bronchus. He refused treatment. His symptoms of polymyalgia rheumatica persisted despite an increase in his steroid dose.

Polymyositis in all these three patients was excluded by normal findings on creatine phosphokinase estimation, muscle biopsies, and electromyograms. Our cases suggest that polymyalgia rheumatica, like polymyositis, should be regarded as a marker of malignancy. We agree with Dr Huskisson and his colleagues that poor response of polymyalgia rheumatica to an adequate dose of corticosteroids should alert one to the possibility of an occult neoplasm.

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SIR,—Dr E C Huskisson and his colleagues (3 December, p 1459) rightly draw our attention to the possibility of underlying serious disease in patients presenting with classical features of polymyalgia rheumatica (PMR).

I wish to add the case of a 64-year-old man with a typical story of PMR who had some tenderness of the shoulder girdle muscles without wasting or weakness and a raised Westergren erythrocyte sedimentation rate (ESR) as the sole laboratory abnormality. Initial radiographs of his cervical spine were normal apart from some mild degenerative changes. There was a prompt abolition of his symptoms after starting prednisone 20 mg daily. However, the ESR failed to fall significantly. Subsequent investigations showed multiple metastases in the cervical vertebrae.

This suggests that an additional clinical clue to the presence of other disorders might be the failure of the ESR to respond to otherwise apparently adequate steroid therapy.

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Legionnaires' disease in London

SIR,—After your leading article on legionnaires' disease (7 January, p 2) and the preliminary case report of a subject with this disease in the *Lancet*¹ we appear also to have a case of legionnaires' disease, which has arisen in a similar area of London.

A 58-year-old housewife was admitted on 11 November 1977 with an eight-day history of pyrexial illness. She has a previous history of scleroderma which is at a low grade of activity. The usual range of investigations of a pyrexia of unknown origin showed a normal leucocyte count and differential and provided no definite diagnosis. On 23 November she developed a slight cough and on examination had scattered crepitations throughout both lung fields; chest x-ray showed bilateral fluffy pneumonic shadowing. A provisional diagnosis of mycoplasmal or viral pneumonia was made and treatment with oxytetracycline was started. Her lung condition worsened and neither mycoplasmal nor viral titres were diagnostic of infection; tetracycline was withdrawn. As she had rapidly spreading pulmonary infiltration and incipient respiratory failure, in the absence of a definitive diagnosis we performed an open lung biopsy. This showed changes characteristic of legionnaires' disease and on silver staining the organism associated with legionnaires' disease was seen. Treatment with erythromycin was started and within three days her fever had subsided. We are currently awaiting results of specific fluorescent antibody titre to confirm our diagnosis.

We would be interested to know whether any other hospitals in West London have had cases of intractable pneumonia admitted during the first half of November. Perhaps a diagnosis of legionnaires' disease should be considered in these cases and serum sent for more specific examination. Recognition of other cases would be the first step in establishing the epidemiological pattern in this newly identified disease and in allaying public disquiet related to it.

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¹ Ashford, R U, Edmonds, M E, and Shanson, D C,
Lancet, 1977, 2, 1364.

Dihomo-gamma-linolenic acid and thrombosis

SIR,—It is indeed gratifying to see confirmation of our studies and conclusions on the anti-thrombotic potential of dihomogamma-linolenic acid (DHHLA) in man (reported by Dr P B A Kernoff and colleagues (3 December, p 1441)), which we first reported this year.¹ We have since substantiated these earlier conclusions, when we reported the potent hypolipaeamic activity of DHHLA, again in both non-human primates and in man.^{2,3} These data, together with the consistent decrease in heparin neutralising factor and potential antiadhesive effect of DHHLA (suggested by the von Willebrand-like persistent inhibition of ristocetin-induced platelet aggregation⁴) must surely vindicate the studies of Hornstra *et al*⁵ and now suggest the use of DHHLA as a dietary supplement, as concluded by Dr Kernoff and colleagues.⁴

