CORRESPONDENCE

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Caring for larger lists

SIR,—G N Marsh's paper about caring for larger lists may well represent a constructive vision for the future of general practice,[1] and we share some of its philosophy. We are concerned, however, that this profile will provide ammunition for our political masters to use against us.

Since the general practice charter of 1966 the thrust of both the Royal College of General Practitioners and the "grassroots" of general practice has been to improve our availability to patients. A smaller list size has become accepted as desirable. Marsh mentions seeing 10-12 patients each hour in caring for his suggested list of 4000 patients. In surveys patients have expressed their main wish as being to have more time with their doctor than they presently have.[2] Ten minutes is now becoming the norm, which seems incompatible with the proposed list of 4000 patients.

Delegating emotional and social problems to well trained professionals in the primary health care team is laudable. To imply that this is a quick and easy option, however, is not necessarily true. Several long, sensitive consultations may be required before a degree of trust and understanding is reached that allows any referral or delegation. Patients may feel rejected if referral is an immediate response to any mention of social or psychological components of their problem.

Marsh does not mention the availability of the team out of normal working hours. Though a "triage" nurse could well be trained to deal with or filter calls, our own list of 13 000 patients (6·5 partners, 2000 patients per partner) generates up to 35 calls directly to the duty doctor on a Sunday (daytime) and up to five calls during each night.[3] Such a rate of working is undesirable: doubling this would not improve our health or our patients' care the next day.

Furthermore, if their existence is to continue, no mention is made of how small practices that cannot afford to employ such a wide range of staff would be expected to perform.

Finally, we are not sure what evidence supports the statement that "seeing more patients increases knowledge resulting in fewer patients requiring hospital care." In our opinion, greater learning occurs when the workload is relaxed enough to permit reflection on all aspects of clinical situations seen. Consultations of six to seven minutes do not facilitate this.

We are concerned that this paper will be construed as suggesting what is possible now and could encourage not a reduction in workload by sharing but a further increase by generating political determination to reduce prematurely the number of general practitioners.

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1 Marsh GN. Caring for larger lists. *BMJ* 1991;**303**:1312-6. (23 November.)
2 You and your GP. *Which?* 1989 Oct:481-5.
3 Pitts J, Whitby ME. Out of hours workload of a suburban general practice: deprivation or expectation? *BMJ* 1990;**300**:113-5.

SIR,—I disagree with G N Marsh's conclusion that we should have lists of 4000 patients, but I do agree that paramedical staff should have a much larger role in primary health care.

If general practitioners are to take over the care of more and more elderly, psychiatric, and handicapped patients in the community we need far more help from visiting nurses, for example. There are plenty of these who are able and willing to do the work and to extend their role into making the first visit and making diagnoses. But will they do it at night? Many enthusiastic planners of community care talk gravely about the importance of providing full paramedical support for general practitioners, but so often this support melts away after 5 pm. The 24 hour contract in general practice is a dream for managers but a nightmare for the general practitioners themselves. We need paramedics who share our 24 hour commitment. Otherwise, given the choice between fewer patients and less sleep, any sensible doctor would choose fewer patients.

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1 Marsh GN. Caring for larger lists. *BMJ* 1991;**303**:1312-6. (23 November.)

SIR,—G N Marsh's article on caring for larger lists raises some interesting questions.[1] Delegation on the extended scale suggested is certainly possible, but is it desirable? Would an extended team prove any more cost effective and efficient than the existing nuclear teams? Would the patients like such an arrangement?

Delegation of tasks is essential in any practice, and the days when the paramedical members of the team were the doctor's handmaidens are, thankfully, long gone. The delegation of management tasks is widely practised. Delegation of clinical tasks can work correctly only when all parties understand the rules of access to the system; without this, identifying the person with responsibility for the episode of illness becomes difficult. Under the present system the person responsible is the general practitioner, who controls access to the other aspects of the health care system as gatekeeper. At present patients do not require any understanding of their illness to gain access to health care: they must first see "their" doctor. Delegation of clinical tasks on an extended scale breaks this principle, which has been widely acknowledged as extremely cost efficient. Would extended delegation prove more cost effective than the current system?

