in November, 1946, the patient had a normal delivery, followed by amenorrhoea and symptoms of myxoedema. Lactation had occurred, but was inadequate.

Examination (August, 1947) showed obvious myxoedema, hypotension (B.P. 90/60), and moderate anaemia (Hb, 12.6 g./100 ml.; R.B.C., 3,300,000; M.C.V., 100 cubic microns; M.C.H.C., 37%). Tests of endocrine function showed: B.M.R., -49% (Boothby and Sandiford standards); plasma cholesterol, 400 mg. per 100 ml.; E.C.G., low voltage and flat T waves; urinary 17-ketosteroids, 1.9 mg./24 hours; water-excretion tests, 56 and 70% excretion of water dose in three hours*; insulin-tolerance test (0.1 unit/kg.), hypoglycaemic unresponsiveness (blood sugar at two hours = 65% of fasting level); urinary excretion of follicle-stimulating hormone (F.S.H.), between 4 and 8 mouse units in 24 hours.

At first, because pituitary failure was suspected, she was given testosterone (600-mg. implant). This did not correct the anaemia or E.C.G. voltage, but caused a slight improvement in her general condition. Later, both of these abnormalities, and also the hypoglycaemic unresponsiveness, were fully corrected by thyroid extract alone (180 mg. a day). Menstruation returned in December, 1947, infrequent at first, but giving way to menorrhagia when the dose of thyroid extract was reduced early in 1953. The urinary F.S.H. excretion had risen to over 48 m.u./24 hours by December, 1951, and to over 96 m.u. the following year. After raising the dose of thyroid extract to 360 mg. a day, the urinary F.S.H. excretion fell to between 24 and 48 m.u.; the menorrhagia took longer to subside. Otherwise the patient has remained well on thyroid treatment alone. Results of the radio-iodine tests (November, 1951) are shown in the Table.

Case 2 (C.C. 127563)

The patient, aged 20, ruptured her uterus and suffered a post-partum haemorrhage, due to uterine inversion, at the end of her first pregnancy, in 1949. She was given a blood transfusion, and lactation was suppressed by oestrogens. Five months later menstruation returned with menorrhagia and other signs of myxoedema.

On examination (November, 1951) there was obvious myxoedema and hypotension (B.P. 90/65). The breasts, sexual hair, and uterine size were normal. Tests of endocrine function showed: B.M.R., -28% (Boothby and Sandiford standards); plasma cholesterol, 374 mg. per 100 ml.; E.C.G., low voltage and flat T waves; urinary 17-ketosteroids, 4.4 and 4.3 mg./24 hours; water-excretion test, only 10% of water dose excreted in three hours (normal exceeds 70%); insulin-tolerance test (0.1 unit/kg.), normal; eosinophil responses to corticotrophin and adrenaline (Thorn test), normal; urinary F.S.H. excretion exceeded 48 m.u./24 hours. Endometrial biopsy showed a hyperplastic proliferative endometrium. Results of the radio-iodine tests (November, 1951) are shown in the Table.

The patient has remained well on treatment with thyroid hormone alone (thyroxine, 0.3 mg. a day), and has since had a normal delivery. The E.C.G. has reverted to normal, and she now excretes 88% of a water dose in three hours. The menorrhagia has responded to thyroxine, and the urinary F.S.H. excretion has fallen to between 5 and 50 m.u./24 hours.

*Normal excretion of water dose (20 ml./kg.) exceeds 70%.

Radio-iodine Tests after Thyrotrophin Injections (T.S.H.)

