4. *Clotting* is accelerated when thromboplastin and plasma are incubated together for more than four minutes before the addition of calcium chloride; it is essential to place no more thromboplastin-plasma mixtures in the water-bath than can be comfortably treated with calcium chloride and read within this time.

REFERENCES

- Allen, E. V., Hines, E. A., Kvale, W. F., and Barker, N. W. (1947). *Ann. intern. Med.*, **27**, 371.
 Bauer, G. (1946). *Lancet*, **1**, 447.
 Birger, I. (1947). *Acta chir. scand.*, **95**, Suppl. 129.
 Bruzelius, S. (1945). *Ibid.*, **92**, Suppl. 100.
 Frykholm, R. (1940). *Surg. Gynec. Obstet.*, **71**, 307.
 Fullerton, H. W. (1940). *Lancet*, **2**, 195.
 Hampton, A. O., and Castleman, B. (1940). *Amer. J. Roentgenol.*, **43**, 305.
 Quick, A. J. (1938). *J. Amer. med. Ass.*, **110**, 1658.
 Roholm, K. (1937). Quoted by J. E. Jorpes in *Heparin*, Oxf. Univ. Press, 1946.
 Sagall, E. L., Bornstein, J., and Wolff, L. (1945). *Arch. intern. Med.*, **76**, 234.
 Wilson, S. J. (1947). *Proc. Soc. exp. Biol., N.Y.*, **66**, 126.
 Witts, L. J., and Hopson, F. C. G. (1940). *Lancet*, **2**, 247.
 Zilliacus, H. (1946). *Acta med. scand.*, Suppl. 171.

DIABETIC COMA

TREATMENT WITH AND WITHOUT THE EARLY
ADMINISTRATION OF GLUCOSE

BY

J. LEE, M.B., M.R.C.P.

D. NAIDOO, B.Sc., M.R.C.P.Ed., D.C.H.

AND

J. A. TORRENS, M.D., F.R.C.P.

(From a Medical Unit, West Middlesex Hospital)

Whether or not glucose should be administered in the treatment of diabetic coma has been the subject of much discussion. On the one hand, Himsworth (1932) states that the "essential therapeutic agent in the treatment of diabetic intoxication is glucose, and unless this substance is present in sufficient amounts insulin is relatively impotent." He claims that the "maximum of sugar and the minimum of insulin" should be given, and "not conversely as is the present practice."

On the other hand, Root (1945a) holds that the harmful effects of glucose may be concealed in the early case of coma by the insulin simultaneously administered. He claims that the moderate case may be converted into a severe one requiring excessive insulin dosage, and that "in advanced coma glucose administration either by mouth or by needle may cause anuria and death."

Among those who administer glucose as a beneficial procedure are Bertram (1932), Lawrence (1936), and Soskin (1945). Joslin (1947) and Root (1945a) are the leading opponents of glucose administration, but they do give carbohydrate in quantities not exceeding 10 g. per hour during the second to sixth hour of treatment.

Physiological evidence supporting the administration of glucose is based largely on experimental work such as that of Soskin and Levine (1937, 1944), and of Mirsky *et al.* (1937). The last-named authors have shown that intravenous injection of large amounts of glucose (a) to anaesthetized nephrectomized depancreatized dogs, (b) to unanaesthetized depancreatized dogs, and (c) to phlorhizinized depancreatized dogs, where insulin was entirely excluded, resulted in the disappearance of ketone bodies. The antiketogenic action of glucose is attributed to the deposition of glycogen in the liver and a consequent cessation of ketone formation. Soskin and Levine's (1937) depancreatized hepatectomized animals utilized larger amounts of carbohydrate without insulin when their blood

sugars were maintained at high levels. Peters (1945), basing his opinion upon a large field of experimental work, comes to the conclusion that the reversal of the metabolic disorder in diabetic coma requires the use of every measure that will accelerate the utilization of carbohydrate. "Chief among these measures are the administration of insulin and the provision of sugar." Soskin and Levine (1944) calculated a large carbohydrate deficit and advised the administration of 500 g. of carbohydrate during the first twenty-four hours of treatment and one-half that amount during subsequent days in order to maintain normal stores of glycogen and carbohydrate metabolism.