Marsh's view of delegation sees the general practitioner at the centre of a large team of health care professionals. Many of these professionals currently function independently and might be somewhat loath to readopt the handmaiden role again. How is this realignment to be addressed?

Marsh sees the task of a general practitioner with 4000 patients primarily as that of diagnostician and initiator of treatment for illness; this is a considerable narrowing of his or her role. Follow up of diagnosed conditions and non-medical problems would become the responsibility of others. I suggest that even with a large list this would narrow rather than broaden the experience of the doctor concerned. The difference between British general practitioners and deskbound doctors in other countries may lie in the general practitioners' broad professional involvement with their patients. If we narrow our remit the effects might be unpredictable. In particular, the loss of general practitioners' personal knowledge of their patients' lifestyles could prove fatal to our system of health care. Little of this knowledge is gained at the time of episodes of major illness. Perhaps people are entitled to come in and chat to us about nothing.

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1 Marsh GN. Caring for larger lists. *BMJ* 1991;**303**:1312-6. (23 November.)

Oral iron chelation is here

SIR,—George J Kontoghiorghes's editorial on oral iron chelation fails to provide an impartial review of this controversial subject because the author is the principal proponent of the oral iron chelator L1; he is an author of seven of nine references that he cites on the clinical use of this drug.[1]

The editorial implies that the oral iron chelator L1 is relatively safe and states that over 200 patients have received it, although it is not clear where the data have been published or how many patients have taken the drug for at least six months. The author concedes that L1 has led to one death and two cases of "transient neutropenia"; but the term transient neutropenia is disingenuous in at least one case—a woman who nearly died from marrow aplasia induced by L1.[2,3] On admission to hospital she had septicaemia (a blood count showed no neutrophils and a platelet count of 10×10^9/l) and required systemic antibiotic treatment and platelet support; no circulating neutrophils were seen for 17 days.[2]

If this woman had died L1 would have caused two deaths among 200 patients who had received the drug, a mortality of around 1%. This makes the statement that "the . . . benefits of treatment with

L1 . . . may outweigh the risks of its possible toxicity, and its introduction in such patients may now be appropriate" seem overenthusiastic. Certainly it is in stark contrast to an earlier editorial in the *Lancet*, which questioned the efficacy of L1 and drew attention to the unacceptable incidence of side effects, recommending that "this compound should no longer be given to patients."[4]

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1 Kontoghiorghes GJ. Oral iron chelation is here. *BMJ* 1991;**303**: 1279-80. (23 November.)
2 Hoffbrand AV, Bartlett AN, Veys PA, O'Connor NTJ, Kontoghiorghes GJ. Agranulocytosis and thrombocytopenia in patient with Blackfan-Diamond anaemia during oral iron chelator trial. *Lancet* 1989;ii:457.
3 Alter BP. Agranulocytosis and thrombocytopenia, Blackfan-Diamond anaemia and oral chelation. *Lancet* 1990;**335**:970.
4 Oral iron chelators [editorial]. *Lancet* 1989;ii:1016-7.

AUTHOR'S REPLY,—The references cited in my editorial are to my knowledge the major if not the only published reports describing clinical studies with L1. The lack of scientific evidence reported in the editorial in the *Lancet* two years ago[1] did not convince any of the centres to stop testing L1, and now the results of these studies overwhelmingly support the suggestion that L1 is a serious candidate for replacing desferrioxamine. In India, for example, a 35-55% reduction in serum ferritin concentration was observed in 52 patients taking L1 at a dosage of 50-100 mg/kg for one to one and a half years (M B Agarwal *et al*, third international conference on oral chelators in the treatment of thalassaemia and other diseases, Nice, November 1991). L1 has so far been taken daily for six months to two and a half years by 109 out of 230 patients who participated in the trials (papers presented at third international conference on oral chelators in the treatment of thalassaemia and other diseases, Nice, November 1991).[2] Details of these trials will be published in a special issue of the journal *Drugs of Today* next year. The death reported in the editorial may not have been caused by L1.[3,4] Similarly, many patients die while receiving desferrioxamine but the cause of death is not related to this drug.

