DISCUSSION

DR. W. EMORY BURNETT: Dr. Harkins and Gentlemen: I would like to congratulate Dr. Egdahl on a beautiful piece of work and so well presented. I think this is a very interesting subject to some of us, probably to all.

I would like to present a little data which corroborates the possible absorptive factor from the peritoneum. Although the patient is not one of spontaneous primary pancreatitis, but is in the group of so-called postoperative pancreatitis, with which we have had moderate experience.

It appears that of the few who developed this syndrome after the last 500 gastric resections, about 60 per cent of those that we posted did not have true pancreatic disease, but they had the symptoms which cause one to make such a diagnosis, including fat necrosis, and a high level of amylase in the peritoneal fluid and blood serum. In these 60 per cent, then, the cause of the syndrome was apparently leakage of secretion from an injured accessory pancreatic duct and not true pancreatitis, although it is almost impossible to distinguish the two situations except by postmortem. This patient was one in which, on the day after gastrectomy, severe symptomatology developed, and aspiration of the peritoneal fluid as well as blood levels revealed high amylase content. Acting on the concept that absorption from the peritoneum might be the only cause for his illness, he was subjected to saline lavage of the peritoneum on three occasions in a period of 36 hours.

I would like you to note the happy result of removal of the reservoir and the decreasing amount of enzyme found before and after each irrigation, these three items on this slide.

The patient's clinical condition rapidly improved simultaneously. The blood levels, although a jump occurred here on the third day, were generally progressively downward along with his rapid improvement.

Random tests on these subsequent days showed continued decrease in the peritoneal enzyme content, and the next slide indicates his graphic sheet during this time with a satisfactory decline in temperature and pulse rate to complete recovery. (Slide)

It is possible that this item of absorption from the peritoneum is a common denominator in some, if not in many, patients with pancreatitis, and that such absorption from the peritoneum may be a big factor in the severity of the illness. In the so-called postoperative type, it may be the basic mechanism. With these remarks, I conclude the discussion. Thank you.

DR. RICHARD EGDAHL (closing): I would like to thank Dr. Burnett for his kind remarks. I would just like to emphasize one thing: duct rupture probably is not necessary in the pathogenesis of pancreatitis.

We have produced bile pancreatitis by the use of pressures around 20 mm. of mercury and have confirmed that duct rupture does not occur by the use of India Ink, which passes out into the interstitium without break in duct continuity through clefts between the acinar cells.

Credit Due

In the May issue of *Annals of Surgery* in the article, Experiences with New Types of Aortic Valvular Protheses, a co-author's name was listed incorrectly. The name should be, *Leopoldo Diaz de Villegas* and not, *Paulo Diaz Vilkgas.*