industry, was found to have a titre of 1:128 in sera taken on days 20 and 33 after the onset of a 'flu-like illness in March 1978 contracted in Majorca. The illness progressed to pneumonia and he was admitted to hospital in England six days after onset. Recovery was slow. By April 1979 his antibody level had dropped to <1:16. Using class specific antiserum, it was demonstrated that only IgM antibodies were present. No antibodies were detected against *L. pneumophila* strains 2, 3, or 4.

While the serological results do not entirely meet the presently accepted rigid criteria, the high specific IgM titre, the negative IFAT result after 12 months, and the clinical picture all support a presumptive diagnosis of Legionnaires' disease.

A third male patient, aged 63 years, became ill in December 1977 with IFAT titres of 1:32 in two sera taken three weeks apart in March 1978 and a third serum taken a year later. The patient who works as a printer's cutter has a 13-year history of chronic bronchitis.

The prevalence of Legionnaires' disease among patients with sporadic, otherwise undiagnosed, pneumonia was found in this study to be 0.6% (2/340) compared with 1.5% (21/2023) reported in a general population study carried out in Nottingham (Macrae *et al.*, 1979). Other workers have reported a prevalence of 1% (Foy *et al.*, 1979) and 4-10% (Renner *et al.*, 1979; Cohen *et al.*, 1979) in groups of otherwise undiagnosed pneumonias.

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Platelet aggregation and serum prolactin

We recently described enhanced platelet aggregation after insulin-induced hypoglycaemia (Hutton *et al.*, 1979). Increased adrenaline release was thought to be largely responsible for this effect but the influence of other hormones released during an insulin stress test could not be excluded. We have therefore examined the effect on platelet aggregation of stimulating the release of one of these hormones, prolactin, with thyrotrophin releasing hormone (TRH).

Seven patients selected in accordance with the criteria used in the previous study were given 200 µg TRH intravenously. There was no significant change in aggregation induced by either adenosine diphosphate (ADP) or adrenaline during the 2-hour test period, although all subjects showed the expected rise in prolactin (Table).

These results make it unlikely that prolactin alone was responsible for the enhanced platelet aggregation observed

during the insulin stress test and thus add weight to our suggestion that our earlier observations resulted from the synergistic effect of adrenaline released *in vivo* with ADP added as the aggregating agent *in vitro*.

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Immunohistochemical demonstration of carcinoembryonic antigen

With reference to several recently published papers on the identification of carcinoembryonic antigen (CEA) in human breast carcinomas, mammary dysplasia, and granular cell myoblastomas using immunoperoxidase techniques and a commercially available antiserum to CEA (Shousha and Lyssiotis, 1978; 1979; Shousha *et al.*, 1979), we feel that some comment should be made regarding the specificity of the immunohistochemistry.

Many anti-CEA sera show cross-reacting activity against related glycoproteins such as non-specific cross-reacting antigen (NCA). These CEA-like materials are found in a large number of

Effect of TRH on prolactin level and platelet aggregation

Time (min)	Serum prolactin (mIU/l)	Platelet aggregation (%)	
		ADP	Adrenaline
Pre-TRH	207 ± 57	59.4 ± 7.4	63.9 ± 22.9
Post-TRH 30	1002 ± 420	52.9 ± 18.7	59.1 ± 15.1
Post-TRH 60	520 ± 218	49.3 ± 12.6	46.4 ± 19.6
Post-TRH 90	359 ± 153	55.7 ± 17.5	56.0 ± 22.5
Post-TRH 120	288 ± 140	63.1 ± 9.4	64.7 ± 24.2