Root (1945b), however, disagrees. He found a deficit of only 27 g. and stressed the point that there has been "an overestimation of the importance of an immediate restoration of glycogen stores in the liver." Franks *et al.* (1947) found that comparable doses of dextrose were retained more efficiently under much smaller doses of insulin when the blood-sugar level was normal than when it was considerably elevated. Mirsky's earlier work, suggesting that maintenance of hyperglycaemia would hasten the abolition of ketosis, is not borne out by Franks's clinical material. Pauls and Drury (1942), working on diabetic rats receiving large quantities of sugar and insulin, provided evidence that only a small part of the sugar was oxidized and that most of it was stored as fat. Drury (1940) showed that glucose may also be converted into fat when it is given at a rate exceeding calorific needs. Astwood's (1942) dogs, receiving glucose continuously at a rate exceeding their metabolic needs, died within 70 hours. Further, Bjerling and Iversen (1934) demonstrated that diabetic acidosis exaggerates the limitation of the power of the tubules to reabsorb glucose, and that there is poor retention of exogenous glucose in the presence of hyperglycaemia. Helmholz and Bollman (1940) produced a marked diuresis in response to high concentrations of glucose brought about by injection, and as a result hydropic degeneration of the tubules occurred and temporary anuria developed. Franks *et al.* (1947) showed a "striking difference" between the glucose and saline groups of cases in the handling of water and chloride. The fluid intake of the dextrose group was only slightly larger than that of the saline group, but the output was nearly four times as great, "largely because of an elevenfold difference in glucose output." Sunderman and Dohan (1941) found that hyperglycaemia causes the withdrawal of water from the tissues, and, conversely, that the administration of insulin, with the consequent fall in blood sugar, caused the transfer of water and electrolytes from the plasma to the tissues. In addition, cases of acidosis lost body water and electrolytes after the withdrawal of insulin (Atchley *et al.*, 1933).

The consistent high mortality for cases of diabetic coma in England due to circulatory failure and "intractable dehydration" led us to reconsider the value of the therapeutic measures employed, particularly with regard to the use of glucose and its effect on procedures adopted to correct dehydration.

Material

The West Middlesex is a hospital of 1,670 beds serving a wide area in south-west Middlesex. A large proportion of cases of diabetic coma are admitted as emergencies, never having been under the management of the physician in charge of the diabetic out-patient clinic. Only those cases which are unconscious and not responsive to external stimuli are considered cases of coma. All others, irrespective of the level of the blood sugar, are treated as cases of diabetic ketosis or pre-coma. The mortality in cases of diabetic pre-coma is negligible whether or not glucose is administered in the early stages of treatment.

The 28 consecutive cases of true diabetic coma which were admitted to this medical unit between September, 1944, and March, 1948, have been studied. Those cases admitted before February, 1946 (Group A), were given glucose throughout the first twenty-four hours of treatment, whereas cases admitted after that date (Group B) had no glucose until the blood sugar had reached normal levels or, alternatively, until the patient was able to sit up and drink the glucose without help.

Procedure

On receipt of information from the admissions officer a bed with electric blankets, enema and gastric lavage equipment, "cutting-down set" with pint flasks of normal saline, together with syringes and bottles for chemical investigations, are held in readiness. On arrival the patient is put to bed, surrounded by warm blankets. The diagnosis is confirmed and the severity of the attendant infection assessed. A gastric lavage and a cleansing enema are given while the house-physician cuts down on a vein, removes blood for chemical testing, injects insulin, and proceeds, as the ward sister finishes her work, to set up the intravenous drip.

Insulin.—The mental state of the patient and the severity of the concurrent infection determine the size of the initial dose of insulin, which is never less than 100 units. Where a patient's mental state is graded 4 (Collen) 200 units are usually administered, a quarter of it intravenously. Insulin is given at intervals of not less than two hours thereafter.