I agree with N T J O'Connor that the agranulocytosis seen in two patients in the United Kingdom who were receiving L1 at a dosage of 105 mg/kg/day divided into two doses is a serious problem in relation to the development of this drug because of the potentially fatal consequences. The mechanism of this toxicity is not known but may be related to a combination of factors.[5] Weekly monitoring of the white cell count and the use of three or four divided doses each day, which will achieve lower peak serum L1 concentrations, may reduce the incidence of this idiosyncratic toxicity. It should be noted, however, that many other drugs in current use, such as clozapine, penicillamine, and even desferrioxamine in a few cases, have also been reported to cause agranulocytosis.[2,5,6] In the absence of an alternative effective treatment such drugs will continue to be given to patients because of the high benefit to risk ratio. Similarly, chronic transfusional iron overload will progressively cause 100% mortality in the absence of chelation.

As desferrioxamine is not widely used and no other chelator is known to be cheap, orally effective, and relatively non-toxic L1 should be seriously considered as an alternative drug for such patients.

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1 Oral iron chelators [editorial]. *Lancet* 1989;ii:1016-7.
2 Kontoghiorghes GJ. Advances in oral iron-chelation in man. *International Journal of Haematology* (in press).
3 Olivieri NF, Koren G, Freedman MH, Roifman C. Rarity of systemic lupus erythematosus after oral iron chelator L1. *Lancet* 1991;**337**:924.
4 Berdoukas V. Antinuclear antibodies in patients taking L1. *Lancet* 1991;**337**:672.
5 Young GAR, Vincent PC. Drug-induced argranulocytosis. *Clinics in Haematology* 1980;**9**:483-509.
6 Baldessarini RJ, Frankenburg FR. Clozapine, a novel antipsychotic agent. *N Engl J Med* 1991;**324**:746-54.

Coronary heart disease

SIR,—Neither *The Health of the Nation* nor Hugh Tunstall-Pedoe's response[1] addressed one major cardiovascular cause of morbidity and mortality—chronic heart failure—which, in the United Kingdom, is usually caused by coronary artery disease.

The limited epidemiological data available suggest that chronic heart failure is becoming increasingly common and affects up to 500 000 people in the United Kingdom.[2,3] The annual incidence in the older age group is similar to those for myocardial infarction and cerebral infarction,[4] conditions that attracted considerable comment in the secretary of state's document and subsequent responses[1,5,6] (table I).

Furthermore, chronic heart failure causes huge morbidity and mortality (more so than myocardial infarction). It accounts for 5% of all adult medical and geriatric admissions (that is, up to 150 000 admissions a year in the United Kingdom). The annual rate of admission to hospital may approach 45%, with each admission lasting for about eight days. The economic implications are obvious.

Many admissions in patients with chronic heart failure might be preventable. Angiotensin enzyme inhibitors reduce the need for admission for worsening heart failure. The annual rate of admission in the studies of left ventricular dysfunction (SOLVD) was about 2% for patients with thromboembolic events and about 4% for those with pulmonary infection.[7] Anticoagulation and vaccination (pneumococcal/influenza) might help reduce these admissions (these might be areas for future study).

The dreadful mortality due to chronic heart failure also deserves mention. Recent studies have confirmed the dismal prognosis reported in the Framingham study, in which half of patients died within five years of diagnosis despite conventional treatment with diuretics and digoxin (mortality worse than that for stage II breast cancer and similar to that for stage II squamous cell carcinoma of the lung).[7,9] These studies have also, however, shown that treatment with angiotensin converting enzyme inhibitors can reduce mortality in chronic heart failure—by 30% at one year in severe chronic heart failure and by 16% at four years in mild to moderate chronic heart failure.[7,9] Angiotensin converting enzyme inhibitors have been shown to be easy to use and relatively free of adverse effects in these patients.[7,9] Furthermore, they are very cost effective (table II).

