Hospital Topics

Elective End-to-side Portacaval Shunt: Results in 64 Cases

K. HOURIGAN, SHEILA SHERLOCK, PHYLLIS GEORGE, S. MINDEL

British Medical Journal, 1971, 4, 473-477

Summary

In a series of 64 cases of elective end-to-side portacaval shunts performed for liver disease the success rate —in that the patient survived with a patent shunt, free of subsequent haemorrhage and severe encephalopathy—was 48%.

The early postoperative death rate was 12·5% and the five-year survival 65%. Bleeding from oesophagogastric varices after blockage of the shunt was responsible for at least half of the early postoperative deaths, and most late deaths were due to liver failure. A decreased chance of late survival was associated with age over 40 years, active chronic hepatitis, and with a preoperative history of hepatocellular jaundice.

Shunt blockage occurred in 16% of patients, and all bled again from oesophagogastric varices. Shunt block is more likely if the portal vein is calcified or thrombosed, and may be more likely if the portal vein diameter, as shown by splenic venography, is 1·5 cm or less.

In survivors with a patent shunt the most serious late complication was chronic, severe portal-systemic encephalopathy, which occurred in 38%. Severe encephalopathy was associated with age over 40 years, a preoperative history of any degree of encephalopathy, diabetes mellitus, and with continued drinking in the alcoholic. Most patients who had portal-systemic encephalopathy in the first year postoperatively developed chronic disabling encephalopathy.

A preoperative history of transient mild or moderate ascites did not seem adversely to influence the outcome.

Introduction

The mortality from bleeding oesophagogastric varices is high and varies from 25%[1] to 54%[2] in reported series of cirrhotic patients in Britain. One-year survival after the first bleed in portal hypertension with cirrhosis varies from 67%[3] in Britain to 30%[4] and 34%[5] in the United States, where poor-risk alcoholics predominate. Elective end-to-side portacaval anastomosis in selected good-risk cases is the recognized treatment of choice for proved bleeding oesophagogastric varices in portal hypertension and will greatly reduce the risk of rebleeding and relieve the patient of the constant fear of sudden exsanguinating haemorrhage. The operation, however, is not without risk; there is a high incidence of postoperative portal-systemic encephalopathy, and an increase in longevity has not yet been shown in a randomized controlled trial.

The purpose of this paper is to review the results of 64 cases of elective end-to-side portacaval shunt performed consecutively at the Royal Free Hospital by one surgeon.

Royal Free Hospital, London W.C.1

K. HOURIGAN, M.R.C.P., M.R.A.C.P., Catoni Research Fellow (Present address: Mater Misericordiae Public Hospital, Brisbane, Queensland, Australia)

SHEILA SHERLOCK, M.D., F.R.C.P.(LOND., ED.), Professor of Medicine

PHYLLIS GEORGE, M.B., B.S., F.R.C.S., Consultant Surgeon

S. MINDEL, M.B., B.S., D.M.R.D., Senior Registrar in Radiology (Present address: Sydney Hospital, Sydney, N.S.W., Australia)

Patients and Methods

Sixty-four patients underwent elective end-to-side portacaval anastomosis consecutively between August 1962 and June 1970. There were 38 males and 26 females aged 13 to 71 (mean 39) years. The period of follow-up ranged from 3 to 96 (mean 50) months. All the patients had definite portal hypertension and had previously bled from proved oesophagogastric varices. In all cases a patent portal vein was shown by percutaneous splenoportography or venous-phase selective abdominal arteriography. At the time of surgery the patients were free of bleeding, liver failure and encephalopathy, clinically detectable ascites, and jaundice and had a serum bilirubin level of less than 2 mg/100 ml (except those with biliary cirrhosis) and a serum albumin of at least 3 g/100 ml; none had any concomitant medical contraindication to major surgery.

There was a history of transient hepatic encephalopathy after haemorrhage in eight cases and a serum albumin of less than 3 g/100 ml in five; ascites (transient and mild to moderate) had been present in 29 cases and serum total bilirubin greater than 2·5 mg/100 ml in 17 (apart from those with biliary cirrhosis) occurring after haemorrhage in most cases. All the patients were assessed preoperatively in this medical unit and the shunts were performed by the one surgeon (P.G.). Regular follow-up was maintained in the medical and surgical unit outpatient departments and final status assessed personally up to the time of writing, loss to follow-up, or death, except in five cases where for geographical reasons the opinion of the attending consultant physician was accepted. In the four cases lost to follow-up no assumptions of progress from the last assessment were made.