Clinical Group		Case No.	Illness Duration (Years)		T.S.H. Injections Before 2nd Test		Oral Radio-iodine Tests After T.S.H. (Pre-T.S.H. Results in Brackets)			T.S.H. Response	
			Hypopituitarism (or Since P.P.H.)	Myxoedema	Preparation* and Daily Dose	No. of Days	48-hr. Neck Total (% Ad. Dose)	Urinary Data			
								0-48 Hr. Excretion (% Ad. Dose)	"T" Index†		
Controls		5 H.W. 6 E.C. 7 P.A.	— — —	— — —	A 4 ml. A 4 ml. T 10 U.S.P.	5 5 1	(Normal = 30-50%) 73 (53) 73 (—)	N. = 37-69% 3.6 (42) 15 (30) 31 (42)	N. = 2.8-13) 28 (4.3) 77 (8) 18 (10)	Yes " "	
Thyroid addict (10 years)		8 M.C.			A 4 ml.	5	23 (1.6)	65 (84)	3.5 (1.7)	"	
Hypopituitarism	Without obvious myxoedema	9 M.B. (a)	7	—	A 2 ml.	8	100 (?)	20 (34)	18 (13)	} ? Yes " "	
		10 M.C. (b)	10	—	T 10 U.S.P.	1	35 (—)	48 (49)	3.1 (2.3)		
		10 M.C.	4	—	A 4 ml.	5	34 (—)	30 (52)	9.1 (1.9)		
		11 E.K.	20	—	A 4 "	5	67 (—)	32 (36)	14 (3)		
	With clinically obvious myxoedema	12 V.H.	3	1-2	A 4 ml.	5	26 (—)	46 (77)	2 (1.4)	} ? Yes ? No "	
		13 A.T.	3	1	A 4 "	5	74 (8)	31 (87)	24 (1.6)		
		14 N.C.	15	5	A 4 "	5	28 (10)	57 (81)	2.6 (0.8)		
		15 I.F.	26	7	A 2 "	8	0.4 (—)	89 (71)	1.1 (1.0)		
		16 J.B. (a)	14	2-3	A 4 "	6	10.5 (5)	70 (73)	1.6 (1.4)		
		17 J.B. (b)	15	3-4	T 10 U.S.P.	1	—	72	1.4		
		17 L.C.	9	6	T 10 U.S.P.	1	0.4 (—)	89 (79)	1.1 (1.8)		
Post-partum myxoedema (present cases)		1 E.I. 2 C.C. 3 L.M. 4 L.V.	(7) (2) (3) (1)	5 1½ 2½ ½	A 2 ml. A 4 " { A 3 ml. (3) + T 10 U.S.P. (2) A 4 ml.	7 9 5 5	< 2 (—) 9 (—) < 5 (—) 2 (—)	92 (94) 69 (72) 77 (97) 79 (81)	1.4 (1.1) 2 (1.2) 4.2 (2.4) 3.6 (1.4)	" " " "	
Primary myxoedema (clinically obvious)		18 E.K. 19 M.H. 20 A.M. 21 F.O. 22 I.T. 23 M.G. 24 E.C. 25 H.E. 26 J.T. 27 H. 28 T.D. 29 V.R.	— — — — — — — — — — — —	2-3 1 2-3 20 4 15 (cretin) 10 4-5 10 2 15 (cretin) 18	A 4 ml. A 4 " A 4 " { A 4 ml. (3) + T 10 U.S.P. (2) " " " A 8 ml. T 10 U.S.P. T 10 U.S.P. T 10 U.S.P. T 10 U.S.P.	5 5 5 5 5 5 5 4 1 1 1 1	7.4 (—) — 1.2 (1.0) 1.4 (0.5) 4.0 — — 3 (1) 2 (—) 3 (3) 1 (0) 1 (—)	74 (75) 83 (68) 81 (77) 84 (84) 79 91 (97) 84 (87) 70 (62) 61 (62) 73 (82) 78 (97) 84 (89)	1.7 (3.0) 0.8 (2.1) 1.1 (1.3) 1.4 (2.0) 2.1 2.1 (2.0) 1.4 (1.2) 1.2 (0.8) 1.5 (0.3) 1.1 (1.7) 1.4 (2.7) 1.1 (1.7)	" " " " " " " " " " " "	

* T.S.H. preparations: A = "Ambinon B"; T = "Thyropar."

Many of the patients received thyroid extract throughout the tests: 10 gr. (0.65 g.) / day (8), 3 gr. (0.2 g.) / day (21, 22), 1½-2 gr. (0.1-0.13 g.) / day (3, 19, 23).

† "T" index = index of extrarenal disposal rate or of thyroid uptake derived from three urinary samples (Fraser *et al.*, 1953).

Case 3 (L.M. H 8208 G)

This patient became severely myxoedematous in 1952, at the age of 25. Three months earlier her first pregnancy had ended with the delivery, by caesarean section, of an overdue child after four days of labour. This was followed by post-partum shock, though the bleeding apparently was not excessive, and by complete failure of lactation. Normal menstruation did not return, though lower abdominal pain occurred at monthly intervals, accompanied latterly by a slight show. The symptoms of myxoedema had been relieved, though incompletely, by a small dose of thyroid extract (60 mg. a day).

On examination (February, 1955) one week after stopping thyroid treatment her skin and general appearance were those of mild myxoedema. The sexual hair, breasts, and vaginal epithelium were normal, and there was no loss of pigment in the skin or nipples.

