

DISCUSSION

DR. J. HERMAN MAHAFFEY: I enjoyed this paper very much and arise for a moment to mention some of our experiences at Houston a few years ago, in which we studied the serum amylase concentration in 51 patients with acute perforation of gastroduodenal ulcers. It is generally accepted that the elevated serum amylase that is seen following acute perforated peptic ulcer is due to absorption of the amylase from the peritoneum after it has spilled out through the perforation, and not as a result of secondary pancreatitis.

We were able in a small number of cases to correlate an elevated serum amylase with the size of the perforation, with the length of time from perforation of the ulcer until the amylase determination was performed—which was usually just preoperatively and before sedation was given—to correlate it with the amount of peritoneal spillage, a point which is frequently inadequately described when one tends to review the operating reports of a group of cases and, lastly, to the number of hours elapsing from the time the patient last ate until the perforation occurred. Those four findings we found influenced considerably the elevation of the serum amylase in acute perforating gastroduodenal ulcers. Generally we did not see an elevated serum amylase level within 12 hours of perforation of an ulcer, and a significant rise in the amylase did not occur until 18 to 24 hours after the ulcer had perforated.

Serious consideration should be given a patient seen 24 hours after the onset of symptoms, with an elevated serum amylase concentration but without demonstrable free air under the diaphragm. Since an elevation in serum amylase concentration may follow an acute gastroduodenal perforation, the finding can never be absolute in the differential diagnosis between acute pancreatitis and a perforated ulcer.

DR. JOHN M. HOWARD: It was my pleasure to read Dr. Coffey's paper ahead of time. It has been a pleasure to follow his work along these lines over the past years. We have studied perforated peptic ulcers in 94 patients, the diagnosis of perforated ulcer being proven at laparotomy. In addition, 75 patients with acute cholecystitis proven at laparotomy had serum amylase determinations prior to operation. In general, about one out of six to one out of eight in these groups will have a significant elevation in serum amylase concentration although at operation pancreatitis may well not be noted. I would like to say that in that group there has not been a fatality due to an error in diagnosis, which brings us back to the point that the clinical examination is the final basis on which the diagnosis must rest.

There are in the literature several studies relative to the peritoneal fluid concentration of amylase. Suffice it to say that in approximately 30 patients with a proven perforation of a peptic ulcer, the

peritoneal fluid amylase was very high in several; so high that the concentration of amylase in the peritoneal fluid could not permit a diagnostic distinction. Two of those patients with high elevations had gastric perforations.

DR. CARRINGTON WILLIAM, JR.: Although there is the disadvantage of the possibility of masking other intraabdominal causes for acute abdominal pain, it has been found by the anesthesiologists in Richmond that the use of continuous epidural anesthesia was much easier to manage than the single paravertebral splanchnic block.

DR. RICHARD T. SHACKELFORD: I rise to discuss Dr. Coffey's paper, not because I have any special knowledge in that particular field but because recently, or not so recently (in 1954), I did have the advantage of seeing a psychiatric patient in a Veterans Hospital with pancreatic disease. He had been in the hospital some 25 years and I have no doubt that he will be there until the millennium. However, we were able to trace the course of his disease and its development of high serum amylase and serum lipase which I thought might of some interest to you.

(Slide) On February 24, 1954, the preoperative serum amylase is as shown here, 150 to 200. He had a cholecystectomy the following day but it was found at operation that he had a small adenoma of the pancreas. It was also excised by a wedge resection and the area was drained. The convalescence was not clinically unusual in any way and was as smooth as the usual cholecystectomy, but the serum amylase went up to above 500 on the fourth postoperative day and has remained there. At the same time the serum lipase continued to rise to equally high levels. These studies were made about every third day from March, 1954, for five months, at which time although the amylase and the lipase were beginning to come down we felt a mass for the first time. With the suspicion that he had a pancreatic cyst, on July 1, 1954, operation was performed and a pancreatic cyst was found and drained by cystgastrostomy. Immediately following that the serum amylase and lipase came down and remained at normal levels through the last determination in July, 1957, three years later. These determinations were made on divisions of the same specimen. One other thing, an x-ray in October, 1954, after a barium swallow, revealed no evidence of a residual cyst, and the gastroscopist was unable to find the stoma which may have healed over. The patient has remained well except for his psychiatric disturbance.

