when he is admitted to hospital and therefore is no longer able to contact the general practitioner who has probably for many years been his guide, philosopher and friend. I am, etc.,

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Pyomyositis in London

SIR,—Dr. D. W. Rogers (29 September, p. 679) rightly describes pyomyositis as the formation of abscesses in one or more skeletal muscles but goes on to describe an abscess "lying in an intermuscular position." The intramuscular location of the abscesses of pyomyositis has been well described.¹⁻⁴ Anand¹ noted that an intermuscular abscess was usually associated with nematode infection, particularly *Dracunculus medinensis*. This is in accord with my own experience in the Nigerian savannah.

The muscles of the buttock and upper thigh are a common site for the abscesses of pyomyositis, which may be single. However, they are also the usual site for a type of abscess which occurs both in the tropics and the temperature zones—that is, one following an infected intramuscular injection. This may contain faecal organisms but could, like 46% of pyomyositis abscesses, contain the same phage type of staphylococcus as is carried on the patient's own skin.⁵

Occasional cases of pyomyositis may indeed be occurring unrecognized in non-tropical countries, but I would suggest that if only those abscesses with the classical features of being multiple and intramuscular are labelled pyomyositis, then the true incidence of the disease contracted outside the tropics would still closely approach zero.—I am, etc.,

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¹ Anand, S. V., and Evans, K. T., *British Journal of Surgery*, 1964, **51**, 917.

² Horn, C. V., and Master, S., *East African Medical Journal*, 1968, **45**, 463.

³ Ransford, O. N., *East African Medical Journal*, 1966, **23**, 278.

⁴ Levin, M. J., Gardiner, P., and Waldvogel, F. A., *New England Journal of Medicine*, 1971, **284**, 196.

⁵ Foster, W. D., *Journal of Hygiene*, 1965, **63**, 517.

Epidemiology of Hypospadias

SIR,—Dr. C. J. Roberts and Mr. S. Lloyd (31 March, p. 768), studying 93,000 births in South Wales in 1964-6, have reported a seasonal cycle for the frequency of hypospadias, an excess of cases being observed in babies conceived in winter and born in the second half of the year. A seasonal cycle was previously reported by Wehrung and Hay,¹ based on 5,145 cases of hypospadias in the United States in 1962-5. Their cases were more common among spring-time births, but only in the western seaboard area of the U.S.A. and only in 1962-3, not in 1964-5. On the other hand, Professor H. Campbell and others (7 July, p. 52) were unable to demonstrate any seasonal variation in hypospadias in the Registrar General's data for the whole of England and Wales for 1967-71, nor could Record and Armstrong (28 July, p. 233) in Birmingham births in 1950-72.

Two earlier studies^{2,3} also failed to show seasonal variation.

We have studied the monthly incidence of this malformation in 59,261 births in the record-linked Jerusalem Perinatal Study^{4,5} (see table 4). There was a high reported frequency with a marked cyclic variation, with two peaks and troughs per year. The maxima were among spring and autumn births and the minima in winter and summer, a pattern very similar to that of Down's syndrome in our population. An artefact due to differential under-reporting is most likely to be responsible; all other minor malformations show a single cycle with a trough in May. Furthermore, under-reporting should be most prevalent in autumn and spring at the times of the Jewish New Year and Passover holidays.

Hypospadias by Month of Birth, West Jerusalem, April 1964-March 1973

Month of Birth	No. of Cases of Hypospadias	Rate per Thousand Male and Female Births
Jan.	14	2.8
Feb.	12	2.9
Mar.	17	3.4
Apr.	22	4.8
May	18	3.6
June	10	2.0
July	8	1.5
Aug.	23	4.3
Sep.	15	3.1
Oct.	16	3.2
Nov.	9	1.9
Dec.	13	2.6
Total	178	3.0

P < 0.025 (Fourier analysis).