The aetiology in the 64 cases is listed in Table I. There was underlying cirrhosis in all but three—two cases of portal vein block in the region of the porta hepatis and one case of chronic sarcoidosis with portal fibrosis. The diagnosis in all cases was supported by liver biopsy. Of the seven patients with biliary

TABLE I—*Aetiology in 64 Patients undergoing Elective End-to-side Portacava Shunts*

Cryptogenic cirrhosis	34
Alcoholic cirrhosis	10
Biliary cirrhosis	7
Active chronic hepatitis and cirrhosis	7
Portal vein block	2
Schistosomiasis	2
Haemochromatosis	1
Sarcoidosis	1

cirrhosis four had primary biliary cirrhosis, two had biliary cirrhosis and ulcerative colitis, and one had biliary cirrhosis and diverticular disease of the jejunum with chronic infection.

Portal vein diameters on preoperative radiographs were measured by one of us (S.M.) who had no knowledge of the findings at surgery or of the results. The measurement was made 1 cm from the bifurcation of the main portal vein. Patency of the shunt was diagnosed by diminution of spleen size, diminution or disappearance of varices on fluoroscopy and endoscopy, and by percutaneous splenic manometry and splenoportography or venous-phase abdominal angiography where indicated. Necropsy was performed in all but two of the patients who died.

Results

EARLY POSTOPERATIVE DEATH

Eight patients (12·5%) died in the early postoperative period (Table II). Cause of death was progressive hepatic failure in three cases, bleeding varices due to proved blocked shunt in four, and upper gastrointestinal bleeding in one case where shunt blockage was suspected but not proved and postmortem examination was not performed. Of the three patients dying of liver failure with a patent shunt one aged 46 (with cryptogenic cirrhosis) had a preoperative history of jaundice, ascites, and encephalopathy after bleeding, another aged 14 (with active chronic hepatitis and cirrhosis) had had transient jaundice only, and the third, aged 19 (with cryptogenic cirrhosis) had had no preoperative jaundice, ascites, or encephalopathy. Serum albumin in all three cases had been more than 3 g/100 ml.

TABLE II—*Details of the 8 Early Postoperative Deaths*

Diagnosis	Age	Shunt	Cause of Death
Cryptogenic cirrhosis	57	Blocked	Bleeding varices
Active chronic hepatitis and cirrhosis	13	? Blocked	Bleeding varices
Biliary cirrhosis	42	Blocked	Bleeding varices
Cryptogenic cirrhosis	19	Patent	Hepatic failure
Alcoholic cirrhosis	49	Blocked	Bleeding varices
Cryptogenic cirrhosis	46	Patent	Hepatic failure
Active chronic hepatitis and cirrhosis	14	Patent	Hepatic failure
Schistosomal cirrhosis	27	Blocked	Bleeding varices

LATE SURVIVAL

Altogether 84% of the patients survived one year or more, 65% survived five years or more, 55% survived seven years or more, and one patient survived eight years (Table III). Eleven patients died after the early postoperative period, nine from liver failure, one after a fractured femur, and one from fungal septicaemia.

TABLE III—*Survival after End-to-side Portacaval Shunt*

	Time (Years)								
	Postop.	6/12	1	2	3	4	5	6	7
No. of patients	64	62	58	50	40	32	23	18	11
Percentage survival	88	87	84	78	75	69	65	50	55

Survival rates were significantly lower in the group with active chronic hepatitis and cirrhosis compared with those of patients with cryptogenic cirrhosis, alcoholic cirrhosis, and biliary cirrhosis (Tables IV-VII) despite a much lower mean age in the active chronic hepatitis group. Those aged 40 years and under survived longer—78% survival at five years compared with 57% survival at five years in those over 40 (Fig. 1).

Eight patients had a preoperative history of hepatic encephalopathy after variceal bleeding. In this group there were two early postoperative deaths, one from liver failure and one from bleeding due to blockage of the shunt. There were three late deaths, two from liver failure at 60 and 89 months, and one at 33 months from variceal bleeding due to a blocked shunt. The remaining three were alive 21, 54, and 63 months postoperatively.

In five patients the serum albumin was less than 3 g/100 ml at some time preoperatively. Two died in the early postoperative period from bleeding due to blockage of the shunt. Two were lost to follow-up at 2 and 10 months and one was alive 64 months postoperatively.

