which the consulting urologist deemed due to use of an indwelling catheter rather than to localized arterial insufficiency. This condition responded to conservative treatment. Sigmoidoscopy on the first and second postoperative days revealed no abnormality of the rectosigmoid colon. Normal bowel function promptly resumed. Popliteal and ankle pulses remained equal and strong. The patient's main complaint was of buttock and thigh claudication, which severely limited ambulation during the first three postoperative weeks. He could walk no more than 30 feet at a time. The buttock and thigh muscles atrophied but no skin changes occurred. Eight weeks after operation the patient could slowly walk one block, his now small muscle groups were firm and muscle strength and ability to walk improved daily. He slowly regained a 20-pound loss of weight.

When interviewed two years after the operation the patient had no complaints other than bilateral buttock claudication when he walked fast for two blocks or walked rapidly up hill. His general health was good. Bilateral popliteal aneurysms remained but circulation to the feet was excellent.

DISCUSSION

Multiple aneurysms in continuity can be safely resected but the morbidity may be great. Some observers estimate that necrosis of the rectum will occur in 10 per cent of patients who have simultaneous ligation of patent inferior mesenteric and hypogastric arteries. I doubt the incidence is so high but I have observed the condition following such operation. In the case here reported the buttock and thigh ischemia would have been less had the deep femoral circulations been maintained, and this possibly could have been done by running branch grafts from the femoral segments to the respective profunda arteries. It is not likely that the diseased hypogastric arteries could have been salvaged.

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Severe Salicylism and Acute Pancreatitis

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In epidemiologic surveys on accidental poisoning, the ingestion of aspirin accounts for 16 to 39 per cent of the cases,[3,14,16] yet mortality due to salicylism is low.[3,14,16] The evaluation of severity and ultimate prognosis by laboratory tests in each case was impossible until Done's report[4] of S_0* levels in salicylism provided clinicians with a direct means of evaluating severity.

In the patient here to be reported upon, the history of ingestion of acetyl salicylic acid was obscure at first. Clinical and laboratory findings suggested acute pancreatitis and further studies (confirmed by careful history) established aspirin intoxication. Relationship between salicylate poisoning and pancreatitis is speculative.

REPORT OF A CASE

A 2-year-old boy awoke from a deep sleep on January 13, 1962, in the early morning hours and complained of some lower abdominal pain. At that time his mother noted that he was breathing rapidly but she was not overly concerned. Over the next nine hours he vomited about five times. The pain continued and he was brought to the Emergency Room at San Francisco General Hospital. Although his mother denied possible ingestion of aspirin, the child appeared acutely ill and was admitted to the hospital.

On entry, the temperature was 101° F, the pulse rate 160, respirations 70 per minute, blood pressure 100/60 mm of mercury, weight 9.5 kg, and the body surface area 0.45 sq meters. The child was semi-stuporous but responded to painful stimuli. There were signs of dehydration—poor skin turgor, sunken eyes and lack of oral secretions. The abdomen was diffusely tender with vague localization from one examination to the next. A tentative diagnosis of salicylism was made. Reaction to a ferric chloride test on the urine was positive but was misinterpreted by an inexperienced observer.

Packed red blood cell volume was 39 per cent. Leukocytes numbered 23,000 per cu mm with 73 per cent segmented polymorphonuclear leukocytes, 22 per cent lymphocytes and 6 per cent monocytes. Specific gravity of the urine was 1.008, pH 5.3, reaction for acetonuria 3+, reducing substance and protein negative, and urinary sediment normal. Glucose content of the blood was 50 mg per 100 ml,

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*S_0—theoretical (extrapolated) salicylate level at zero time.

TABLE 1.—*Laboratory Data*

	Admission	Hours Later 12	27	40
SERUM:				
Sodium (mEq per liter)	140.0	137.0	148.0	.....
Potassium (mEq per liter)	5.4	3.8	5.2	.....
Carbon dioxide (mEq per liter)	11.0	9.0	15.0	.....
PCO_2 (mM/L)*	.....	17.5	17.5	.....
pH	.....	7.36	7.56	.....
Amylase (units)	1560.0	.....	.....	.....
Glucose (mg per 100 ml)	50.0	.....	.....	.....
Salicylate (mg per 100 ml)	.....	58.0	41.0	13.0
DIALYSATE:				
Salicylate removed (mg)	.....	.....	390.0	110.0
URINE:				
pH	5.3	5.5	.....	6.0
Acetone	+ + +	.....	.....	.....
Reducing substance	0	.....	.....	.....