Chronic heart failure is therefore a major public health problem that has been neglected in the United Kingdom. As few as one fifth of patients with chronic heart failure in the United Kingdom are treated with an angiotensin converting enzyme inhibitor. More effort must be made to ensure that chronic heart failure is recognised and treated. Issues such as the earlier detection and prevention of progression of left ventricular dysfunction need to be discussed, particularly in the light of the positive findings in the prevention arm of the studies of left ventricular dysfunction.

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TABLE I—*Age adjusted annual incidence of myocardial infarction, cerebral infarction, and chronic heart failure/1000 at 30 year follow up in Framingham study*[6]

	Myocardial infarction		Cerebral infarction		Chronic heart failure	
Age (years)	Men	Women	Men	Women	Men	Women
35-64	6	2	1	1	3	2
65-94	13	7	5	4	10	8

TABLE II—*Cost effectiveness of treatments*

Treatment	Problems prevented per 1000 years of treatment or *per 1000 patients treated
Treatment of mild hypertension	1-2 strokes
Lipid lowering treatment (gemfibrozil)	2-3 cardiac events
Intravenous β blockade after myocardial infarction	6 deaths*
Oral β blockade after myocardial infarction	17 deaths
Intravenous streptokinase after myocardial infarction	25 deaths*
Enalapril for severe chronic heart failure	160 deaths
Enalapril for mild or moderate chronic heart failure	16 deaths 116 admissions

1 Tunstall-Pedoe H. Coronary heart disease. *BMJ* 1991;**303**: 701-4. (21 September.)
2 Sutton GC. Epidemiologic aspects of heart failure. *Am Heart J* 1990;**120**:1538-40.
3 Smith WM. Epidemiology of congestive heart failure. *Am J Cardiol* 1985;55:3-8A.
4 Kannel WB, Cupples A. Epidemiology and risk profile of cardiac failure. *Cardiovascular Drugs and Therapy* 1988;**2**:387-95.
5 Dennis M, Warlow C. Strategy for stroke. *BMJ* 1991;**303**:636-8. (14 September.)
6 Wolff PA, Kannel WB, Cupples LA, d'Agostino R. Update on epidemiology of stroke. In: Rose FC, ed. *Stroke: epidemiological, therapeutic and socioeconomic aspects.* London: Royal Society of Medicine Services, 1986. (Royal Society of Medicine Services International congress and symposium series No 99.)
7 SOLVD Investigators. Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure. *N Engl J Med* 1991;**325**:293-302.
8 CONSENSUS Trial Study Group. Effects of enalapril on mortality in severe congestive heart failure: results of the cooperative north Scandinavian enalapril survival study (CONSENSUS). *N Engl J Med* 1987;**316**:1429-35.
9 Cohn JN, Johnson G, Ziesche S, Cobb F, Francis G, Tristani F, *et al*. A comparison of enalapril with hydralazine-isosorbide dinitrate in the treatment of chronic congestive heart failure. *N Engl J Med* 1991;**325**:303-10.

AUTHOR'S REPLY,—*The Health of the Nation* did not set out to target all major causes of mortality and morbidity. The key areas chosen had to be major causes of premature death or avoidable ill health; areas where effective interventions are possible; and, thirdly, and most relevant to J McMurray and H J Dargie's argument, "ones in which it is possible to set objectives and targets and monitor progress towards achievement through indicators."[1] McMurray and Dargie make an impassioned plea for recognition of chronic heart failure but admit that the bulk of the problem occurs in those over the age of 65. The uphill task that they have in getting chronic heart failure recognised as a key area is shown by the routine mortality statistics for England and Wales (table), in which it seems to account for only one death in 1000 below age 65.[2]

Heart failure shares the fate of other conditions such as hypertension, hypercholesterolaemia, cigarette smoking, and ventricular fibrillation, which contribute either as risk factors or as pathological mechanisms. They are likely to be left off the death certificate and, if they do appear, will be coded as the cause of death only if no specific underlying cause is coded with them. Such factors or processes cannot be studied from routine