Tests of endocrine function showed: B.M.R., -14% (Robertson and Reid standards); plasma cholesterol, 270 mg. per 100 ml.; E.C.G., normal; urinary 17-ketosteroids, 11.4 mg./24 hours; water-excretion test, 103% of water dose excreted in three hours (normal exceeds 70%); insulin-tolerance test (0.1 unit/kg.), return to 80% of fasting level at 45 minutes and to 84% at 2 hours; urinary F.S.H. excretion, less than 5 m.u./24 hours.* Results of the radio-iodine tests (February, 1955) are shown in the Table. Three months after these investigations had been completed the patient was found to be pregnant, and this was later confirmed radiologically.

Case 4 (L.V. 131003)

The patient, aged 40, was admitted with virus pneumonia in May, 1952. At a confinement 12 months earlier the

*Occasionally found in normal subjects at this laboratory.

blood loss had been slightly excessive, and was followed by heavy lochial bleeding for two months. Menstruation had then returned, with moderate menorrhagia. For at least four months she had complained of lassitude, pallor, anorexia, constipation, and paraesthesiae of the hands.

On examination she was pale and febrile, with signs of right basal pneumonia, but without any obvious myxoedema.

Investigations showed: Hb, 10.2 g./100 ml.; R.B.C., 3,100,000; M.C.V., 108 cubic microns; M.C.H.C., 30%; W.B.C., 8,000; sternal marrow, normoblastic hyperplasia; direct Coombs test positive, indirect positive at 20° C., negative at 37° C.; B.M.R., -25% (Boothby and Sandiford standards); plasma cholesterol, 240 mg. per 100 ml.; E.C.G., low voltage. Diagnoses of virus pneumonia, with haemolytic anaemia due to cold agglutination, and probable hypothyroidism were made.

On readmission three months later, tests of endocrine function showed plasma cholesterol, 325 mg. per 100 ml.; urinary 17-ketosteroids, 1.6 and 2.5 mg./24 hours; water-excretion tests, 59 and 71% excretion of water dose in three hours (normal exceeds 70%); insulin-tolerance test (0.1 unit/kg.), increased insulin sensitivity and hypoglycaemic unresponsiveness (blood sugar, 63% of fasting level at two hours); urinary F.S.H. excretion, less than 6 m.u./24 hours.* Results of radio-iodine tests (August, 1952) are shown in the Table.

On treatment with thyroid extract alone (120 mg. a day) the patient has remained well, with a normal E.C.G., and the haemoglobin has risen to 81%.

Radio-iodine Tests Before and After Thyrotrophin

In these four patients there was no significant uptake of radio-iodine either before or after a five- to nine-day course

of T.S.H. injections (see Table). With the knowledge that the T.S.H. was potent (see below) and on the assumption that it was given over a long enough period, these results would point to irreversible atrophy of the thyroid gland.

Normally the uptake of radio-iodine increases within 24 hours of an adequate dose of T.S.H., unless there is disease or atrophy of the thyroid gland. When secondary (hypopituitary) myxoedema has lasted for some years or is severe, so that the thyroid gland may be sluggish to respond, it may be necessary to give the T.S.H. for several days. Our practice, therefore, has been to give this hormone for at least five days before the second radio-iodine test. This procedure has already been recommended for testing patients whose myxoedema is suspected of being secondary to hypopituitarism (Querido and Stanbury, 1950; Werner *et al.*, 1950; Perloff *et al.*, 1951). Regardless of whether they are taking thyroid hormone, hypopituitary patients should respond to T.S.H., if this be given for several days, unless there is also severe thyroid atrophy. Such a test cannot assess anterior pituitary function directly, and can only determine whether an inactive thyroid gland is able to respond to the normal thyrotrophic stimulus. When there is no response, as in the four cases described, this does not exclude the possibility that the adenohypophysis may also be irreversibly damaged; anterior pituitary function must then be assessed by other tests.

In the Table we have included our data from radio-iodine tests in 25 other patients after T.S.H. injections. In those who served as controls, without evidence of thyroid or pituitary disease, the uptake of radio-iodine increased after T.S.H. In the thyroid addict, who for 10 years had been taking thyroid extract for neurasthenia and more recently at a dosage of 0.6 g. a day, the uptake was brought into the low normal range. By contrast there was no significant uptake after T.S.H. in any of the 12 patients whose myxoedema appeared clinically to be due to primary thyroid disease, whether or not they had been receiving thyroid hormone. The nine patients with typical hypopituitarism fell into two groups when assessed by their thyroid status—three patients without and six patients with obvious clinical features of myxoedema. Of the first group, two showed a definite and one a probable response to T.S.H. The response in the second group was definite in one, doubtful in two, and absent in three patients, although, with one exception (No. 17), T.S.H. was given for five to eight days.