Fluids.—The Group A or glucose cases receive intravenous normal saline with 4.3% glucose, and Group B or saline cases receive normal saline intravenously until there is adequate correction of the hypochloruria, after which half-normal

definition of coma varies widely and there is no entirely satisfactory method of assessing the severity of individual cases. Timing the duration of coma is not always a simple matter, and it is difficult to obtain a history of exactly when, in the insidious course of diabetic ketosis, a patient lapsed from one mental state to another. Further, there is no definite evidence that the duration of coma up to about eight hours, taken by itself, correlates closely with the ultimate outcome. Collen's indices have been used, as they appear to be the most accurate available guide to prognosis (Collen, 1942). He computed his severity index by means of the following formula

$$\frac{30(\text{M.S.} + \text{C}) + 50 + \text{A} - \text{diastolic B.P.}}{4}$$

where M.S. is the mental state and C the complications.

M.S. is graded thus: 0 = conscious, 1 = drowsy, 2 = semi-conscious, 3 = unconscious but with response to pain, 4 = unconscious with no response to pain.

C is "complications," graded numerically from 0 to 5 in terms of danger to life. Grade 1 is "infection or other complication present" but of no real consequence. Grade 5 is "infection or other complication present" which by itself would seriously endanger life. Examples: Grade 1 — carbuncles (Case B1), pyelitis (Case B6); Grade 3 — lobar pneumonia (Case B14); Grade 5 — bilateral empyema (Case B8).

A = Age in years.

Indices of over 60% are considered invariably fatal and those of below 30% bear a good prognosis. As Franks remarks, it would appear that only those cases in the "intermediate group are suitable for the evaluation of a therapeutic regimen."

TABLE I.—Group A Cases

Case No.	Age and Sex	Duration of Coma (hours)	M.S.	C.	B.P.	Collen's Index %	Blood Sugar (mg. per 100 ml.)						Alkali Reserve (Vols. CO ₂ %)				Insulin Units (Total Dose)					Fluids (pints)		Deaths
							Adm.	4 hrs.	8 hrs.	12 hrs.	16 hrs.	20 hrs.	Adm.	4 hrs.	8 hrs.	12 hrs.	Adm.	4 hrs.	8 hrs.	12 hrs.	24 hrs.	Intra-venous 24 hrs.	Total 24 hrs.	
1	55 F	4	4	2	100/55	57	880	760					8	12			200	400	600			6		Died
2	61 M	4	3	2	145/90	43	768	610	463				17				200	400	600	800		8		
3	26 M	3			110/70	24	660	580	480	312	180	214	12	18	31		200	400	560	680	800	9	14	Died
4	31 F	2	3	1	90/60	35	510	500	495	216	204	214	15	16	18	26	200	360	440	480	600	12	15	
5	56 F	4	4	2	130/75	53	804	830					9	8	13		200	400	760			8		Died
6	18 F	6	3	—	100/65	23	822	750	598	580	312	226	18	21	28	33	100	300	500	680	800	11	17	
7	32 M	6	4	2	50/30	58	985	810	805	620			5	8			200	380	580	700		11		Died
8	37 F	4	4	—	70/50	38	708	648	630	524	475		14	19			200	400	600	800	1,060	8	15	
9	29 F	4	3	1	100/75	31	540	438	256	180	84		13	15	21	26	100	300	460	580	740	8	13	Died
10	14 F	2	4	1	95/65	37	618	616	543	205	198	216	17	30	35		200	460	600	740	840	9	12	

saline is given. Every case receives not less than 2 pints (1.14 litres) of fluid intravenously in the first hour. In the older diabetic, or in cases in which cardiac embarrassment is expected, an intragastric drip is set up after the fourth to sixth hour, even though altered blood may have been present in the stomach contents during lavage.

Glucose.—The saline cases do not receive glucose in the first four-hour period, and in the second four-hour period it is given only if the patient becomes fully conscious. In the third four-hour period glucose is always administered, usually by mouth. Not more than 100 g. is given over the first 24-hour period in any one case in the saline series. The glucose cases receive a standard solution of 4.3% glucose in normal saline, whether by vein, mouth, or intragastric drip.

