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

provided when sensitive data are included on the form.

JANE RICHARDS

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Safety of medical containers

SIR,—Medical containers should be stable, relatively unbreakable, and easy to use as well as proof against leakage and contamination of

the outside by the contents. Zuckermann and Taylor noted the risk of hepatitis attending removal of a wet plug from a blood container.¹ A new hazard that has recently crept into the laboratory is the bronchoscopy suction collector. The photograph shows one recently received in our laboratory. It was impossible to remove the snap-on

lid without producing an aerosol and contaminating the fingers. British Standard (BS 5213: 1975)—Specification for Medical Specimen Containers for Microbiology—has existed for two years but, though most laboratories now insist on containers with screw caps, containers complying with the British Standard have not yet become generally available, possibly because manufacturers anticipate that hospital administrators would not be willing to pay for them while cheaper but less safe alternatives are available.

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¹ Zuckermann, A. J., and Taylor, P. E., *British Medical Journal*, 1969, 2, 633.

Thalidomide and cranial nerve abnormalities

SIR,—Recent correspondence on thalidomide deformities (24-31 December, p 1672) prompts the suggestion that reference to an article on this subject¹ would help to solve the aetiological problems posed by facial and abducens nerve palsy in thalidomide children.

Both are motor nerves and would therefore suffer in some thalidomide cases when the neuromuscular junction failed to establish itself owing to the dampening effect of the drug on the initial impulses. Thalidomide causes deformity by preventing the initial and essential nerve impulses from reaching the neuromuscular junction in accordance with embryological laws. The failure of junction results in arrested development and resorption of the part or parts affected. Innervation of the limbs proceeds in a proximodistal direction in the fetus so that the proximal parts of the limbs are resorbed first, giving rise to the typical reduction deformities.

The brain is not affected as there are no nerve impulses in this structure during the organogenetic stage of development, but the

cranial motor nerves can be affected. Proximodistal spread would explain the electromyographic pattern described for the facial nerve and it can be said that the muscles round the eyes and mouth could not be affected in isolation. Postnatal improvement could occur in the remaining muscles, but it does not indicate nerve recovery. Nerve conduction would be in accord with the "all or none" principle.

Proprioception also fails to be established for the affected or resorbed parts and this is reflected in the proprioceptive nuclei. Consequently the affected child has no central proprioceptive record of the missing parts and so has no feeling of loss or inadequacy as would happen with a postnatal amputation. Within himself he is complete, his central proprioception corresponding exactly to his external or actual conformation. He has no use for artificial aids and is normal at forebrain level.

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¹ Gordon, G. C., *Developmental Medicine and Child Neurology*, 1966, 8, 761.

IUCDs—a contraindication to removal

SIR,—Histories such as that described by Dr D F E Thallon (7 January, p 51) are given by patients from time to time to the regret of the family planning practitioner, and it seems that pregnancy must result from implantation of an ovum fertilised by an intercourse before removal of the IUCD.

For this reason the teaching in family planning training clinics has always been that an IUCD should be removed either after a menstrual period or after a period of abstinence from sexual intercourse but not normally mid-cycle. This advice is contained in the Family Planning Association's Handbook of 1973.

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SIR,—I think Dr D F E Thallon (7 January, p 51) is too conservative in suggesting that an intrauterine contraceptive device (IUCD) should not be removed for seven days on each side of mid-cycle. A patient of mine conceived in a cycle in which her IUCD was taken out on day 26. Menstruation usually occurred every 30 days. Even if ovulation is estimated to occur 14 days before the expected period date this IUCD was not removed until 10 days later. Unlike Dr Thallon's patient, the outcome was a happy one, because in this instance the device was removed after two years' use to allow conception.

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Problems with IUCD tails

SIR,—Mr R A Sparks (19 November, p 1351) draws attention to some of the problems associated with the tail of the intrauterine contraceptive device (IUCD). Another difficulty is that, while most practitioners who fit an IUCD place emphasis on teaching the woman to check that it has not been expelled, a large number of patients are unable to detect

the IUCD tail. We recently published an analysis of 172 women bearing IUCDs with tails fitted for at least six months who were seen consecutively by one family planning doctor.¹ Of these, 17 were unable or unwilling to examine themselves, while a third of the remainder (57/155) were persistently unable to find the tail. Of the factors possibly concerned, there was a weak positive association between frequency of detection and increased parity and higher social class but no correlation with length of tail. A lower detection rate for the Copper-7 (59.3%) than for the Lippe's Loop (73.8%) may only reflect small-number variation. However, the Lippe's Loop has two tails of thicker nylon while the tail of the Copper-7 is a single thread. The numbers of other types of IUCD used were too small for any comparison to be attempted.

The motivation of the woman is probably the most important single factor in successful vaginal self-examination. If she is made anxious by vaginal manipulation a woman will either not examine herself or do so less thoroughly. In the past such patients have been recognised as being unsuitable for vaginal diaphragm or cervical cap contraception and perhaps such inhibitions should be appraised when considering fitting an IUCD. Without self-examination no IUCD wearer can be sure that she still carries an effective contraceptive method.

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¹ Chamberlain, G., *Fertility and Sterility*, 1977, 28, 1121.

Postoperative chest infections

SIR,—Though your recent leading article on postoperative chest infections (10 December, p 1500) rightly emphasises the usefulness of rational antibiotic therapy in established postoperative chest infection, we feel it gives too important a role overall to their possible use in prophylaxis. In calling for careful well-constructed trials to answer the "important unanswered questions" on the usefulness of antibiotics and physiotherapy in preventing postoperative chest infections it is clear that you have ignored several contributions, among them our own¹ on this subject.

Postoperative pulmonary collapse may vary in extent from the collapse of a group of alveoli or a small lobule to that of a segment, a lobe, or rarely a whole lung. It is clear that infection does not complicate all cases of minor pulmonary collapse and only the less fortunate patients progress from lobular collapse to the more serious complication of marked pulmonary collapse and consolidation.² Alveolar or lobular collapse is reversible by repeated sustained maximum inspiration (SMIs).³ We have shown that the number of postoperative chest complications is significantly reduced when patients are encouraged to take frequent SMIs in the early postoperative period compared with patients who have had nothing but routine pre- and postoperative physiotherapy. There was also a suggestion in our study that frequent SMIs were more effective in treating lobular collapse than was physiotherapy. In our study the pulmonary complication rate was 71% in the physiotherapy group, and 17% of these were serious complications. It is clear that physiotherapy as it is practised at the moment in the United Kingdom is not effective in preventing postoperative pul-