I do not know that we can draw any conclusions from a single case but I wanted to demonstrate one case where we knew the origin of the pancreatic cyst, saw the serum amylase and serum lipase rise during its development and saw the results following internal drainage of the cyst. Apparently as soon as it was drained both levels returned to normal.

MR. GUY BLACKBURN: In listening to this discussion I have the impression that there is no great difference of opinion about pancreatitis on the two sides of the ocean. I started, in our country, by thinking that diagnosticians were not very good at pancreatitis. I progressed to the stage of relying on the pathologists and soon realized that they too were not very good in diagnosing pancreatitis. But both these statements must be tempered by the humiliating thought that, surgically speaking, we as surgeons are not very good when it comes to the treatment of pancreatitis.

As to the determination of serum amylase we tend, I think, to use the Somogyi scale, and I have no experience of the micro method of Teller that is being advocated by Mackenzie and others. The Wohlgemuth method we found a little difficult to interpret and I thought I was finding the Somogyi method easier until one day a patient came in with what we thought was typical relapsing pancreatitis, because the diagnosis had first been made when the abdomen had inadvertently been opened before in a similar attack. When the patient had a second attack I had a scientifically minded house surgeon, who sent the patient's serum to the laboratory for serum amylase determination, and took the precaution of sending some of his own. The names were disguised, the patient was treated by splanchnic injection, and two more specimens of blood were taken about four to six hours afterward. On the first occasion his reading was some 100 units higher than the patient's and on the second occasion it was 200. If you know how to interpret that, I don't.

With regard to the relief of symptoms of pancreatitis by splanchnic block, I remember Dr. Ochsner coming to Guy's one day, where I was able to show him a patient with pancreatitis. He advocated a splanchnic block, which was carried out with relief. In subsequent cases, however, we were not uniformly successful in relieving the pain for any length of time.

The reasons are hard to seek but I have a feeling, and I do not speak disparagingly, because we are very fortunate in this respect, that what for want of a better term I must call the hard core of

tradition in the nursing profession is responsible for the discomfort that some patients experience after what appears to be a relieving form of injection. Nurses are always told that if a doctor turns a patient on his side and makes an injection into the spinal theca, the patient must be laid flat afterward: if he complains of a little pain in the back, the sympathetic and kindly nurse may even put a pillow under the back and, if there is one thing that will aggravate pancreatic pain as you all know very well, it is hyperextension of the lumbar spine.

Finally, I must say that my residents, would never pay me the compliment of asking me to give them a demonstration of splanchnic injection.

DR. ROBERT J. COFFEY (closing): I would like to thank those who were kind enough to discuss my paper. In response to Dr. Mahaffey's remarks we have been unable to correlate the time interval between perforation and repair of the perforated ulcer with elevation of the amylase. It may be that we were fortunate in seeing the majority of these cases before a lapse of 24 hours. The one fact with which we were clearly impressed in this study is that active secretion of the exocrine pancreas in the presence of impaired drainage from its ductal system consistently produces hyperamylasemia: on the other hand, necrosis of the pancreas may occur without an elevation of the serum amylase. We are currently studying another enzyme, desoxyribonuclease (DNase), that appears to be more specifically related to necrosis of the pancreas, a finding that may have some prognostic and diagnostic significance.

Dr. Howard has pointed out that the amylase level may be elevated after gastric resection. We have observed that after both the Bilioth I and Bilioth II types of procedure such elevations may persist for periods of two or three weeks. Such protracted elevations are usually related to leakage of the anastomosis or duodenal stump.

The case reported by Dr. Shackelford clearly demonstrates that a sustained elevation of the serum amylase should suggest development of a pseudocyst or peripancreatic collection.