Potassium.—Since the publication of Holler's (1946) case and the subsequent cases by Nicholson and Branning (1947) and by Martin and Wertman (1947), about 1 dr. (4 g.) of potassium chloride is left in the stomach after lavage in the most severe cases.

Results

A comparison of therapeutic results between different series of cases of diabetic coma is not always easy. The

Group A or Glucose Cases

Group A comprises ten cases ranging from 14 to 61 years, with an average of 35.9 years. Seven cases were in females. The figures given for the duration of coma are approximations. On admission all the cases described were unconscious and did not respond to external stimuli. Five cases responded feebly to a strong painful stimulus applied to the sole of the foot. Complications were few in this group and were mild in nature. The diastolic blood pressure ranged from 30 to 90 mm. Hg. Collen's formula was worked out and indices of between 23 and 58% (average 39.9%, mean deviation 10.2) were found. The four patients (Cases 1, 2, 5, 7) who died had indices of 57, 43, 53, and 58% respectively.

The initial blood sugar for the ten cases averaged 729.5 mg. per 100 ml., the lowest being 510 mg. and the highest 985 mg. The fall during the first four hours was calculated for each case: the readings for Case 5 rose, but the mean fall was 75.3 mg. per 100 ml.

Fluid intake for the survivors over the first 24 hours was 14.3 pints (8.13 litres), of which 9.5 pints (5.39 litres) were given intravenously and the remainder by other routes, such as by intragastric drip, by rectum, and by mouth. Glucose was administered in this group at the rate of 46 g. in the first hour,

23 g. in the second, and between 20 and 30 g. in the succeeding two hours. The average total glucose intake was 330 g. in 24 hours. The volume of the urinary output was measured in five cases. The output was 43.3% of the fluid intake.

The initial dose of insulin was either 100 or 200 units, with similar amounts given at two-hourly intervals until the readings showed definite signs of a fall. An average of 807 units was

The most significant result is that the mortality in Group A was 40% and in Group B 11%. Further, the fall in blood sugar in the first eight hours was more pronounced in the saline group and the output of urine (eight cases) was 21% of the total intake for the twenty-four hours, whereas in the glucose cases urinary output (five cases) amounted to 43% of the intake. Nearly 50% more fluid

TABLE II.—Group B Cases

Case No.	Age and Sex	Duration of Coma (hours)	M.S.	C.	B.P.	Collen's Index %	Blood Sugar (mg. per 100 ml.)					Alkali Reserve (Vols. CO ₂ %)				Insulin Units (Total Dose)					Fluids (pints)		Deaths	
							Adm.	4 hrs.	8 hrs.	12 hrs.	16 hrs.	20 hrs.	Adm.	4 hrs.	8 hrs.	12 hrs.	Adm.	4 hrs.	8 hrs.	12 hrs.	24 hrs.	Intra-venous 24 hrs.		Total 24 hrs.
1	16 M	3	3	1	100/60	32	984	540	350	212	180		16	38			200	360	420	480	580	6	9	Died
2	42 F	2	3	2	105/65	44	780	420	318	232			18	18	30	50	200	360	480	600	700	5	8½	
3	48 F	5	4	1	100/50	50	1,050	788	280				11	26			200	380	460	520	640	6	9	
4	55 F	5	3	1	110/55	43	835	418	242				15	30			100	300	420	500	620	5	11	
5	42 M	1	3	1	100/70	35	900	816	340				12	14			200	380	460	520	640	2*	7	
6	40 F	3	3	1	125/70	35	597	194	120				14	40			200	360	420	480	580	4	9	
7	45 M	4	3	—	130/80	26	980	630	318				14	26			100	300	500	660	860	8	11	
8	22 M	6	4	5	40/0	86	954	?	648				9	11			300	600	760	920	1,100	8	9½	
9	25 F	5	4	—	95/55	35	1,150	678	328				10	25			100	300	500	640	740	5	10	
10	33 F	3	3	1	125/75	32	786	486	320				16	32			100	300	460	540	680	6	10	
11	19 M	½	3	2	125/80	35	1,074	728	—	246			15	—	39		200	400	600	850	1,050	8	11	
12	17 F	3	3	1	110/75	28	832	528	—	308			22	42			100	300	460	620	780	5	10	
13	11 F	5	4	1	95/65	37	542	282	102	265			15	32	45		100	300	360	400	480	4	9	
14	27 F	2	3	3	120/80	44	764	324					18	—			100	300	480	540	680	5	12	
15	42 F	2	4	3	115/60	60	924	502	280				10	10			200	400	560			7	Died	
16	13 F	Coma on adm.	4	—	90/50	33	630	380					?	30			80	240	360	460	560	5		9
17	44 M	2	3	3	110/80	49	920	540	324				17	48			200	380	460	520	640	7	14	
18	58 F	4	3	2	135/80	45	860	500					9	21			200	400	560	720	860	4	11	