Those who had had hepatocellular jaundice (69% survival at one year) fared significantly worse than those who had not (91% survival at one year) (Fig. 2). A preoperative history of ascites did not seem to influence survival (Fig. 3).

TABLE IV—*Survival after Portacaval Shunt in Patients with Active Chronic Hepatitis*

	Time (Years)								
	Postop.	6/12	1	2	3	4	5	6	7
No. of patients	7	7	7	7	4	2	2	2	1
Percentage survival	71	71	57	57	50	0	0	0	0

TABLE V—*Survival after Portacaval Shunt in Patients with Cryptogenic Cirrhosis*

	Time (Years)								
	Postop.	6/12	1	2	3	4	5	6	7
No. of patients	34	32	30	27	23	18	14	10	7
Percentage survival	91	91	90	81	83	83	79	60	57

TABLE VI—*Survival after Portacaval Shunt in Patients with Alcoholic Cirrhosis*

	Time (Years)								
	Postop.	6/12	1	2	3	4	5	6	7
No. of patients	10	10	9	6	5	4	2	1	0
Percentage survival	90	90	89	83	80	25	0	0	0

TABLE VII—*Survival after Portacaval Shunt in Patients with Biliary Cirrhosis*

	Time (Years)								
	Postop.	6/12	1	2	3	4	5	6	7
No. of patients	7	7	7	5	4	4	3	3	2
Percentage survival	86	86	86	80	50	50	67	33	50

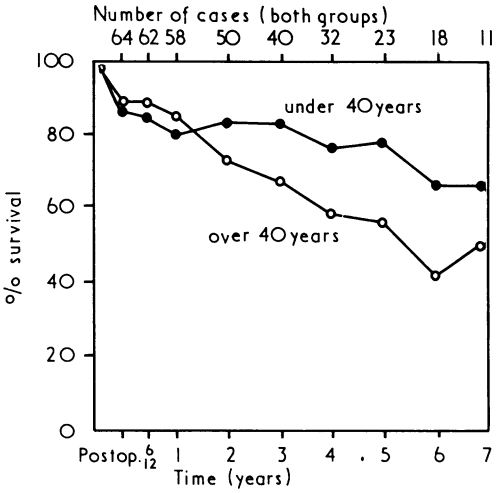

FIG. 1—Relation of age to survival after end-to-side portacaval shunt.

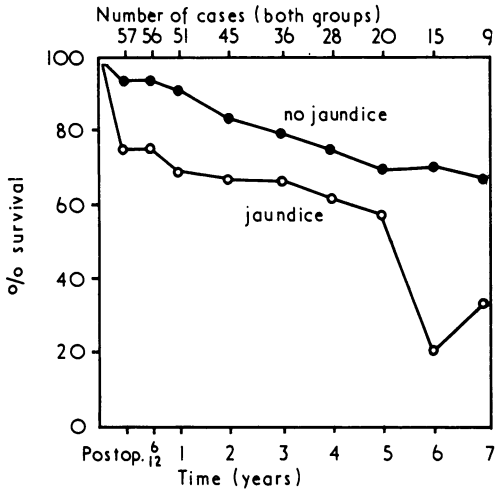

FIG. 2—Relation of preoperative hepatocellular jaundice to survival after end-to-side portacaval shunt.

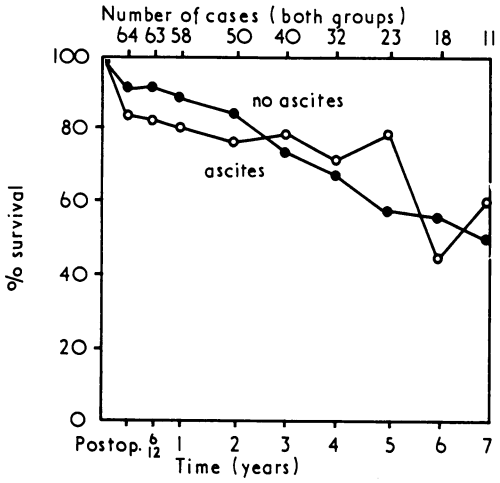

FIG. 3—Relation of preoperative ascites to survival after end-to-side portacaval shunt.

