isoprenaline inhaler during an acute asthmatic attack in man, by giving 0·5 μg/kg isoprenaline intravenously every 30 seconds to hypoxic dogs during a five-minute exposure to fluorocarbon 11, we again failed to induce cardiac sensitization. The Pao_2 values of approximately 50 mm Hg recorded in these experiments are comparable to those found by Tai and Reed[3] in patients with clinically severe asthma, but the blood concentration of fluorocarbon and the dose of isoprenaline were far in excess of those likely to result in man from the use of pressurized inhalers. The concentrations of fluorocarbon 11 in the blood were at least twenty times greater than those found in man by Dollery *et al.*[4] following a massive overdose of 30 puffs of an inhaler in two minutes, and the dose of isoprenaline was equivalent to at least 50 puffs of an inhaler in the space of five minutes.

Our results, therefore, do not support the view that the unexplained deaths among asthma sufferers could have been due to cardiac sensitization resulting from the use of pressurized aerosols containing isoprenaline.

These results were presented in detail to the European Society for the Study of Drug Toxicity in Berlin, June 1971, and are to be published in the Proceedings.

—We are, etc.,

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Infant Cot Deaths

SIR,—Among the many hypotheses put forward to account for these tragic infant deaths the suggestion of the possibility of airway obstruction of the larynx by closure of the epiglottis has not been mentioned.

The most common postmortem findings reported have been those of a minor upper respiratory tract infection. This would suggest that the infant would have a cough with some tacky mucus. If one would postulate that the sequence of events that lead to death would be that the infant coughs up a little mucus, and, as all infants do, attempts to swallow the mucus that reaches the pharynx. This results in closure of the laryngeal airway by the epiglottis, which becomes "gummed" down by some residual mucus, effectively blocking the next step, which would be an inspiration, thus forcing the epiglottis down further over the larynx. As the tidal air has been expelled in the process of coughing, there is no way for the infant, or even an adult, to make a forced expiration to force the epiglottis open. Death is rapid and silent—without a struggle from asphyxiation or vagal inhibition.

The rapid death leaves little time for morbid pathological changes to be found and in the process of handling the dead infant some residual air is forced from the lungs, opening the epiglottis and destroying the only evidence of cause of death.

If this hypothesis, which would account for the lack of any significant postmortem changes, is accepted, and it would appear to be a reasonably valid one, we have come to the reluctant conclusion that there is no solution to avert this tragic problem.—I am, etc.,

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Severe Malarial Infection

SIR,—The report from Ibadan entitled "Severe Malarial Infection in a Patient with Sickle-cell Anaemia" (22 May, p. 445) is by no means as unusual as the authors would appear to suggest. When working at Harari Hospital, Rhodesia, I encountered several cases of acute malaria (due to *P. falciparum*) in patients with sickle-cell disease. The diagnosis of sickling was confirmed by haemoglobin electrophoresis, and though I do not have the records at hand I can remember at least one child with sickle-cell anaemia who died in an acute attack of malaria. I know that other members of the medical staff there had the same experience.

We knew of course from the original observation of Beet,[1] and the work of Brain,[2] Raper,[3] [4] and others, of the increased resistance to malaria conferred by the sickling trait. However, it was our impression that malarial infections occurred in the homozygous state which were quite often severe, which could precipitate a crisis, and which could rapidly lead to death through dehydration, sequestration, and lysis of red cells. When presented with a "sickle-cell crisis," then, it was always our practice to disregard theoretical considerations of increased resistance to malaria and to make a search for the parasite.—I am, etc.,

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Humidity in Hospital

SIR,—Dr. J. R. R. Wray's letter (19 June, p. 708) drawing attention to excessively dry atmospheres in new hospitals and houses is timely. There is now much advertising of the latest heating methods, which produce even hotter and drier atmosphere, so that the relative humidity in both hospitals and houses is commonly down to 20-25% in cold weather. This low humidity at a room temperature of 70°F (22°C) causes severe drying of naturally moist surfaces. This is unpleasant for the healthy, and dangerous for the diseased, respiratory tract. Dr. Wray very rightly warns that cheap hygrometers are often unreliable. I suggest that *any* air hygrometer needs constant checking; the whirling wet bulb instrument is the one of choice.—I am, etc.,

DOUGLAS NEEDHAM

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Terminology

SIR,—What is the purpose of the word "disease" in the phrase "chronic obstructive airways disease" (5 June, p. 561)? Presumably this phrase is intended to refer to a group of patients with a specified disorder of function in the respiratory system, chronic airways obstruction. We have become accustomed to the use of phrases indicative of disorders of function in other systems (for example, systemic hypertension, intestinal obstruction, raised intracranial pressure, congestive heart failure) or even on the vascular side of the lungs themselves (pulmonary hypertension), without thinking it necessary to add "disease."

Why is it so generally thought necessary to add this word in referring to abnormally high resistance to gas-flow in the lungs? To my mind, in this context it is worse than useless; its presence distracts attention from the main purpose of the use of this sort of term, namely to refer to groups of patients characterized by a common disorder of function, without commitment to implications about aetiology, structural changes, or other characteristics.—I am, etc.,

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Multiple Eponyms

SIR,—I wonder whether Dr. T. J. David (12 June, p. 655) would accept for his record the description of craniofaciomandibular dyostosis syndrome by the Troquart-Apert-Franceschetti - Greig - Helmholz - Harrington - Marie - Sainton - Crouzon - Treacher - Collins syndrome. If not, perhaps he will allow me to add Böök-Hesselvik-Buckley-Yakovlev - Park - Powers - Jackson - Scott - Banks - Brown - Harper - Meisenbach - *et al.*—I am, etc.,

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Infantile Subdural Haematoma

SIR,—The interesting paper by Mr. A. N. Guthkelch (22 May, p. 430) is of particular concern to Home Office pathologists, for whom certain cases of fatal subdural haematoma in infancy have been a problem for some time. Such fatalities are best considered in the three main distinguishable categories.

(1) Those with obviously related head injury such as bruising or fractures or both.

(2) Those with injuries elsewhere on the body, but no significant injury to the head.

(3) Those with no significant injury anywhere.

The first category presents no problem in that the injuries clearly lead to death. The second category is more difficult, particularly if the other injuries are of long standing, but there should be no hesitation in attributing the fatal subdural haematoma to the general violence implicit when there are injuries of appropriate severity and duration to other parts of the body.

In the third category I have for some

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