*_Case 5 received only two pints of fluid intravenously, the third and fourth pints having inadvertently dripped into the bedclothes. Nevertheless, the patient rehydrated adequately.

given to each surviving case in 24 hours. The urine became acetone-free in this group of cases in 13 hours (range 10 to 24).

Group B or Saline Cases

This group comprises eighteen cases ranging from 11 to 58 years, with an average of 33.3 years. Twelve cases were in females. Six cases fell into the most serious grading of mental state, whereas the remaining twelve responded to strong painful stimuli applied to the sole of the foot. The diastolic blood pressure fell in some cases to dangerous levels. The lowest recording of 40/0 mm. Hg was for Case 8, who was nearly moribund. The highest readings, 135/80 mm. Hg, were in Case 18, whose pressures rose in convalescence to 185/110. There were all degrees of complications, eight being Grade 1, three Grade 2, three Grade 3, and one Grade 5. Case 15 had lobar pneumonia and Case 8 suffered from bilateral empyemata. These two patients (severity indices 60 and 86% respectively) died. Collen's indices for the whole group ranged from 26 to 86%, with an average of 41.6% (mean deviation 9.8).

The mean initial blood sugar was 865 mg. per 100 ml., the lowest being 542 mg. and the highest 1,150 mg. The calculated fall in the first four hours was 339 mg. The total 24-hour fluid intake for the sixteen surviving patients was almost exactly 10 pints (5.68 litres), of which 5 pints (2.84 litres) were given intravenously. The total urinary output in those cases (eight in number) where the urine was successfully collected and measured amounted to 21% of the total 24-hour intake. The initial dose of insulin was either 100 or 200 units. Three hundred units were given in one case (100 intravenously), but the fourth-hour sugar reading was unfortunately lost. The average total dose of insulin given over 24 hours was 693 units. Acetone disappeared from the urine in 14 hours (range 11 to 24+).

The two groups of cases considered are of nearly equal severity and the age distribution is very similar. In other respects, such as depth and duration of coma, incidence of complications, level of diastolic pressure, and height of initial blood sugar, the groups of cases are comparable.

The procedure adopted in treatment throughout the first twenty-four hours was identical in the two groups except for the fact that Group B cases did not have glucose in the early stages of treatment.

was required to produce an adequate clinical state of hydration in those surviving cases given glucose.

Comment

There is a big difference in the mortality of two groups of comparable cases treated with and without glucose by what was otherwise the same therapeutic technique. Arguments in favour of the point of view that glucose is utilized at high blood-sugar levels, up to 2,000 mg. per 100 ml. (Wierzuchowski and Bielinski, 1939), and that its administration in cases of coma restores carbohydrate metabolism, abolishes acidosis, and replenishes the liver glycogen would seem to be physiologically incontrovertible. Peters (1945) has made out a very good case for glucose administration on these grounds. Much of the literature and the mortality statistics relating to this controversy are from America, where cases described as those of coma include cases of what would be labelled pre-coma or ketosis in Britain. It would seem, therefore, that American authors dealing with large numbers of cases of ketosis and coma find that only a small proportion present as emergencies of dehydration.

It is possible that because of this there has been, as Root suggests, "an overemphasis of the importance of an immediate restoration of glycogen stores in the liver." The controversy is still being fought on physiological issues. The main point disputed is whether or not a patient in diabetic coma can utilize glucose at a high blood-sugar level.

