Post-gastrectomy deficiency syndromes

Sir,

The short report by Dr M.J. Richardson on the detection of post-gastrectomy syndromes (January *Journal,* p.35–36) is a worthwhile contribution to this subject. We would like to comment on two aspects of the report; first on whether all gastric surgery, not just gastrectomy, warrants follow-up and, secondly, on the interpretation and importance of the findings.

The national prevalence of post-gastric surgery patients is difficult to establish but it is reported that some 35000 operations a year were being performed in the heyday of the gastrectomies in the 1950s. We reviewed our own small rural list of 1700 patients and found only three post-gastrectomy patients, but a further 22 patients who had undergone other gastric operations including seven vagotomy and gastroenterostomies and six vagotomy and pyloroplasties. These latter two groups are known to be at risk of anaemia[1] (albeit smaller than the risk following gastrectomy), and should, in our view be included. Richardson reports on 25 patients from a practice list of 9400 and includes, presumably, only post-gastrectomy patients. Problems with ascertainment and with differences in methods of surgery between our areas may exist.

The interpretation of haematological abnormalities in symptomless patients is a thorny problem. Surprisingly high rates of abnormality have been reported in addition to those quoted by Richardson; these include a vitamin B12 deficiency in 56 per cent of post-gastric surgery patients after 10 years, rising to 81 per cent after 15 years[2] and a raised alkaline phosphatase level in 27 per cent of post-gastric surgery patients.[3] However, levels just below the 'normal range' are common in the age group under consideration. For instance a haemoglobin level between 12 and 14 g dl^{-1} in males, though considered to be abnormal in one report,[4] is unlikely to cause symptoms of any kind.

After reviewing the literature, we concluded that our patients were unlikely to benefit from any but clinical and haematological examination unless they had symptoms which indicated investigation. Though no less than 12 of the 24 patients we saw (one remained unseen during the survey) had abdominal symptoms, including two with known recurrent ulcers, none had symptoms of anaemia, osteoporosis or other biochemical disturbance. We limited our review to a clinical examination, a blood count, and a determination of serum vitamin B12 and folate levels. Had we used the common lower limit of haemoglobin level, as did Richardson — 13.6 g dl^{-1} for males and 11.6 g dl^{-1} for females — eight of our patients would have been labelled anaemic. We felt it was unjustifiable to treat this group and our decision has been vindicated by no fall in values at three-year follow up. We set our lower limit at 12.0 g dl^{-1} for both sexes and treated those with haemoglobin levels of 9.5, 10.7 and 11.5 g dl^{-1}. One 80-year-old male had a low vitamin B12 level of 107ng l^{-1} but his haemoglobin level was 14.9 g dl^{-1} and mean corpuscular volume 91 μm^3. All his serum folate levels were normal.

In the light of our findings and those of Dr Richardson and others we remain convinced that the screening of these patients by clinical methods and with simple haematological examination is all that is required in the absence of symptoms. Furthermore, we consider it questionable practice to prescribe for marginally abnormal laboratory results in asymptomatic patients.

M.J. AYLETT
S.J. MILLER

Glendale Surgery
6 Glendale Road
Wooler
Northumberland

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Familial hypercholesterolaemia

Sir,

We are glad to see that our letter (February *Journal,* p.103) elicited a reply with references (June *Journal,* p.299). It is clear from these references that there is no evidence that treatment of hypercholesterolaemia prolongs life, as the authors themselves readily admit. Drs Lorimer and Mann also state that their strong advocacy of active therapy is based on clinical impression, sound theoretical grounds and strong circumstantial evidence. This is not good enough, especially if general practitioners are not furnished with the references to the original work.

Lorimer and Mann quote the Oslo trial as having shown an appreciable reduction in coronary heart disease, following advice on diet and smoking. It was pointed out by Pocock[1] that the significance of the mortality difference between the control and intervention groups hinged on a single unexplained sudden death in the control group. Results of the coronary intervention study,[2] quoted by Lorimer and Mann as evidence that cholestyramine therapy retards progression of coronary artery disease in familial hypercholesterolaemia patients showed no difference between the control and treated groups. At the end of this trial, definite progression of coronary arteriosclerosis (progression agreed upon by at least two of three panels made up of three angiographic experts each) was observed in 20 of 57 controls and 15 of 59 treated patients; this difference was not statistically significant. During the five-year study, seven patients in the placebo group and five patients in the cholestyramine group died — a difference readily attributable to chance.

