Points

Stress hyperglycaemia in non-diabetic patients with myocardial infarction

Drs M W BECKETT (Accident and Emergency Department, Central Middlesex Hospital, London NW10 7NS) and D J SHAWE (Northwick Park Hospital, Harrow) write: We were interested in the study of Dr G A Oswald and colleagues (11 October, 917) showing the association in non-diabetic patients between hyperglycaemia after myocardial infarction and eventual mortality. However, little information is given about the mode of death, and it would be useful to see an analysis comparing the results in those who died of intractable cardiac failure and those who suffered "cardiac arrest" without previously developing severe failure. Plasma catecholamine values are greatly raised in patients who have suffered a myocardial infarct and have then suffered a cardiac arrest shortly after the blood sample was taken,[1] and it would be of interest to know to what extent a raised blood glucose concentration was found to be a predictor of cardiac arrest. Those caring for a patient with acute myocardial infarction have to judge the risk of sudden cardiac arrest when deciding whether to keep a patient at home or whether to use a scarce coronary care bed. A blood glucose estimation can be available in a few minutes, and it would be most useful if the risk of sudden ventricular fibrillation associated with hyperglycaemia could be more accurately measured.

1 Little RA, Frayn KN, Randall PE, *et al.* Plasma catecholamines in the acute phase of the response to myocardial infarction. *Arch Emerg Med* 1986;**3**:20-7.

Look before you quote

Mr A G GORDON (London SE5 8AD) writes: Kay Dickersin and Peg Hewitt (18 October, p 1000) draw attention to the widespread problem of incorrect citation of primary references but offer no remedy. Yet much more can be done explicitly to condemn this practice and action can be taken against offenders, which would greatly improve the situation, at least for scientific journals. The only relevant part of the Vancouver code is the woolly sentence, "The references must be verified by the author(s) against the original documents."[1] I found variants of this sentence in three out of 23 widely selected medical journals plus *Nature* and *Science* (12%). The rest ignored the issue. I suggest that this sentence should be replaced by, "Primary sources must be cited only if they have been read personally in total; otherwise cite secondary sources only." A similar statement appears in the *Indian Journal of Pediatrics*. This directs attention to the accuracy of the quotation rather than that of the citation. All journals should contain wording at least as strong as this and should reject papers where authors have obviously cited something they have not read. Perhaps authors should explicitly have to state "I have read all my cited sources."

1 International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1982;**284**:1766-70.

Muscle power after glucose-potassium loading in undernourished patients

Dr COLM LANIGAN (King's College School of Medicine and Dentistry, London SE5 8RX) writes: I read the paper by Mr S T F Chan and colleagues (25 October, p 1055) with interest but would like to add that high carbohydrate feeding can precipitate respiratory failure in patients with chronic obstructive lung disease,[1,2] a group who are often weak and severely malnourished[3,4] and not infrequently present to surgeons. Feeding patients with severe chronic obstructive lung disease should therefore be of benefit,[5] but the metabolism of a high carbohydrate diet produces increased amounts of carbon dioxide for excretion[6]: assuming the patient requires 12·54 MJ a day, the difference between a high and a low carbohydrate diet can be 67 litres of carbon dioxide per day, which should normally be eliminated through the lungs by increased ventilation. The inability of patients with severe chronic obstructive disease to sustain higher minute ventilation rates owing to the increased work of breathing,[7] combined with blunted responses to hypoxia and hypercapnia,[8] and the adverse effects of hypercapnia on muscle contractility,[9] can precipitate overt respiratory failure.[1,2] Low carbohydrate, high lipid (polyunsaturated) regimens should be used if enteral feeding is required in patients with chronic obstructive lung disease.

1 Askanazi J, Wlwyn DH, Silverberg PA, *et al.* Respiratory distress due to a high carbohydrate load: a case report. *Surgery* 1980;**87**:596-8.
2 Covelli HD, Black JW, Olsen MS, *et al.* Respiratory failure precipitated by high carbohydrate loads. *Ann Intern Med* 1981;**95**:579-81.
3 Arora NS, Rochester DF. Respiratory muscle strength and maximal voluntary ventilation in undernourished patients. *Am Rev Respir Dis* 1982;**126**:5-8.
4 Hunter AM, Carey MA, Larsh HW. The nutritional status of patients with chronic obstructive lung disease. *Am Rev Respir Dis* 1981;**124**:376-81.
5 Goldstein SA, Thomaslow B, Askanazi J. Functional changes during nutritional repletion in patients with lung disease. *Clinics in Chest Medicine* 1986;**7**:141-51.
6 Gieseke T, Gurushanthaiah G, Glauser FL. Effects of carbohydrate metabolism upon dioxide excretion in patients with airway disease. *Chest* 1977;**71**:55-8.
7 McKerrow CB, Otis AB. Oxygen cost of hyperventilation. *J Appl Physiol* 1956;**9**:375-9.
8 Doekel RC, Zwillich CW, Scoggin CH, *et al.* Clinical semistarvation: depression of hypoxic ventilatory response. *N Engl J Med* 1976;**295**:358-61.
9 Vianna LG, Jenkins SC, Soutar SA, Lanigan C, Gray B, Moxham J. Effect of acute hypercapnia on quadriceps contractility. *Bull Eur Physiopathol Respir* 1986;**22**:162S.