Still undetermined are such subsidiary points as whether the endogenous glucose is sufficient for purposes of carbohydrate metabolism when insulin is supplied, what quantities of glucose are retained at what level of blood sugar and under what doses of insulin, and whether or not the early administration of glucose abolishes ketosis more quickly. None of these points is by any means decided for the diabetic human being, although experimental animals with induced glycosuria give more or less conclusive results in favour of glucose utilization and glycogen restoration at high blood-sugar levels.

It is possible that the higher mortality for cases which were given glucose is not connected with the action of glucose on carbohydrate metabolism, which is probably beneficial, but on its interference with the process of cellular rehydration. The maintenance of hyperglycaemia induces an intense and prolonged glycosuria and, associated with it, a proportional diuresis. The urinary output bears a linear ratio to the glucose output (Franks *et al.*, 1947). The urinary output in our Group A cases was 43.3% of the total intake, and the fluid intake in these cases was 43% more than in the cases not given glucose.

The administration of glucose in the first four hours of treatment, by maintaining a heightened blood sugar, serves to increase water excretion, to worsen the state of cellular dehydration, and to add to the circulatory collapse and shock. This process may be of no great consequence in the milder cases, but where the clinical state of the patient is poor it may cause death.

The results of this study indicate that glucose should not be administered in the early stages of the treatment of diabetic coma. The withholding of glucose for at least the first four to six hours of treatment ensures rapid rehydration and decreases the mortality significantly in serious cases of coma with circulatory collapse.

Summary

A clinical study of 28 consecutive cases of diabetic coma treated with and without the early administration of glucose is described.

Ten cases received glucose and 18 only saline in the first four to six hours of treatment. The two groups of cases were comparable.

There was a mortality of 40% in the glucose cases and of 11% in the saline cases.

The results of this study indicate that the administration of glucose to serious cases of diabetic coma in the early stages of treatment interferes with rapid rehydration and increases the mortality significantly.

BIBLIOGRAPHY

- Astwood, E. B., *et al.* (1942). *J. clin. Invest.*, **21**, 621.
 Atchley, D. W., *et al.* (1933). *Ibid.*, **12**, 297.
 Bertram, F. (1932). *Klin. Wschr.*, **11**, 1998.
 — (1939). *Die Zuckerkrankheit*. Leipzig.
 Bjering, T., and Iversen, P. (1934). *Acta med. scand.*, **82**, 193.
 Collen, M. F. (1942). *Arch. intern. med.*, **70**, 347.
 Darrow, D. C., and Yannet, H. (1935). *J. clin. Invest.*, **14**, 266.
 Drury, D. R. (1940). *Amer. J. Physiol.*, **131**, 536.
 — and Palmer, J. J. (1938). *Proc. Soc. exp. Biol., N.Y.*, **38**, 394.
 Franks, M., *et al.* (1947). *Arch. intern. Med.*, **80**, 739.
 Hartmann, A. F. (1935). *Ibid.*, **56**, 413.
 Helmholz, H. F., and Bollman, J. L. (1940). *J. Lab. clin. Med.*, **25**, 1180.
 Himsworth, H. P. (1932). *Lancet*, **2**, 165.
 Holler, J. W. (1946). *J. Amer. med. Ass.*, **131**, 1186.
 John, H. J. (1935). *Ibid.*, **105**, 587.
 Joslin, E. P. (1947). *Treatment of Diabetes Mellitus*, 8th ed. Philadelphia.
 Lawrence, R. D. (1936). *British Medical Journal*, **2**, 81.
 Martin, H. E., and Wertman, M. (1947). *J. clin. Invest.*, **26**, 217.
 Mirsky, I. A., *et al.* (1937). *Amer. J. Physiol.*, **118**, 290.
 — (1941). *J. clin. Endocrinol.*, **1**, 307.
 Nicholson, W. M., and Branning, W. S. (1947). *J. Amer. med. Ass.*, **134**, 1292.
 Pauls, F., and Drury, D. R. (1942). *J. biol. Chem.*, **145**, 481.
 Peters, J. P. (1945). *Yale J. Biol. Med.*, **17**, 705.
 — (1946). *Amer. J. digest. Dis.*, **13**, 127.
 Root, H. F. (1945a). *J. Amer. med. Ass.*, **127**, 557.
 — (1945b). *J. clin. Endocrinol.*, **5**, 353.
 — and Carpenter, T. M. (1943). *Amer. J. med. Sci.*, **206**, 234.
 — and Riseman, J. E. F. (1938). *J. Amer. med. Ass.*, **110**, 1730.
 Soskin S. (1945). *Ibid.*, **127**, 563 (in discussion).
 — and Levine, R. (1937). *Amer. J. Physiol.*, **120**, 761.
 — (1944). *Amer. J. digest. Dis.*, **11**, 305.
 Sunderman, F. W. (1943). *Amer. J. med. Sci.*, **205**, 102.
 — and Dohan, F. C. (1941). *Amer. J. Physiol.*, **132**, 418.
 Weiner, H. J. (1938). *Amer. J. med. Sci.*, **196**, 211.
 Wierzechowski, M., and Bielinski, Z. (1939). *C. R. Soc. Biol.*, **130**, 1552.

