Some of the symptoms attributable to hyperventilation arise from cerebral vasoconstriction and hypoxia, whereas others are related to selective depression of the parasympathetic branch of the autonomic nervous system, which results in a picture of sympathetic dominance with features such as tachycardia and palmar and axillary sweating.12 Patients suffering from hyperventilation may present with symptoms relating to virtually any organ or system and the condition can mimic angina.4

The neglect of hyperventilation as a positive diagnosis in general medicine has doubtless led to much fruitless investigation and frustration on the part of both doctor and patient. For those patients suffering from hyperventilation who have presented to psychiatrists or psychiatrically minded general practitioners one wonders how many have attracted the label "neurotic" or had their symptoms attributed, by circular logic, to an underlying "anxiety state."

Since becoming aware of Lum's work I have referred a number of anxious patients to a physiotherapist colleague for breathing retraining. My limited experience with this approach to date encourages me to believe that it is well worth while to keep hyperventilation in mind as a causal or contributory factor in patients presenting with anxiety and panic attacks.

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- Lum LC. The syndrome of habitual chronic hyperventilation. In: Hill OW, ed. Modern trends in psychosomatic medicine, Vol 3. London: Butterworths, 1976, 196-230.
  Lum LC. Hyperventilation and anxiety state. J Roy Soc Med 1981;74:1-4.
  Rice RL. Symptom patterns of the hyperventilation syndrome. Am J Med 1950;8:691.
  Evans DW, Lum LC. Hyperventilation as a cause of chest pain mimicking angina. Pract Cardiol 1981;7: 131-9.

## Cold weather and testicular torsion

SIR,-We were interested in the paper by M R B Shukla and others (20 November, p 1459) on cold weather and testicular torsion and in Mr Roger Williamson's letter (30 April, p 1436) commenting on the paper.

We have studied all cases of testicular torsion presenting to the Dundee hospitals over the 10 years 1973-82. Our results, however, are somewhat at variance with the experience of Mr Shukla and others when considering the distribution of cases over the 12 months of the year: 134 cases occurred which were proved at operation. The distribution of cases by calendar months was: January, 11 cases; February, 12; March, 19; April, 8; May, 11; June, 8; July, 6; August, 11; September, 11; October, 10; November, 14; December, 13. Therefore 36 cases (27%) occurred during the warmer months of May to August. The  $\chi^2$  test of goodness of fit when applied to these figures showed no significant difference from a normal distribution ( $\chi^2$  = 10.89, df=11, 0.5>p>0.1). In reviewing these 134 cases we considered it was not valid to compare the time of occurrence of torsion with the outside ambient temperature because in about 55% of cases where the place of onset of pain was recorded in the notes the patient was indoors at the time. Also the area served by the Dundee hospitals includes coastal and inland areas and a wide range of elevations above sea level with little geographical correlation of temperature with that recorded

at the local weather station. Hence it is impossible to determine the precise ambient temperature for each case.

Although our experiences seem to be statistically different from those of Mr Shukla and others, when the two distributions are compared by the  $\chi^2$  test they do not differ significantly (0.5>p>0.1,  $\chi^2 = 10.9$ , df=11). But when the two sets of figures are summated (total 180 cases) and compared to a uniform distribution, then p < 0.05,  $\chi^2 = 20.68$ , df = 11, as in the smaller series of Mr Shukla and others. Both series include relatively small numbers, but perhaps a larger series would show a more definite trend of monthly or seasonal variation. It should be noted, however, that testicular torsion is not unknown in hotter climates-for example, 36 cases were reported from Port of Spain, Trinidad, over a six year period.1 It might therefore be useful to carry out a prospective study in two regions with differing annual temperature patterns to elucidate further any monthly variation or association with cold weather.

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Sant GR. Testicular torsion in Trinidad. J. R. Coll. Surg Edin 1982;27:353-7.

## Serum creatinine concentration and renal function in rheumatic diseases

SIR,—Dr Ola Nived and others show an alarming ignorance in their short report (26 February, p 684). They wonder if "the serum creatinine concentration, which is commonly taken as a useful index of kidney function, may not be reliable for that purpose in rheumatoid arthritis," and they go through a bizarre exercise of comparing the mean serum creatinine concentration and simultaneous clearance of 51Cr-edetic acid in a group of patients with inflammatory joint disease and matched hospital controls. At least they come to the correct conclusion when they realise that the serum creatinine concentration varies not only with glomerular filtration rate but with muscle mass, a principle fully developed many years ago. They could have saved themselves the trouble and expense of this investigation.

The fault lies in their interpretation of a "normal" value. The "normal" range of serum creatinine concentration is based on large numbers of hospital patients with normal glomerular filtration rates and largely reflects variations in their muscle mass. Most people do not change their muscle mass in the short term, so a change in serum creatinine concentration is mostly governed by a change in renal function. No doubt many of your discerning readers are aware that a serum creatinine value of 100  $\mu$ mol/l (1·1 mg/100 ml) in a 60 year old man weighing 73 kg represents a glomerular filtration rate of only 71 ml/min/ 1.73 m<sup>2</sup>—that is, moderate renal failure within the "normal" range. Five years later, having lost 5 kg in weight from inflammatory joint disease, the same "normal" creatinine in this man conceals a further decline in renal function to 63 ml/min/1·73m2. Cockcroft and Gault2 provided an easy formula for clinical use to calculate the glomerular filtration rate from the patient's age, weight, and plasma creatinine concentration. This gave good comparison

between the simultaneously measured inulin clearance and has long proved its worth in clinical practice.3 Serum creatinine concentration is still a good estimate of renal function even in rheumatoid arthritis; the problem lies in learning how to interpret it.

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- Smith HW. The kidney. New York: Oxford University Press, 1951:182-94.
  Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron 1976; 16:31-41.
  Charleson HA, Bailey RR, Stewart A. Quick prediction
- of creatinine clearance without the necessity urine collection. NZ Med J 1980;92:425-6.
- \*\*\*We sent a copy of this letter to the authors, who reply below.—ED, BMJ.

SIR,—Dr C P Swainson's crisp comments on our short report on serum creatinine concentration in rheumatic disease are somewhat surprising because his and our concluding sentences convey the same message.

Our observation that serum creatinine concentration is lower in patients with rheumatic disease than in other patients with comparable glomerular filtration rates does not seem to have been published before. We considered the observation important for those dealing with patients with inflammatory joint disease. Our findings were based on direct measurements and were independent of special formulas, nomograms, or assumptions about creatinine excretion rate or muscle mass. Inevitably, formulas for the prediction of glomerular filtration rate from measured serum creatinine concentration and body weight can be misleading because body weight does not reflect only muscle mass. Changes in body weight, such as the loss of 5 kg in Dr Swainson's example, may be caused by loss of tissue other than muscle.

The important practical point for the busy doctor to remember is that a serum creatinine value as low as 80 μmol/l (0.9 mg/100 ml), for example, does not exclude appreciably reduced glomerular filtration rate in patients with rheumatic disease. Clearly, there is no real controversy between the antipodes concerning the importance of muscle mass and creatinine excretion for the correct interpretation of a serum creatinine concentration value.

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## Location of parathyroid adenomas by thallium-201 and technetium-99m subtraction scanning

SIR,—While we agree with sentiment of Mr A E Young and others (30 April, p 1384) that a "reliable method for locating abnormal parathyroid glands would be of great value to surgeons," our preliminary experience at the Middlesex Hospital and our information from other centres with experience in this technique leaves us unable to recommend thallium-201 and technetium-99m subtraction scanning as a useful preoperative procedure.