*Partial pressure carbon dioxide, millimols per liter.

TABLE 2.—*Data on Therapy of Patient with Salicylism*

	Admission	Hours Later 12	27	37
INTRAVENOUS:				
Water (cc)	2,000	800	900	.....
Sodium (mEq)	120	17	30	.....
Cl (mEq)	37	32	40	.....
Potassium (mEq)	.....	15	10	.....
Glucose (gm)	100	40	45	.....
Lactate (mEq)	83	.....	.....	.....
DIALYSIS:				
Peridial®*	.....	11 bottles†		.....
6 mEq KCl	.....			
1.5 per cent Dextrose	.....			
Ringer's lactate*	.....	.....	6 bottles‡	
1.5 per cent Dextrose	.....	.....		

*Contents:

	cc H_2O	mEq Na	K	Ca	Mg	Cl	HCO_3	Osmolarity
Peridial®	1000	140	....	4.0	1.5	102.5	43	371.0
Ringer's lactate	1000	130	4.0	3.0	....	109.0	28	355.5

†1,000 ml each (from 12th to 27th hours).
‡27 to 37 hours.

sodium 140 mEq per liter, potassium 5.4 mEq per liter, and carbon dioxide 11 mEq per liter. The cerebrospinal fluid protein content was 46 mg per 100 ml, sugar 71 mg per ml and white blood cell count 3 per cu mm. The fluid was negative for Gram staining organisms. No abnormality was noted in examination of aspirated abdominal fluid.

Careful review of the clinical findings suggested acute pancreatitis. Serum amylase shortly after admission was 1,560 units on duplicate specimens. During the next 12 hours the patient received, intravenously, 2,000 ml of water, 120 mEq of sodium, 37 mEq of chloride, 83 mEq of lactate and 100 gm of glucose. Dehydration disappeared, respirations continued at 60 per minute, and the child remained semi-stuporous. A diagnosis of salicylism was considered in spite of the previous ferric chloride test that had been reported as negative.

In exploring the patient's environment, it was discovered that his father and siblings cared for him while his mother worked from 3 p.m. until midnight. Further laboratory tests confirmed the suspicion of salicylism; reactions to ferric chloride tests on serum and urine specimens were positive. The serum salicylate level 12 hours after admission was 58 mg per ml—above the range rated as "severe" on Done's chart (Chart 1). Broken adult aspirin tablets were found later in the child's play area. A careful review suggested that he had ingested a quantity of aspirin 30 hours before the salicylate determination was carried out. Pertinent laboratory and therapeutic data are presented in Tables 1 and 2.

Twelve hours after the patient was admitted, the urine pH was 5.5, after generous alkali therapy. Peritoneal dialysis was instituted because of the patient's clinical state, the significance of the salicylate level at 30 hours (Chart 1), failure to respond to adequate alkali therapy and the risk attendant upon further alkali therapy.

During the next 15 hours, 390 mg of salicylate was removed by peritoneal dialysis, using 11 (1,000 ml) bottles of Peridial® with 6 mEq of potassium chloride and 15 gm of dextrose added to each bottle. During this time serum pH rose to 7.56, due in part to the amount of bicarbonate in Peridial® (Table 1).

Because of this alkalosis, the dialysis fluid was changed to lactated Ringer's solution with 15 gm dextrose added to each 1,000-ml bottle. With the use of six bottles over the next 10-hour period, 110 mg of salicylate was removed. Tachypnea disappeared and the child seemed alert. Urine pH was 6.0 and serum salicylate decreased to 13 mg per liter. Peritoneal dialysis and intravenous therapy were then discontinued and complete recovery ensued quickly.