SUBACUTE PANCREATITIS

BY

IAN MACNAB, F.R.C.S.

Late Surgical Registrar, General Hospital, Birmingham

The most commonly recognized types of pancreatitis are the acute haemorrhagic form, with its dramatic clinical picture and high mortality rate, and the chronic type, with a long-drawn-out history of dyspepsia associated with general ill-health. Of the other degrees of pancreatitis, there is the variety which gives rise to a prolonged illness, with abscess formation, and the low-grade pancreatitis which may later progress to pseudocyst formation.

All these types are well known, but there remains another degree of the condition—subacute pancreatitis—which is not so widely recognized. Subacute pancreatitis is a fairly rapidly developing inflammation of the pancreas, generally associated with much oedema, which, though presenting as a surgical emergency, does not produce the same urgent clinical picture as is presented by the haemorrhagic form. There is a marked tendency to recurrence, and the initial attack is generally followed by several similar attacks over a period of time, producing the syndrome of recurrent pancreatitis. During one year at the General Hospital, Birmingham, there were 19 cases of subacute pancreatitis, of which 13 were recurrent attacks.

Pathology : Clinical Picture

At operation the gland is found to be enlarged and oedematous. The enlargement is sometimes very gross, and in three cases abdominal palpation revealed a mass which was found at operation to be an enlarged pancreas. In one instance the oedematous pancreas produced pyloric obstruction. The surface of the gland may present petechial haemorrhages and the peripancreatic lymph nodes are enlarged. There may be some free fluid in the peritoneum, but it is not blood-stained as in haemorrhagic pancreatitis. In two cases there was fat necrosis in the omentum.

The clinical findings are shown in Tables I and II. The patient, usually middle-aged and obese, develops upper abdominal pain which, though not severe at first, gradually increases in intensity over several hours until it is unbearable. The pain may be situated in the epigastrium or in the right or left hypochondrium. In some cases it is felt right across the abdomen under the subcostal margin. The pain, which may continue unabated for several hours, does not vary in intensity. When asked to describe the pain the patient generally states that it is deep-seated and boring in nature and that it passes through to the back. Vomiting occurs early, and when the stomach contents have been emptied incessant retching adds to the patient's distress. The temperature is normal, but the pulse rate is generally raised. There is some tenderness and guarding in the upper abdomen, and this may be present anywhere in the subcostal region. There is tenderness in one or both loins and in the costovertebral angles posteriorly. There are some inconstant signs, such as jaundice, due to obstruction of the common bile duct by the swollen pancreas, and occasionally glycosuria. In cases presenting recurrent attacks the clinical picture is similar, but vomiting is not severe, and the tenderness, when present, is only slight.

Laboratory Investigations

Only two of the multitude of investigations described for pancreatitis have proved of use in establishing the diagnosis—estimation of the level of the serum lipase and serum