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References


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Letters to the Editor

Hypothermia and pancreatitis

Dr Foulis' recent study relating hypothermia with the morphology of the associated acute pancreatitis is of great interest. This work, together with Dr Foulis' previous comprehensive survey relating the histological pattern of pancreatitis with various clinical diagnoses may shed some light on the pathogenesis of pancreatitis.

We would like to report certain findings of our own and consider their relevance to Dr Foulis' studies.

We have shown that at temperatures below 37°C the inhibitory action of prostacyclin (PGI₂) on in vitro platelet aggregation is diminished. Furthermore, the in vitro synthesis of PGI₂ by vascular endothelium is decreased at low temperatures. We have also suggested that at low temperatures the vasodilatory action of PGI₂ is diminished. The raised serum non-esterified fatty acid (NEFA) concentrations reported in hypothermic patients may be of relevance since we have shown that high NEFA concentrations inhibit vascular PGI₂ synthesis and accelerate PGI₂ decay in albumin solutions.

Hypothermia may thus impair synthesis, increase the rate of decay and diminish the potency of PGI₂. These changes would result in platelet activation and predisposition to thrombosis and ischaemia. Abnormal platelet behaviour has been demonstrated in an animal study where thrombocytopenia occurred during induced hypothermia. An increased incidence of thrombotic phenomena in patients with hypothermia is evident from Dr Foulis' own observations and from the literature he cites.

Ischaemic damage to the pancreas may occur in hypothermic patients as a result of platelet activation (as discussed above) or of "microcirculatory shock" as suggested by Dr Foulis. Such damage to the pancreas may cause the release of various enzymes. Amongst these, elastase, is thought to play a role in the destruction of vessel walls in patients with pancreatitis. Damage to vascular endothelium would further impair PGI₂ synthesis and expose deeper layers of vessels which may initiate platelet aggregation due to their collagen content. The release of trypsin is thought to activate the coagulation cascade, enhancing the ten-
dency towards thrombosis. The uncontrolled release of pancreatic lipase and fat necrosis in acute pancreatitis may lead to a further increase in serum NEFA concentration. The low serum albumin concentrations occurring in acute pancreatitis would accelerate the rate of decay of PGI, especially in the presence of raised serum NEFA concentrations.

These factors would further enhance platelet hyperaggregability. More extensive ischaemic pancreatic damage may then occur leading to further release of pancreatic enzymes, thus setting up a vicious circle.

The mechanisms we suggest are consistent with Dr Foulis' conclusion that hypothermia may lead to pancreatic damage with an ischaemic pattern.

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Gliming motility of Acinetobacter anitratius

There is evidence to suggest that Acinetobacter anitratius syn A calcoaceticus can glide on the surface of solid media. This property was noted by one of us in batch 2 of NCTC 7844 strain of A anitratius. On inoculation on a MacConkey plate, the latter produced 1.5-2 mm diam lactose-fermenting compact colonies on the surface of the medium in 18-24 h at 37°C. The plate was then kept at room temperature for 18-24 h. This led to the development of many thin-like convexly arched offshoots around the periphery of the compact colonies. Hanging-drop preparations of the offshoots showed many rod-like forms attaching by one end to the underside of coverslip and performing fast pendular type flexion and extension movement by the distal freely hanging end. This type of movement had been reported by Bauer in myxococci and was responsible for the development of the convexly arched offshoots. This movement gradually became sluggish till it was lost in 7-10 days in the culture maintained on nutrient agar slopes at room temperature. It was not seen however, in the next two batches of the NCTC strain and in local isolates of A anitratius. However gliding motility could be induced in batch 4 of the NCTC strain and in 10 local isolates of A anitratius on peptone-deficient (0.1% Bactopeptone) agar (1-5% Bacto agar) slopes kept moist by covering the surface with a thin layer of phosphate buffer of pH 7.2-7.4 ± 0.2. The buffer not only kept the surface of the medium moist but also provided the ideal pH for the development of gliding motility in 18-24 h. A loopful of the growth occurring during this time was taken in the buffer and put in between coverslip and slide and seen under the low and high power of a microscope for evidence of gliding motility. Microscopic examinations showed a large number of cocccobacillary forms following each other, gliding to and fro slowly over the surface of the slide in the direction of their longitudinal axis towards the periphery of coverslip where air was present. The cells changed their direction occasionally on encountering non-mobile cells and sometimes retracted their path. The cells did not flex or twitch but occasional filamentous forms present in the group showed slow flexion and extension movement at one end. Occasional isolated cocccobacillary forms used to dart away at a great speed towards one direction; none of the cells showed presence of flagella on staining. The movement lasted till the wet preparations were dry and for maintenance of motility, subcultures of the strains had to be made on fresh medium at an interval of 3-4 days. This technique as compared to others is simple and can be carried out as a routine diagnostic procedure. The observation of gliding motility in A anitratius suggests that it could be a myxobacterium as pointed out by Lautrop.

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