EDITORIAL

Pancreatitis — An Unsolved Problem

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For many years attempts to unravel the pathophysiology of inflammatory disorders of the pancreas as well as efforts at treatment have centered on the exocrine duct system. To many students of the disease and particularly to those with clinical responsibilities it has, gradually become apparent that this approach has for the most part, been unrewarding and that a different perspective is required.

An attempt to determine why patients who had undergone operations designed to decompress the exocrine duct system continued to have symptoms led to observations which link the lymphatic system with both acute and chronic forms of pancreatitis. Representing what is sometimes referred to as "old fashioned descriptive physiology" these recent studies have nevertheless clarified a previously unrecognized functional relationship between the exocrine system and pancreatic lymphatics. Elsewhere in this issue *Papp,* one of the early workers in this field, and co-authors describe the results of experiments which were undertaken to settle some disputed points regarding this relationship. With respect to this and similar studies it may be important to recall that although pancreatic lymphatics are anatomically closely associated with the exocrine system there is no direct structural communication between the two. As pancreatic enzymes are normally conveyed to the thoracic duct in pancreatic lymph and as exocrine ducts are permeable to particulate dye at low pressures, it seems safe to assume that protein molecules normally leak from the confines of the duct system into interstitial fluid and ultimately are collected in pancreatic lymphatics. This conforms to a traditional view of lymphatic function except that protein molecules derive from pancreatic juice rather than from plasma. In man stimulation of pancreatic secretion after a prolonged fast could lead to sudden overdistention of exocrine ducts and displacement of abnormal amounts of osmotically active enzyme-protein into interstitial fluid and in turn into lymphatics. In such an event transport capacity of pancreatic lymphatics and the thoracic duct could become a critial factor. Thus considered, the edema and perilymphatic fat necrosis which follow experimental ductal ligation have a clinical counterpart. Some of the clinical implications from the study by *Papp* and from earlier observations in patients with chronic pancreatitis are not altogether encouraging in that impaired lymphatic transport of extravasated protein in the fibrosed gland resembles that which develops in fibrosed skin of the lower leg following lymphedema.

Clinicians will not be satisfied with the present level of knowledge regarding mechanisms involved in pancreatitis. Eagerness to bring new information to practical application at the bedside or operating table, misdirected at times, can also result in additional knowledge and ultimately in information that is useful.

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