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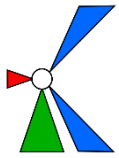

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of the disease and then rapidly return to normal. The early increase of pan-PLA2 values probably reflects the time of destruction of pancreatic tissue. Group II PLA2s occur in synovial fluid, platelets, placenta, and inflammatory cells and exudate. Besides acute pancreatitis, increased catalytic activity of PLA2 in serum is associated with many diseases involving infection, tissue destruction, and inflammation including septic shock, rheumatoid arthritis, and multiple injuries. Ourselves and others have recently found, by new immunoassays, increased concentrations of syn-PLA2 in serum samples from patients with septic fever and infections. The blood culture was positive in three patients in the present study. In these patients the concentration of syn-PLA2 in serum was very high (>1000 µg/l) at the time of the positive cultures. The cellular source of group II PLA2 found in serum samples from patients with acute pancreatitis and other inflammatory diseases remains unknown. It was proposed recently that group II PLA2 might represent an acute phase reactant. Hepatoma cells in culture secrete PLA2 into the culture medium when stimulated by interleukin 6. We have recently found by our immunoassay considerable amounts of syn-PLA2 in synovial fluid and seminal plasma, and detected the enzyme by immunohistochemistry in cartilage and Paneth cells of the intestinal mucosa (Nevalainen T J and Haapanen??. Unpublished data). The determination of immunoreactive synovial-type PLA2 as described in this paper might be helpful in the early assessment of the severity of acute pancreatitis. The therapeutic implications of the present findings are to be established in acute pancreatitis. PLA2 has been considered earlier to act mainly as a harmful agent in the pathology of various inflammatory diseases including acute pancreatitis. Pancreatic PLA2 seems to be non-toxic to pancreatic acinar cells, however, although toxic effects have also been found. Human group II PLA2 purified from cartilage seems non-toxic to cells in culture (Nevalainen T J and coworkers. Unpublished results). As an acute phase reactant, the role of group II PLA2 in infections and inflammatory diseases might be related to the host's defence against invading micro-organisms and tissue destruction. We conclude that the catalytic activity of PLA2 in serum in acute pancreatitis is due to the presence of a synovial type group II PLA2. The PLA2 activity is not dependent on the concentration of pancreatic group I PLA2 in serum. Eradicating Helicobacter pylori infection lowers gastrin mediated acid secretion by two thirds in patients with duodenal ulcer Abstract Helicobacter pylori (H pylori) raises serum gastrin but it is unclear whether this stimulates increased acid secretion. Gastrin mediated acid secretion and plasma gastrin after the intravenous infusion of gastrin releasing peptide was studied in **nine H pylori negative** and **nine H pylori positive healthy volunteers**, and in **11 duodenal ulcer patients**.

**Nine of the last group**

were re-examined one one after **eradication of H pylori**. The median acid output (mmol/h) to gastrin releasing peptide (40 pmol/ kg/h) in **the H pylori positive healthy volunteers** was 15.1 (range 3.3-38.3), which was three times that of **the H pylori negative healthy volunteers** (median=5.5, range 1.0-9.0) ($p<0.02$). The

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| | median acid output in **the duodenal ulcer patients with H pylori** was 37 (range 8.5-57), which was >six times that of the **H pylori negative healthy volunteers**. **Eradication of H pylori** in **the duodenal ulcer patients** lowered their acid secretion by a median of 66% (range 30%-80%) ($p<0.01$) and to values equivalent to the H pylori positive healthy volunteers. The pepsin output in response to gastrin releasing peptide followed the same pattern as the acid output. The median plasma gastrin concentrations during gastrin releasing peptide were similar in the **H pylori positive duodenal ulcer patients** (150 ng/l, range 95-400) and **H pylori positive healthy volunteers** (129 ng/l, range 23-420) and both were appreciably higher than **H pylori negative healthy volunteers** (60 ng/l, range 28-135) ($p<0.005$ for each). **Eradication of TH pylori** lowered the plasma gastrin in **the duodenal ulcer patients** to values equivalent to the H pylori negative healthy volunteers. These findings show a threefold increase in acid secretion in H pylori positive healthy volunteers that is explained by H pylori induced hypergastrinaemia and a sixfold increase in acid secretion in the duodenal ulcer patients that is explained by the combination of H pylori induced hypergastrinaemia and an exaggerated acid response to stimulation by gastrin. Eradicating H pylori lowers gastrin mediate acid secretion by 66% in duodenal ulcer patients as a result of the resolution of the hypergastrinaemia. Increased gastrin mediated acid secretion seems to be the key factor in the pathophysiology of duodenal ulceration and explains the role of H pylori infection in the disorder. Helicobacter pylori (H pylori) infection is now recognised to be the main acquired factor in the pathogenesis of duodenal ulcer duodenal ulcer disease. It is present in >95% of duodenal ulcer patients and numerous studies have shown that eradicating the infection dramatically lowers the ulcer relapse rate. The mechanism by which this infection, which predominantly affects the antral mucosa, predisposes to ulceration of the duodenum is unknown. Also, the reason why only a small proportion of subjects with this common infection develop duodenal ulceration is unclear. We and others have shown that both duodenal ulcer patients and healthy volunteers with H pylori have increased basal and meal stimulated gastrin concentrations that fall after eradication of the infection. Though gastrin is recognised to be the main mediator of meal stimulated acid secretion, the effect of H pylori on acid secretion remains unclear. A major |