Secondary but irreversible atrophy of the thyroid gland can evidently occur as a result of long-standing hypopituitarism, and will be found among cases of this condition with obvious myxoedema. Except in the three patients with hypopituitary myxoedema who did not respond, these results agreed with those of earlier reports (Querido and Stanbury, 1950; Werner *et al.*, 1950; Perloff *et al.*, 1951). A failure to respond to T.S.H. has been reported in only one other severe and long-standing case of hypopituitarism (Querido and Stanbury, 1950), and more recently in 2 out of 20 cases of pituitary insufficiency (Vale *et al.*, 1955).

Discussion

These patients had obvious myxoedema. At any early stage of the disease it was shown, by means of radio-iodine tests after the administration of T.S.H., that the thyroid failure was irreversible, and so probably due to primary atrophy of the gland. It is possible, though the necessary evidence is so far lacking, that a failure of pituitary thyrotrophic function may lead occasionally, and at an early stage, to irreversible thyroid atrophy, for instance, in a patient already destined to develop primary myxoedema. If this, however, were the mechanism in the present four cases, it would also have to be assumed that the only pituitary function affected was that of T.S.H. secretion, since the adrenal and gonadal functions were still intact, and lactation had failed only in one case (No. 3). Apparently, no proved

example of such a selective thyrotrophic failure has yet been reported (Fraser and Garrod, 1954), though failure of thyrotrophic and corticotrophic, without that of gonadotrophic, function has been described in one case (Wilson *et al.*, 1954, 1955).

The onset of myxoedema is usually slow and insidious. In the present four cases, therefore, it is possible that the thyroid gland had already started to atrophy before or during pregnancy, and that this process had merely become accelerated by the occurrence of a mild subclinical pituitary necrosis. Against this possibility were the very high titres of urinary gonadotrophins in two of these cases, and the histories of lactation in three.

Our T.S.H. tests in patients with hypopituitarism have shown that occasionally the thyroid gland may become irreversibly atrophied after a decade or more of this disease. This was certainly so in two of the present cases, and has also been reported in one other patient by Querido and Stanbury (1950). These last three patients all had gross Simmonds's disease, of long duration in two. It was thought possible, however, that primary myxoedema might have been coincidental in Case 16, which has already been described elsewhere (Garrod and Gilliland, 1954). In this instance a severe post-partum haemorrhage, followed by failure of lactation, had occurred 14 years earlier, although other symptoms of pituitary failure had been present for only two years. Fibrosis or complete atrophy of the thyroid gland has been found at necropsy in a small proportion of hypopituitary patients (Sheehan and Summers, 1949). Our findings suggest that the thyroid gland will become irreversibly atrophied in some, though not all, patients with Simmonds's disease who are obviously myxoedematous. Provided that this possibility is borne in mind, radio-iodine tests after stimulation by T.S.H. should prove useful in assessing patients whose myxoedema is thought to be secondary to hypopituitarism.

In conclusion, there are three reasons for believing that myxoedema was due to primary thyroid failure, rather than secondary to hypopituitarism, in these four patients: (1) the thyroid failure proved to be irreversible at an early stage of the disease; (2) after treatment with thyroid hormone alone there was no evidence of hypopituitarism; and (3) thyroid hormone alone fully corrected the disabilities.

Summary

Four cases are reported in which the onset of myxoedema appeared to date from post-partum shock or haemorrhage, but in which there was no evidence of hypopituitarism.

In none of these patients were there any signs of adrenal or gonadal insufficiency when tested after treatment with thyroid hormone alone, which fully corrected their disabilities.

Though the myxoedema was of recent onset, there was no thyroid uptake of radio-iodine, even after thyrotrophin (T.S.H.) injections had been given for five to nine days.

The results of radio-iodine tests in 25 other cases, before and after T.S.H. injections, are also presented. In two severe cases out of nine patients with hypopituitarism, and in all of 12 patients with primary myxoedema, there was no thyroid uptake of radio-iodine after an adequate course of T.S.H. injections, whereas all of the remaining patients (including a thyroid addict of 10 years' duration) showed an increased thyroid uptake.

Our thanks are due to Professor A. Kekwick for allowing us to study Case 3, and to Miss J. Briscoe and Mrs. M. Gray, of the radio-isotope laboratory of the Postgraduate Medical School, and Dr. R. Ekin, of the physics department, the Middlesex Hospital, for help with the radio-iodine tests.