This variety of seasonal patterns for hypospadias in different studies seems inconsistent with a direct aetiological effect of hours of daylight, temperature, or climate as postulated by Dr. Roberts and Mr. Lloyd. A more promising direction of research is likely to be a search for specific infectious agents or the use of hormones in early pregnancy.—We are, etc.,

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¹ Wehrung, D. A., and Hay, S., *British Journal of Preventive and Social Medicine*, 1970, **24**, 24.

² Sorensen, H. R., *Hypospadias: With Special Reference to Aetiology*. Copenhagen, Munksgaard, 1953.

³ Slater, B. C. S., Watson, G. I., and McDonald, J. C., *British Journal of Preventive and Social Medicine*, 1964, **18**, 1.

⁴ Davies, A. M., Prywes, R., Tzur, B., Weiskopf, P., and Stark, U. V., *Israel Journal of Medical Science*, 1969, **5**, 1095.

⁵ Harlap, S., et al., *Israel Journal of Medical Science*, 1971, **7**, 1520.

Laterality of Fractures

SIR,—Drs. T. G. Williams and B. P. Heather (6 October, p. 52) suggest that injury is more common to the non-dominant forearm.

In a paper on "Absence from Work after Fractures of the Wrist and Hand"¹ my colleagues and I noted 93 injuries to the dominant wrist and hand compared with 57 on the non-dominant side. The differences were most noticeable for those involved in heavy manual or sedentary work, there being no difference in those employed in light manual work (see table). The fractures considered were those of the metacarpals, scaphoid, and lower end of the radius and ulna.

I would not suggest that injuries are necessarily more common in the dominant

Employment	Side Injured	No.
Heavy manual	Dominant	41
	Non-dominant	24
Light manual	Dominant	25
	Non-dominant	26
Sedentary	Dominant	27
	Non-dominant	7
Dominant 93 Non-Dominant 57		

hand but that the issue is not proved.—I am, etc.,

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¹ Goodwill, C. J., Bridges, P. K., and Gardner, D. C., *Annals of Physical Medicine*, 1969, **10**, 180.

SIR,—Drs. T. G. Williams and B. P. Heather (6 October, p. 52), reporting that their patients tended to injure mainly the non-dominant forearm, wondered if this tendency had been noted previously. I would draw their attention to an article I published in 1969¹ on Smith's fractures in which I recorded that only 12 out of 44 patients had injured their dominant wrist.—I am, etc.,

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¹ Woodyard, J. E., *Journal of Bone and Joint Surgery*, 1969, **51B**, 324.

Propellant a Factor in Asthma Deaths?

SIR,—Dr. V. E. Archer (29 September, p. 696) suggests that aerosol propellants may be a factor in the causation of death in asthmatic patients. There is no doubt that fluorocarbon or haloalkane compounds in high concentration are cardiotoxic and have been responsible for "sniffing" deaths, but this is an entirely different situation from the asthmatic patient using a pressurized bronchodilator aerosol. In a review of the pertinent published studies,¹ it concluded that "though fluorocarbon propellants may be hazardous if they are deliberately abused, the weight of the evidence attests to both the safety and efficacy of pressurized bronchodilator aerosols when properly used to relieve asthma."

Dr. Archer gives the impression of having examined the relevant literature, yet he fails to mention a large body of published reports which fail to sustain his views and has selected from the references listed in his letter only those excerpts which support his thesis. For example, he refers to the study by Taylor and Harris² in mice as evidence that anoxia enhances the cardiotoxicity of the haloalkanes. It has been amply demonstrated that in that study it was the anoxia alone which was cardiotoxic.^{3,6} Other workers found no evidence that anoxia enhances the cardiotoxicity of the propellants.^{3,4,7,8}

That adrenaline in large doses given intravenously to dogs previously exposed to high concentrations of fluorocarbon compounds will produce cardiac arrhythmias was established by Reinhardt et al.⁹ in 1971 and confirmed by Clark and Tinston^{7,8} and by McClure.⁴ Under similar conditions, the last two investigators were unable to produce cardiac arrhythmias by the intravenous injection of large doses of isoprenaline. Clark and Tinston⁸ summarize their findings as