COMPLICATIONS OF BLOCKED SHUNT

In 10 cases the shunt blocked during the early postoperative period. In all further bleeding from varices occurred, usually within three days postoperatively. This resulted in four early postoperative deaths and two deaths at 22 and 33 months after the operation. The state of the portal vein at operation was reviewed in these patients whose shunt blocked and the condition of the vessel was assessed from preoperative radiographs. In one case calcification was present in the wall of the vein. In another portal vein thrombosis at the porta hepatis was shown to be the cause of portal hypertension. In a third case mural thrombosis was diagnosed radiographically and later proved at operation. Mural thrombosis, however, was diagnosed in one case and portal vein thrombosis at the porta hepatis in one case in the group with patent shunts. Analysis of the available data on portal vein diameter (Fig. 4) in cases with a patent shunt and those with a blocked shunt shows no significant difference at the 5% level between the two groups, though there is a trend suggesting that the shunt is more likely to block if the portal vein is 1·5 cm or less. No correlation was found between shunt block and immediate preoperative blood-clotting indices, intrasplenic pressure, mean age, or frequency of previous abdominal surgery anatomically adjacent to or remote from the portal vein.

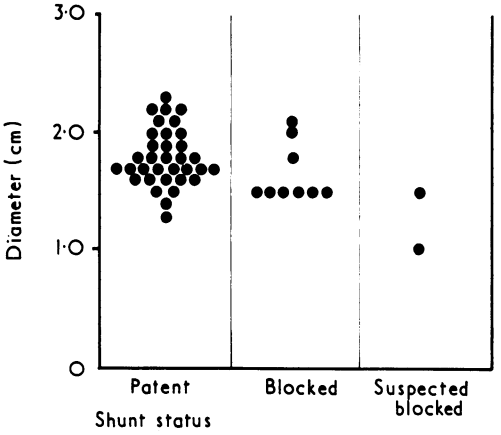

FIG. 4—Relation of preoperative portal vein diameter to the state of the shunt postoperatively.

A total of 17 patients bled from the upper gastrointestinal tract in the early postoperative period. Ten of these were proved to have a blocked shunt, and in another this was suspected but not proved. In six further cases the bleeds were small (consisting usually of one episode of haematemesis or melaena) and not massive or persistent as in most cases of blocked shunt, the shunt was proved to be patent, and haemorrhage was probably due to acute gastric erosions.

Late postoperative bleeding in all of six cases was due to proved blocked shunt. There were no instances of late bleeding due to peptic ulceration.

PORTAL-SYSTEMIC ENCEPHALOPATHY

Severe chronic portal-systemic encephalopathy was the most serious late complication of the operation (Table VIII) and was seen in 19 out of 50 (38%) patients who survived with a patent shunt. Most patients affected developed signs of severe involvement within 12 months of surgery. One-third of the survivors with patent shunts were seriously disabled by encephalopathy at one year, and over 40% after more than five years.

TABLE VIII—*Post-shunt Encephalopathy. Late Postoperative Severe Chronic Portal-Systemic Encephalopathy in 50 Survivors with Patent Shunts*

Aetiology	No. of Cases	Patients with Encephalopathy	
		No.	%
Cryptogenic cirrhosis	28	11	39
Alcoholic cirrhosis	9	5	56
Biliary cirrhosis	4	0	0
Active chronic hepatitis with cirrhosis	5	3	60
Portal vein block	1	0	0
Schistosomal cirrhosis	1	0	0
Haemochromatosis	1	0	0
Sarcoidosis	1	0	0
Total	50	19	38

The incidence in patients with cryptogenic cirrhosis (39%) was similar to the overall percentage (38%); there were no cases in those with biliary cirrhosis (4), portal vein block (1), schistosomiasis (1), haemochromatosis (1), or sarcoidosis (1). Five out of nine alcoholics (56%) and three out of five patients suffering from cirrhosis with active chronic hepatitis (60%) developed severe encephalopathy. In six patients with alcoholic liver disease substantiated information on present drinking habits was available—of three who stopped drinking none had severe encephalopathy; of three who continued drinking all had severe disabling encephalopathy.

Thirteen patients with patent shunt developed acute hepatic encephalopathy while still in hospital after surgery—eight developed severe chronic encephalopathy and two were followed for less than 12 months. Of the remaining three who did not

develop acute hepatic encephalopathy two were alcoholics who had stopped drinking and the third had schistosomiasis.