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RETROMESOCOLIC HERNIA

BY

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Intra-abdominal herniae, excluding those which follow operative procedures such as gastro-jejunal anastomosis with or without gastrectomy (Stammers, 1954), are rare; a little over half are retromesocolic, occurring in the region of the duodeno-jejunal flexure.

Moynihan and Dobson (1906) described nine duodenal fossae; herniation occurred into only four of these. It would appear that herniation never occurs into the very common superior and inferior duodenal fossae, one or both of which are present in over three-quarters of the population; neither does it occur into the somewhat less common posterior duodenal and duodenal-jejunal fossae.

The most common retromesocolic hernia is the left duodenal hernia which takes place into the paraduodenal fossa (in the free border of which lies the inferior mesenteric vein). Somewhat less common is the mesocolic hernia into the mesocolic fossa, bordered by the ascending branch of the left colic artery; and the right duodenal hernia, which takes place into the mesentericoparietal fossa behind the superior mesenteric artery. More rarely still is seen the infra-duodenal hernia into the fossa of the same name.

Moynihan and Dobson's ninth fossa is the intermesocolic fossa (recessus intermesocolicus transversus of Brösike). This fossa is very rare; Brösike described six cases, and Toldt one probably of this nature (Moynihan and Dobson, 1906). Moynihan and Dobson considered that this fossa was never likely to develop hernial contents, but Schwalbe (1904) suggested that a hernia into this fossa may rupture through the mesocolon into the lesser sac, thus explaining certain transmesocolic herniae such as the case described by Chalmers (1905).

Description of Case

A girl aged 5 was admitted to hospital on September 14, 1954, having suffered for some 24 hours from generalized abdominal pain, associated with nausea but not with vomiting. The pain was getting worse and was colicky in nature. There had been no previous similar attacks. On examination the child was fretful, her temperature was 100.4° F. (38° C.), her tongue was clean. The abdomen was tense and tender all over, more on the right side than the left, but

there was no rigidity. The bowel sounds were active. Rectal examination revealed nothing abnormal. The tenderness was sufficient to suggest a diagnosis of appendicitis.

At laparotomy shortly after admission, through a McBurney incision, the right iliac fossa was found to be empty and the caecum could not be found. The small bowel was considerably distended. The incision was extended upwards and an unsuccessful search made for a sub-hepatic caecum: the transverse colon when traced to the right appeared to terminate at a normally placed hepatic flexure. The ileum was then followed; the dilated terminal ileum disappeared upwards behind the transverse mesocolon. Further palpation then revealed the caecum and appendix, with terminal ileum and ascending colon, to be rotated upward and lying in a pouch of peritoneum some 2 in. (5 cm.) deep behind the transverse mesocolon, to the right of the middle colic artery, and in front of the duodenum and pancreas (see Diagram).

The caecum lying in the sac was felt to be dilated, and the appendix, which was lying in front of the caecum with the tip directed downwards, was tense. During the manipulations slight traction was exerted on the terminal ileum, and suddenly the contents of the pouch were reduced. The terminal ileum, the caecum, and the ascending colon, all somewhat dilated, lay comfortably in their normal position, and showed no tendency to redisplacement. The appendix was now retrocaecal, with the tip pointing upwards; being tense and injected, it was removed, and was later found to contain many threadworms. The abdomen was closed and the child made an uninterrupted recovery, being discharged from hospital within a week.

Comment.—The clinical picture in this case was not typical of appendicitis, and it is probable that the symptoms were due to obstruction from hernial incarceration, precipitated by an increase in the tension in the hernial sac as a result of a mild inflammation of the appendix due to threadworms. Alternatively, the somewhat tense and injected appearance of the appendix may have been entirely secondary to the obstruction, the threadworms being a purely incidental finding. Whether the herniation into the pre-formed sac occurred at the onset of symptoms the day before, or whether it occurred some time previously, it is not possible to say, though there were no fine adhesions present to suggest a long-standing herniation. Simple reduction of the hernia is considered by Hansmann and Morton (1939) to be all that is necessary in such cases; they believe that agglutination of adjacent folds of peritoneum rapidly occurs, fixing the reduced parts in their normal position.

Discussion

The intermesocolic fossa of Brösike, which may occur on the right side as well as on the left, is the only described fossa which may possibly correspond to the above case. There are certain differences, however. The right fossa of Brösike would appear to be a narrower fossa running in the layers of the transverse mesocolon towards the right, with a relatively narrow opening directed downwards and to the left, the fossa itself being rather a potential than an actual

Retromesocolic hernia. Diagram to show position of sac behind transverse colon and transverse mesocolon.