We are, however, obliged to comment on their reference to the differential efficacy of DHHLA as the acid or methyl ester. Our data have been used in this case out of context, since these results *in vitro* were compared with those in Dr Kernoff's paper, also *in vitro*, using the methyl ester and acid respectively.⁶ The activity of the methyl ester used in this and our original study has also been confirmed by Vane and his colleagues and is from the same source. In our opinion, there is unlikely to be any difference in activity *in vivo* using the free acid or ester. Indeed, Dr A L Willis—a colleague of Dr Kernoff—has previously claimed that the methyl ester was without activity and that the free acid was inactive on occasions depending on the batch.⁷ The inconsistencies observed in the study by Dr Kernoff and his co-authors are probably more readily explained in terms of batch variation and in the very varied dose schedule used.

Finally, in relation to dose, may we add a note of caution which may go some way to answer the apparent anomalies mentioned in your editorial of the same issue and in the present report. We have shown from studies in various animals, including non-human primates, that the dose of DHHLA is critical and should be held at less than 2 mg/kg.¹⁻³ It is quite possible to "overdose" with DHHLA, producing a "thrombogenic" condition. These results have been confirmed by Hornstra⁸ and, since Dr Kernoff and colleagues have now reduced the proposed oral dose from 0.4 g/kg (or greater) per day⁹ to that suggested in our original communication, no doubt they are also aware of the dangers of overdosing. Since

DHHLA probably represents the penultimate agent in the biosynthetic chain to prostaglandin (PG) E₁, such potency might be expected. Similarly, if the rate limiting conversion to PGG₁/PGH₁ or to arachidonic acid and the thrombogenic cyclic endoperoxides and thromboxanes depends on the availability of substrate, such dose-dependent, species-dependent, and indeed subject-dependent anomalies are not surprising.

We propose that DHHLA may be a useful normalising drug in patients at high risk but caution is needed at this stage in experimenting with normal volunteers.

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¹ Sim, A K, and McCraw, A P, *Thrombosis Research*, 1977, 10, 385.

² Sim, A K, and McCraw, A P, *Thrombosis and Haemostasis*, 1977, 38, 166.

³ Bryce, S G A, *et al*, paper presented at the 20th International Conference on Biochemistry of Lipids, September 1977.

⁴ Kernoff, P B A, *et al*, *British Medical Journal*, 1977, 2, 1441.

⁵ Hornstra, G, *et al*, *Lancet*, 1973, 1, 1155.

⁶ Farrow, J W, and Willis, A L, *British Journal of Pharmacology*, 1975, 55, 316P.

⁷ Willis, A L, personal communication, 1977.

⁸ Hornstra, G, personal communication, 1977.

⁹ Willis, A L, *et al*, *Prostaglandins*, 1974, 8, 509.

Confidentiality and life insurance

SIR,—Like Dr T M Pickard (10 December, p 1544) and Dr A J Caro (7 January, p 50), I am very concerned about the information that I divulge to insurance companies about my patients and particularly for a reason not yet discussed. Those who attend the Annual Representative Meeting know that I am very interested in confidentiality in relation to the computerisation of medical records. Much of the information that we give to insurance companies on behalf of our patients is stored on computers and neither we nor our patients have any further control over it.

I agree with Dr Pickard that many of our patients do not realise the significance of the permission to inquire about their medical history that they willingly and innocently give to the insurance agent, often not appreciating that it is permission to us to answer any question about their past history that the company may ask. Most suppose that we confirm the incidents that they have already admitted to the insurance company and label and date them more accurately; very few realise the extent of the questions we have to answer. I therefore think that the patient should see the form that his doctor will be sent and signify his permission on that form itself. If it asks questions he does not wish his doctor to answer he can think again or try another company—the forms are all slightly different, a maddening aspect when one patient takes out several policies at one time. We can all do without life assurance and mortgages can be arranged without medical reports. Some patients will not mind what is disclosed, others will prefer to remain unexposed, but all should realise what the choice involves. I also believe that if a company uses a computer to store its medical data it should state the fact on the form the patient signs.

The last aspect of this subject that concerns me is the increasing habit of the local manager requesting the information rather than the medical officer to the company, and I usually add "Chief Medical Officer" to the envelope