In eight cases where there was a history of transient encephalopathy preoperatively after bleeding, four out of six who survived had severe chronic encephalopathy, and the two patients who survived without severe encephalopathy were alcoholics who stopped drinking. The presence of ascites or jaundice preoperatively did not significantly affect the incidence of postoperative encephalopathy. Out of 21 cases where transient ascites had been detected before the shunt operation 9 (41%) developed severe encephalopathy (compared with 38% in the whole series of 50 survivors with patent shunt), and out of 13 cases where transient hepatocellular jaundice had been present before the operation 6 (46%) developed severe encephalopathy (compared with 41% in the whole series of 46 "non-biliary" survivors with a patent shunt).

Out of 23 cases where there had been no preoperative jaundice, ascites, portal-systemic encephalopathy, or hypoalbuminaemia 9 (39%) developed severe encephalopathy. The frequency of postoperative encephalopathy in those over 40 years was clearly greater, occurring in 50% compared with 26% in those under 40. Four of the six patients who survived after blockage of the shunt were disabled by encephalopathy, and one of those unaffected was not cirrhotic. In seven patients diabetes mellitus was diagnosed before the shunt operation; of the six survivors four developed severe encephalopathy.

Other neurological and psychiatric complications, not due to alcoholism, were seen in eight patients in association with periodic stupor. Five had persistent pyramidal tract signs with extensor plantar responses; in two of these there was an associated mild Parkinsonian facies and tremor and in one grand-mal epilepsy. Two patients had persistent signs of cerebellar disease and two showed persistent psychotic behaviour.

In addition to the 19 cases of severe encephalopathy eight survivors with patent shunts showed evidence of "mild" encephalopathy, such as flapping tremor or inverted sleep rhythm under stress, but all eight were fully controlled with protein restriction and neomycin therapy.

OTHER COMPLICATIONS

Ascites was not a troublesome complication and was easily controlled with moderate salt restriction and occasionally a diuretic taken by mouth. Twelve (24%) of the 50 survivors with a patent shunt developed ascites, and most of them (67%) had a previous history of this complication. Only 38% of those with a history of ascites before the shunt operation developed ascites afterwards, and 4 (14%) of those who had never had ascites developed it after the operation. Four of the six survivors with a blocked shunt developed ascites postoperatively. Ascites was common and gross in cases of early postoperative death whether from liver failure or from bleeding varices due to a blocked shunt.

Ankle oedema was common but was never gross and was usually transient. Survivors with patent and blocked shunts were similarly affected—23 out of 50 in the former and 3 out of 6 in the latter.

Four patients (8%) developed a symptomatic chronic duodenal ulcer after a successful shunt operation, and one of these had a chronic gastric ulcer. Six patients had proved chronic duodenal ulcer before surgery though none seemed to be aggravated by construction of the shunt.

There were five instances of haemosiderosis, five of persistent unconjugated hyperbilirubinaemia, and two of high-output cardiac failure.

Discussion

In portacaval shunt surgery the operation is a success when the patient survives with a patent shunt free of subsequent haemorrhages and severe encephalopathy. By these criteria success was achieved in 31 (48%) cases in this series.

In this study special attention was given to the preoperative state in an attempt to define those patients likely to suffer the three major modes of failure—namely, early postoperative death, shunt block, and severe portal-systemic encephalopathy.

An early postoperative mortality of 12·5% is slightly higher than the 6-10% in other individual series of elective end-to-side portacaval shunts,[6-10] though Grace *et al.*[11] in their review of 154 papers and 306 shunts found an average mortality of 18·3%. Bleeding from varices due to a blocked shunt was directly responsible for at least half of the postoperative mortality in the present series; three patients died from liver failure and only one of these appeared an excellent candidate to withstand surgery. A five-year survival of 65% compares favourably with other reported series,[7 9 10-13] where survival after five years ranged from 20% to 70%. Inclusion of poor-risk alcoholics, where operative mortality approaches 40%,[7] may account for the wide variation.

In this series long-term survival was better in those aged 40 years and under, though clearly the exception was the group with cirrhosis and active chronic hepatitis, where all but one were under 30 (mean age 22·6 years), but only two survived three years. Active chronic hepatitis is usually a relentlessly progressive disease and the poor prognosis may be made even worse by the shunting of portal blood away from the liver. A preoperative history of hepatocellular jaundice was associated with lower survival rates. Survival was the same whether ascites had been present or not. The small group who had had previous encephalopathy seemed to survive as long as the rest.