Calcium changes in non-endocrine disease

Drs I R GUNN and R B PAYNE (Department of Chemical Pathology, St James's University Hospital, Leeds LS9 7TF) write: Dr Colin G Semple (25 October, p 1049) touched on the problem of interpreting serum calcium concentrations in severely ill patients with, for example, burns, sepsis, or pancreatitis. There is another reason for misleading reports of low ionised calcium values in ill patients. Measurements are currently adjusted to a standard pH of 7·40, usually by using a formula which "corrects" for CO_2 loss and pH change during transport and analysis of the specimen.[1] Ill patients are often acidaemic, so calcium is released from protein binding. Adjustment of ionised calcium to a standard pH in such cases is inappropriate, relegating the usefulness of the technique to the same status as that of total calcium measurement. In severely ill patients ionised calcium should be measured at the in vivo pH by taking simple precautions in handling the sample.[2] Low values should be interpreted with caution if there is severe hypoalbuminaemia.

1 Thode J, Fogh-Anderson N, Wimberley PD, Sørensen AM, Siggaard-Andersen O. Relation between pH and ionized calcium in vitro and in vivo in man. *Scand J Clin Lab Invest* 1983;**43**(suppl 165):79-82.
2 Thode J. Actual ionized calcium and pH in blood collected in capillary or evacuated tubes *Scand J Clin Lab Invest* 1986; **46**:89-93.

Zinc state in anorexia nervosa

Drs W W DINSMORE, DOROTHY MCMASTER, and J T ALDERDICE (Department of Medicine, Queen's University, Belfast BT12 6BJ) write: We agree with Dr C C Ainley and colleagues (18 October, p 992) that the assessment of body zinc state is difficult. The only real indicator of zinc deficiency is a favourable response to supplementation. But from our knowledge of zinc metabolism it is reasonable to assume that anorexics as a group are at risk of being zinc deficient and that all patients will require an adequate supply of zinc once an anabolic state is entered. We have some evidence that anorexics may have a decreased ability to absorb zinc.[1] After an overnight fast eight anorexics and eight matched controls were each fed a standard meal of 60 g ham, 60 g bread, 15 g butter, one cup of tea, and 100 ml orange juice to which was added 10 g glucose and 50 mg zinc (as acetate). The meal provided 50 g carbohydrate, 18 g protein, and 20 g fat. Blood was sampled via an intravenous catheter at 0, 30, 60, 90, 120, 180, and 240 minutes. The mean fasting serum zinc concentration of the anorexic group was 11·9 (SE 0·9) μmol/l (778(59) μg/l) and of the control group 12·4 (SE 0·4) μmol/l (811(26) μg/l), both of which were lower than the values reported by Dr Ainley and colleagues. The interesting finding, however, was that 90, 120, and 180 minutes after the meal the mean serum zinc concentration of the anorexic group was significantly lower than that of the control group. Thus although anorexics are probably in normal zinc balance in the premorbid state, the initiation of food restriction would lead to a lowering of zinc intake and the development of possible zinc malabsorption. The resulting negative zinc balance would in turn lead to further anorexia, and hence a self perpetuating state would be established. In view of this hypothesis and until further studies clarify the position it would seem reasonable to consider supervised supplementation on an individual patient basis.

1 Dinsmore WW, Alderdice JT, McMaster D, Adams CE, Love AHG. Zinc absorption in anorexia nervosa. *Lancet* 1985;i: 1041-2.

NHS waiting lists—the other side of the coin

Mr DICK GREENWOOD (Glenfield General Hospital, Leicester LE3 9QP) writes: General surgical facilities became available at England's newest teaching hospital 18 months ago. My "waiting list" is virtually nonexistent (largely reversed in that I await the patients' convenience). New routine outpatient referrals are seen within two weeks and urgent ones at the next clinic. Most are seen personally. Clinic and theatre sessions start on time and rarely over-run. Morale is high, there is no atmosphere of crisis, and the continual presence of medical students is a stimulating influence.

While juniors work a 1 in 3 emergency rota, junior staffing levels are lower than those available to me over the preceding 20 years. I can therefore concern myself more directly in supervising patients under my care, visiting them most days.

No doubt there are many reasons why my experience is at variance with that reported from other centres. It is, however, appropriate that the matter be put into perspective.

Arthritis and HLA-B27 in Papua New Guinea

Dr S A W SALFIELD (Department of Paediatrics, Rotherham District General Hospital, Rotherham S60 2UD) writes: Dr J E Richens and colleagues (8 November, p 1209) omit to mention the evidence that "tropical arthritis" in Papua New Guinea and other countries may be related to filariasis.[1-3] Infection with *Wuchereria bancrofti* is endemic in the coastal and low lying areas of Papua New Guinea such as most of the Sepik district. I described 19 patients in the Sepik district with "tropical arthritis" and noctural microfilaraemia.[1] The arthritis dramatically improved when they were treated with diethylcarbamazine citrate. The microfilaraemia was absent after treatment in 11 of the 12 patients treated in hospital. None of a control group of 50 hospital patients with other complaints had nocturnal microfilaraemia. Tropical arthritis in Papua New Guinea is probably not one disease. In the hot and humid low lying areas where the mosquito vectors proliferate filariasis is probably a prominent cause, but this is not likely in the authors' patients from the Eastern Highlands around Goroka, where filariasis is not common.

1 Salfield S. Filarial arthritis in the Sepik district of Papua New Guinea. *Med J Aust* 1975;**1**:264-7.
2 Das GC, Sen SB. Chylous arthritis. *Br Med J* 1968;ii:27.
3 Ismail MM, Nagaratnam N. Arthritis possibly due to filariasis. *Trans R Soc Trop Med Hyg* 1973;**67**:3.