

suggests, however, that both pain threshold and pain tolerance were raised in their swimmers.

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¹ Libman E. *JAMA* 1934;102:335.

² Keele KD. *Lancet* 1954;i:636-9.

³ Harrison IB, Bigelow NH. *Proc Ass Res Nerv Dis* 1943;23:154.

⁴ Lewis T. *Pain*. New York: Macmillan, 1942.

* * * We sent this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—The comments of Dr K O Keele on our article on pain perception in competitive swimmers (11 July, p 91) are interesting and suggest an extension to our investigation. In this study of ischaemic pain perception it was found that the highly trained swimmer showed clearly elevated pain tolerance levels. In contrast, pain threshold levels were only a little higher than those of the comparison groups of less experienced athletes, and the differences were not in fact statistically significant. It is possible, of course, that differences in pain threshold levels could have been demonstrated if larger groups of subjects had been used.

Dr Keele suggests on the basis of his own work¹ that not only tolerance levels of pain perception, but also pain thresholds should have been found to be higher in our competitive swimmers. This suggestion would seem to be based on his observation that some subjects exercise to levels of muscular fatigue when undergoing the ischaemic pain test. His evidence indicates that such subjects are generally hyposensitive to pain when other indices of pain sensitivity are used. Muscular fatigue did occur more frequently in our competitive swimmers than the comparison groups when tested for ischaemic pain tolerance. But all subjects reported experiencing considerable levels of pain in contrast to Keele's hyposensitive subjects, who apparently experienced little or no pain in similar circumstances.

It should be remembered, however, that for the competitive swimmer pain has informational value, especially during training. This may not be so for other athletes such as boxers, and it may well be that such athletes do show elevation of pain thresholds as well as pain tolerances. If this is the case then the important role that "significance" plays in pain perception would again be emphasised.

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¹ Keele KD. *Lancet* 1954;i:636-9.

Stridor

SIR,—In my letter about stridor (8 August, p 435) I indicated the importance of establishing whether or not epiglottitis was present in cases of stridor. This can be done safely by the ear, nose, and throat specialist in the presence of a competent anaesthetist. I emphasised that facilities for tracheostomy should be available in case an emergency attempt at intubation should be unsuccessful. However, since writing about this matter I have heard of the use of a high-pressure oxygen source in this situation.¹

An intravenous cannula was inserted in the midline through the cricothyroid membrane and the needle withdrawn, leaving the cannula

in place in the airway below the vocal chords. A high-pressure oxygen source (wall pressure 60-80 lbf/in²) (414-552 kPa) with an on-and-off valve and Luer lock connection were attached to the intravenous cannula. Thus a 4-year-old child was ventilated with expiration taking place through the glottis while more leisurely intubation was carried out.

Some care has to be taken to see that the cannula is in place in the airway, otherwise marked surgical emphysema will occur. The availability of this technique may prove useful in the management of upper-airway obstruction in the future, reducing the need for tracheostomy.

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¹ Davies IJT, Belam OH. *Practitioner* 1967;199:76-7.

Renal damage and glue sniffing

SIR,—We were interested in the reported association of mesangiocapillary glomerulonephritis and glue sniffing reported by Dr G Venkataraman (28 November, p 1467), as we have recently seen a similar example.

An 18-year-old labourer presented with a two-week history of ankle swelling. There was no family history of renal disease. He admitted to at least six episodes of glue sniffing (Evostik) over the previous eight months. Investigations revealed serum urea 5 mmol/l (30 mg/100 ml), creatinine 114 μ mol/l (1.3 mg/100 ml), total serum protein 43 g/l, albumin 26 g/l, and a 24-h urinary protein excretion of 8.7 g. Serum complement, immune complexes, antistreptolysin-O titre, and intravenous urogram were normal. A renal biopsy showed mesangiocapillary glomerulonephritis type I, with diffuse finely granular immunofluorescence for IgG and IgM and focal fluorescence for C3 and C1q along the glomerular capillary basement membrane.