DISCUSSION

Abdominal pain, fever, increased leukocyte content in the blood with a shift to the left in cell differential, and an amylase content of 1,500 units suggest that this child had pancreatitis in association with salicylism. It cannot be determined from the available data if toxic amounts of acetyl salicylic acid were responsible for the pancreatitis. Search of the literature did not reveal any previously reported association between salicylism and pancreatitis. [10,12,17,25] Although it is known that gastric ulceration provokes excessive amylase elaboration, the possible production of gastric ulceration by acetyl salicylic acid is controversial. Some reports indicate that overdosage or prolonged use of

acetyl salicylic acid will produce gastric ulceration with hematemesis and melena,[5,13] although others have failed to confirm these findings.[21]

An increase in serum amylase is seen in a variety of other extra-pancreatic conditions, among which are perforated peptic ulcer,[7] parotitis, abscess or obstruction of salivary ducts, carcinoma at the ampulla of Vater, acute alcoholism, biliary dyskinesia from opiate administration,[1,19,24] extrahepatic obstruction, cholecystitis, choledocholithiasis, ruptured ectopic pregnancy, high intestinal obstruction,[7] myocardial infarction, peritonitis and renal insufficiency with uremia.* The amylase test is quite specific and normal values in the presence of acute pancreatitis are uncommon.[12] Although elevated levels have been reported in the conditions enumerated, a concentration in excess of 500[22] to 1000[11] units is almost diagnostic of pancreatic disease.

Excretion of salicylate is directly proportional to urine pH. Alkali therapy generally alters the urine and serum acidity in salicylism.[20,26] Alkalinization of the urine with a dose of 3.5 mEq of sodium lactate per kilogram of body weight,[20] to a total dose of less than 54 mEq of sodium bicarbonate,[26] usually brings about systemic alkalosis. Although 8 mEq of sodium lacate per kilogram was given to this patient before the diagnosis was established, this amount did not produce alkalosis and did not alkalinize the urine.

The efficacy, efficiency and safety of peritoneal dialysis in severe salicylism has been well established.[9,15] In our patient, assuming an extracellular volume of 250 ml per kilogram[2] and uniform dispersion of extracellular salicylate, 145 mg of salicylate was circulating in the extracellular compartment before dialysis was carried out. During a critical period of 25 hours, 500 mg of salicylate was removed by peritoneal dialysis, presumably most of it from the intracellular compartment. Etteldorf[9] showed in experimental animals that albumin in dialyzing fluid will continue to bind salicylates against a gradient. His results in seven human cases, in which albumin was added to the dialyzing fluid, were encouraging but effects were not comparable to those observed in animals. The use of a dialyzing solution rich in albumin might well have been effective in the present case, but this is a matter for speculation.

The high salicylate level plotted in Chart 1 at the time of admission correlated with the clinical status. According to Done,[4] fall of the salicylate level with time is a first order reaction. With peritoneal dialysis one might expect more rapid decline of salicylate levels. In the case here reported, 390 mg of salicylate was removed by peritoneal dialysis over a 15-hour period, and the blood serum salicylate level fell according to a first order reaction. Thirteen hours thereafter 110 mg of salicylate had been removed by dialysis, the serum level was 13 mg per 100 ml, moving it from the severe to the moderate zone on Chart 1.

*References Nos. 6, 12, 17, 18, 22, 23.

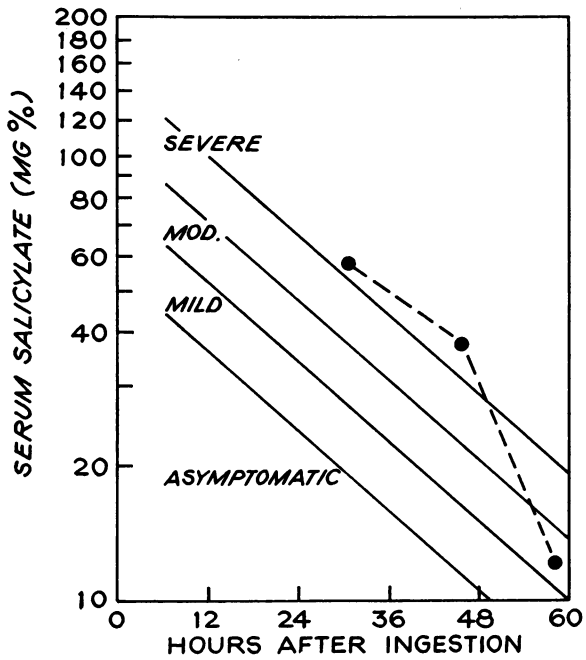

Chart 1.—Sodium salicylate levels plotted on Done's nomogram (with permission).

SUMMARY

A case of salicylism is presented in which there is evidence suggestive of acute pancreatitis. The possible relationship of these two entities is discussed.

The administration of 8 mEq of sodium lactate per kilogram of body weight, which failed to produce alkalosis or alkaline urine, was followed by peritoneal dialysis which effected removal of 500 mg of salicylate.

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