Blockage of the shunt not only fails to prevent variceal bleeding but also carries a high mortality. A blocked shunt rate of 16% is higher than usually reported,[10 11 14] and this is probably due to a policy of performing postoperative splenic venography or venous-phase abdominal arteriography whenever blockage is suspected because of recurrence of bleeding or failure of splenomegaly or variceal formations to diminish in size.

Reports of blocked portacaval shunts are sparse and there are no similar correlations between preoperative portal vein diameter and subsequent patency of the shunt. The results in this series suggest that a portal vein of a diameter (as seen on venography) of 1·5 cm or less may be more likely to block after anastomosis. Leger *et al.*[15] also noted that patients with calcification or thrombosis in the portal vein are poor candidates for shunt because of their tendency to block postoperatively.

Chronic portal-systemic encephalopathy was the major late complication seen, and the incidence of 38% of survivors with a patent shunt is similar to the 33% quoted by Read *et al.*[16] in a comparable series. Lower rates quoted by North American authors[7 17] possibly reflect the different cirrhotic population in the United States, where alcoholic disease predominates—abstinence from alcohol is known to decrease the chance of developing encephalopathy; this was apparent in this series in the three patients with alcoholic cirrhosis who stopped drinking and in whom encephalopathy did not develop.

A history of previous encephalopathy even under stress of gastrointestinal haemorrhage was the most reliable indication that severe encephalopathy would follow the shunt operation. An increased susceptibility with ageing to disabling encephalopathy after the construction of a shunt is well known[16] and is again seen in this series. Diabetics similarly displayed an increased rate of severe encephalopathy, occurring in four out of six diabetic survivors. These findings concur with those of Voorhees *et al.*[17] who noted a twofold increase of encephalopathy in their diabetic patients. This increased susceptibility probably reflects the cerebral atherosclerosis expected in older age groups and diabetics. Encephalopathy was not seen in any of four cases with biliary cirrhosis, where hepatocellular function is relatively well maintained, or in three cases of non-cirrhotic portal hypertension where hepatocellular function was normal or only

minimally impaired. No correlation was found between postoperative encephalopathy and a preoperative history of hepatocellular jaundice or ascites. Though there may be no preoperative encephalopathy, jaundice, ascites, or hypoalbuminaemia encephalopathy may still develop, and the frequency in these cases was similar to that of the whole series. There is a need for an accurate method of predicting preoperatively those likely to develop encephalopathy after shunting. Read *et al.*[18] suggested that frequency analysis of the E.E.G. may be the method of choice in this regard, and, in addition, may detect subclinical encephalopathy postoperatively when appropriate therapy can prevent clinical deterioration and possibly delay permanent brain damage.

End-to-side portacaval shunt is less effective than the side-to-side procedure in relieving a hepatic venous outflow block,[19] and leaking hepatic lymph will remain a potential source of ascitic fluid; nevertheless, postoperative ascites was not a serious problem in survivors, partly due to selection of cases, since persistent or gross ascites excluded patients from surgery.

Though a true increase in the incidence of peptic ulceration after shunt operations has not yet been conclusively shown in a controlled trial 8% of survivors with a patent shunt developed a chronic peptic ulcer, an incidence similar to the 8·9% quoted by Grace *et al.*[11] Aggravation of existing chronic duodenal ulcers was not seen after the operation.

Results in the present series are consistent with the view that the candidate for elective end-to-side portacaval shunt should be young (preferably below 40 years of age) and should not have had hepatocellular jaundice or portal-systemic encephalopathy even after bleeding. If the underlying cause of the portal hypertension is non-cirrhotic or biliary in origin this is an added advantage. The presence of preoperative transient mild or moderate ascites does not seem adversely to influence the outcome. Patients with active chronic hepatitis seem to do poorly after portacaval shunt. In alcoholic liver disease abstinence from ethanol lessens the chance of encephalopathy after a shunt operation. Diabetics frequently suffer disabling encephalopathy postoperatively. A patent shunt is more likely if the portal vein is not calcified or thrombosed and may be more likely if the portal vein diameter, as measured on venography, is greater than 1·5 cm.

We wish to thank the Ingram Trust for financial support.