Although the presence of mesangiocapillary glomerulonephritis may be coincidental, the possibility of an association with glue sniffing must be borne in mind.

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Atopic eczema after viral infection

SIR,—The case report by Dr R St C Barnetson and others (24 October, p 1086), of atopic eczema following infectious mononucleosis highlights an interesting point concerning the pathogenesis of this condition.

Infectious mononucleosis has been shown to cause an increase in suppressor T-lymphocytes¹ and a reduction in IgE levels.² In atopic patients, however, these changes in suppressor T-lymphocytes (usually low) and IgE (usually high) do not apparently occur during the various phases of viral infections.³ This "non-response" of the suppressor T-cells and inability to regulate IgE production could explain why viral infections such as infectious mononucleosis might precipitate eczema in genetically predisposed individuals, in whom there is a failure of normal suppressor mechanisms (possibly due to thymic hormone deficiency^{4 5}).

There has been a great deal of interest recently in the relation of atopic eczema to certain food allergens.⁶ It may be that other "triggers"—such as viral infections—are as

important in the pathogenesis of atopic eczema in individuals with abnormal immunoregulatory mechanisms.

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¹ Crawford DH, Brickell P, Tidman N, McConnell I, Hoffbrand AV, Janossy G. *Clin Exp Immunol* 1981;43:291-7.

² Bahna SL, Horowitz CA, Heiner CD. *Pediatr Res* 1977;11:484.

³ Perelmutter L, Potvin L. *Ann Allergy* 1980;45:18-22.

⁴ Garaci E, Ronchetti R, Del Gobbo V, Tramutoli G, Rinaldi-Garaci C, Imperato C. *J Allergy Clin Immunol* 1978;62:357-62.

⁵ Byrom NA, Staughton RCD, Campbell M-A, et al. *Br J Dermatol* 1979;100:499-510.

⁶ Atherton DJ, Sewell M, Soothill JP, Wells RS, Chilvers CED. *Lancet* 1978;i:401-3.

Effects of lay groups on weight reduction

SIR,—The article from Oslo on the short-term and long-term effects of lay groups on weight reduction (24 October, p 1093) was encouraging in the field wherein results have been exceptionally poor in the past.

I wish to correct two minor errors. In table IV I am quoted as having produced an average weight loss of 2.7 kg in 76 patients after eight weeks. In fact, the figures were taken from table 13 in my book, which relates to results after five to eight years.¹ In table V they quote me as giving long-term results of 1-13% after five years. This was my summary of results prior to 1969 in the world literature. My own results (pp 125, 127) show that after 10-18 years 48% of 150 patients had maintained a weight loss of at least 5% of their original weight, while 29 of them (19%) had maintained a weight loss of over 20 lb (9 kg) for the same length of time.

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¹ Craddock D. *Obesity and its management*. 3rd ed. London: Churchill Livingstone, 1978:119.

Frozen shoulder: adhesive capsulitis

SIR,—Your leading article "Frozen shoulder: adhesive capsulitis" (17 October, p 1005) by Professor M I V Jayson does nothing to clarify an already confused situation. It would appear that patients with painful, stiff shoulders of the "adhesive capsulitis" type, are to continue to be told by their doctors that there is no effective treatment and the condition is self-limiting within 18-24 months, and that analgesics and physiotherapy are to be prescribed. Unfortunately, few patients are likely to understand the reasons for such a policy, and most will continue to complain of rest pain at night and restriction of movements severe enough to prevent a return to work.

Those of us who see a large number of painful, stiff shoulders of this type appreciate that most patients can be rendered pain free by one to three paired injections, at weekly intervals, of a mixture of lignocaine and 20 mg of a steroid preparation (such as methylprednisolone acetate or triamcinolone acetonide) into the subacromial bursa and glenohumeral joint cavity. An injection into the subacromial bursa or joint cavity alone carries a very low rate of success. Movement of the shoulder increases as the patient is able to move the