Lorimer and Mann argue that the increase in the number of deaths from cancer in patients treated for hypercholesterolaemia was observed in only one trial (the World Health Organization clofibrate trial), ignoring several other studies.[3,4] They also suggest that there was no evidence of deaths from cancer in the active-therapy group of the Lipid Research Clinics Programme.[5] In fact, an editorial in the *British Medical Journal* expressed concern about six rare buccal/pharyngeal cancers in the cholestyramine-treated group.[6] While the overall numbers of deaths from cancer in the placebo and cholestyramine groups were equal, there were 11 incident cases and one death from all gastrointestinal cancers in the placebo group and 21 incident cases and eight deaths in the cholestyramine group. This was viewed with concern by the authors of the Lipid Research Clinics Trial, since cholestyramine was reported to be a promoter of colon cancer in animal studies.[5] While there is no reason for a cancer scare in cholestyramine users, it should not be forgotten that evidence is lacking to support the view that treatment with cholestyramine benefits anyone except the pharmaceutical industry. In the Lipid Research Clinics Programme, treatment with cholestyramine of 1600 men with type II hypercholesterolaemia for seven years showed no significant advantage over the placebo group, when a two-tailed probability t-test was applied. The observed difference of 1.6 per cent in absolute risk (in favour of cholestyramine) had confidence limits of −0.1 per cent

and 3.5 per cent. The cost of cholestyramine per death from myocardial infarction prevented was $2 068 300 and per deaths from any cause prevented, $9 307 500.[7] In the UK one fatal coronary event prevented with cholestyramine would cost about £1 million.[8,9]

JAMES S. McCORMICK
PETR SKRABANEK

Department of Community Health
University of Dublin
196 Pearse Street
Dublin 2
Ireland

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Wheezing in early childhood

Sir,

Dr Strachan's detailed study of wheezing in early childhood (April *Journal,* pp. 182-184) provides a useful account of the natural history of this common sign and gives the basis for a guardedly optimistic prognosis in younger children.

Perhaps it is not surprising that wheeze is so common in children, since relatively small absolute changes in airway diameter, due for example to virus infection, will lead to near closure with consequent wall oscillation.[1] The current tendency to equate all wheezing with asthma may not be appropriate. Certainly children aged seven years with a history of wheezing since starting school usually have asthma[2] and this diagnosis should be actively considered in all children with recurrent respiratory symptoms,[3] although asthmatics do not appear to present more frequently than controls before their illness becomes clinically overt.[4]

For general practitioners perhaps the best definition of asthma is that which responds to anti-asthma therapy, and we need clearer guidelines for the management of respiratory symptoms in the preschool child. Dr Strachan has provided the basis for this.

T.P. USHERWOOD

University of Glasgow
Department of General Practice
Woodside Health Centre
Barr Street
Glasgow G20 7LR

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Sir,

I was pleased to see the paper by Dr Strachan, (April *Journal,* pp. 182-184) on such an important topic. It set out to compare patient questionnaires with general practitioner records with regard to childhood wheezing. The main conclusions were that there was a surprising lack of overlap between the two methods of inquiry, and that the majority of children who develop wheezing in infancy appear to have a favourable outcome.

Other sources of bias should be considered regarding the conclusion that the two methods of inquiry were inconsistent. The author does not mention what the doctor actually diagnosed during those consultations for wheeze, and by implication, what the parents were told of the nature of the problem. In an audit on childhood asthma, we found a marked delay in the diagnosis of asthma and ascribed this to an unwillingness on the part of the doctor to record a diagnosis of asthma.[1] Our results and those of other workers showed that in the majority of cases those children who were diagnosed as asthmatic were likely to receive appropriate therapy (anti-asthma therapy).[2-4] In contrast, those undiagnosed asthmatic children consulting for respiratory symptoms were unlikely to receive appropriate therapy. These families are therefore unaware of their childs' asthma, and are unlikely to take much note of the respiratory symptoms because they have not been educated in this regard. I feel this factor will affect any questionnaire study because the parents are less likely to remember these consultations.

Another point I would like to make is that it has been well established that asthmatic children under ten years of age present for other reasons than wheeze. In our audit, before diagnosis, all but one of our 52 asthmatic patients had presented with cough at some stage, 25 per cent had not presented with wheeze and one third had presented with difficulty in sleeping. Inadvertent exclusion of children with these and other less stereotyped symptoms of asthma would also bias a study of this type. It would therefore be interesting to know how many of the 36 per cent population of wheezy children in the study by Dr Strachan had actually been diagnosed as asthmatic and what criteria were used to make the diagnosis.

Based on the conclusion that most wheezy children in infancy have a favourable outcome I would like to draw attention to a 20-year follow-up, by Blair[5] of 244 asthmatic children in an east London general practice. He found that there was no correlation between an early age of onset and severity of asthma either during the first five years of follow-up or with its persistence after 20 years. Of note, he found that after 20 years, only 28 per cent (68 patients), had been asymptomatic during the preceding two years. Asthmatic children therefore do not necessarily grow out of their asthma.

It is important therefore, to remember that although asthmatic children improve with age (in most cases), it still remains our duty as doctors to educate ourselves to make a positive diagnosis of asthma where appropriate, and to educate our patients about their illness. This will hopefully reduce the morbidity and mortality rates of childhood asthma.

MARK LEVY

173 Castle Road
Northolt
Middlesex UB5 4SQ

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