References

1 Read, A. E., *British Medical Journal*, 1968, **1**, 427.
2 Hislop *et al.*, *Lancet*, 1966. **1**, 945.
3 Sherlock, S., *British Journal of Surgery*, 1964, **51**, 746.
4 Taylor, F. W., and Jontz, J. G., *Archives of Surgery*, 1959, **78**, 786.
5 Garceau, A. J., and Chalmers, T. C., *New England Journal of Medicine*, 1963, **268**, 469.
6 Brown, G. J. A., and Walker, R. M., *Lancet*, 1967, **2**, 854.
7 Panke, W. F., Rousselot, L. M., and Burchell, A. R., *Annals of Surgery*, 1968, **168**, 957.
8 Resnick, R. H., *et al.*, *Annals of Internal Medicine*, 1969, **70**, 675.
9 McDermott, W. V., jun., Palazzi, H., Nardi, G. L., and Mondet, A., *New England Journal of Medicine*, 1961, **264**, 419.
10 Walker, R. M., Shaldon, C., and Vowles, K. D. J., *Lancet*, 1961, **2**, 727.
11 Grace, N. D., Meunch, H., and Chalmers, T. C., *Gastroenterology*, 1966, **50**, 945.
12 Zeegan, R., Stansfeld, A. G., Dawson, A. M., and Hunt, A. H., *Gut*, 1970, **11**, 610.
13 Reynolds, T. B., *Annals of the New York Academy of Sciences*, 1970, **170**, 1, 379.
14 Rousselot, L. M., Panke, W. F., Bono, R. F., and Moreno, A. J., *American Journal of Medicine*, 1963, **34**, 297.
15 Leger, L., Lenriot, J.-P., Dentan, T., and Lemaigre, G., *Journal de Chirurgie*, 1970, **99**, 5.
16 Read, A. E., Laidlaw, J., and Sherlock, S., *Lancet*, 1961, **1**, 961.
17 Voorhees, A. B., jun., and Price, J. B., jun. *Annals of the New York Academy of Sciences*, 1970, **170**, 259.
18 Read, A. E., McCarthy, C. F., Ajdukiewicz, A. B., and Brown, G. J. A., *Lancet*, 1968, **2**, 999.
19 Reynolds, T. B., Hudson, N. M., Mikkelsen, W. P., Turrill, F. L., and Redeker, A. G., *New England Journal of Medicine*, 1966, **274**, 706.

Results in 1,000 Cases of Therapeutic Abortion Managed by Vacuum Aspiration

K. C. LOUNG, A. E. R. BUCKLE, MARY M. ANDERSON

British Medical Journal, 1971, **4**, 477-479

Summary

A prospective study of 1,000 cases of termination of pregnancy by vacuum aspiration is presented and the safety of the method emphasized. By setting up special clinics and an additional weekly operating session, the maximum delay from a request to appointment is 5 days, and, if operation is advised, from appointment to operation a further 7 days.

Introduction

We have previously reported on the use of vacuum aspiration in therapeutic abortion[1] and present now the results from a consecutive series of 1,000 cases so managed. Since our previous report much interest has been aroused in the use of intravenous prostaglandin E_2,[2 3] intrauterine prostaglandins E_2 and $F_2\alpha$,[4] and prostaglandins E_2 and $F_2\alpha$ in the form of vaginal tablets[5] for therapeutic abortion. Clinical trials of these substances are currently limited to some four centres in the United Kingdom, and while the outcome of the work is awaited with interest most gynaecologists will for the present have to rely on conventional methods—namely, curettage, vacuum aspiration, and, for more advanced cases, intra-amniotic saline, intrauterine paste, or, more rarely, hysterotomy.

The technique in current use differs slightly from that which we previously described. General anaesthesia has in most cases been replaced by a regimen whereby after premedication with Omnopon 10 mg and scopolamine 0·2 mg the patient is treated in the operating theatre with intravenous diazepam 10 mg, intravenous pentazocine 30 mg, and methohexitone sodium 100 mg together with 0·5 mg of ergometrine. If the patient is disturbed by the operation further methohexitone may be given, though none has been necessary to date. In 20 cases where the pregnancy had not advanced beyond eight weeks by dates we used the Rocket vacuum aspiration catheter* (6 mm diameter) under intramuscular diazepam 10 mg and intravenous

Department of Obstetrics and Gynaecology, Lewisham General Hospital, London S.E.13

K. C. LOUNG, M.R.C.O.G., Registrar
A. E. R. BUCKLE, F.R.C.S., F.R.C.O.G., Consultant
MARY M. ANDERSON, M.R.C.O.G., Consultant

*Obtainable from Rocket of London, Ltd., Imperial Way, Watford, Herts.