George Swift Lecture

Sir,

I read with interest Dr Julian Tudor Hart's comments on 'Division of labour in general practice' (February *Journal*, pp. 63-68).

He recognizes the capabilities of practice nurses to act autonomously within the limitations of training and experience. Dr Hart says 'Encouraged to act autonomously, nurses become excellent clinicians'. However, I believe that this view needs further amplification. One needs to detail the areas in which encouragement can be given, and as a starting point I would suggest the following:

1. Provision of a good consulting/treatment room with up-to-date facilities and equipment.
2. An appointment system which is sensitive to the needs of both the practice nurse and the patients, practice nurses having flexibility to plan their own workload. The time spent with each patient should be determined by the sister with reference to the patient's needs.
3. Easy communication with the general practitioner for referral and advice.
4. Involvement in practice policy making.
5. The opportunity and financial support to attend professional courses.
6. Full Whitley entitlement in terms of salary, holiday allowance and provision of pension, as enjoyed by National Health Service colleagues.

By making these provisions practice nurses then have both the professional and financial status commensurate with their responsibilities. The practice nurse becomes part of a team and thereby benefits from the support of that team which itself is strengthened by the involvement of other committed professionals.

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Familial hypercholesterolaemia

Sir,

Professor McCormick and Dr Skrabanek have commented (February *Journal*, p.103) on the booklet, *Familial hypercholesterolaemia — notes for general practitioners* which was an insert in the November issue of the *Journal*. They were dismayed to find the notes unreferenced and they suggest that lowering cholesterol levels is associated not only with a decrease in the number of deaths from coronary heart disease but also with an increase in the number of deaths from other causes, especially cancer.

The booklet was published by the Simon Broome Heart Research Trust and was not intended to be a comprehensive reference work. The aim was to increase awareness of a relatively common disorder quite distinct from all other forms of hypercholesterolaemia in terms of inheritance (autosomal dominant), mechanism (defect of low-density lipoprotein receptors),[1] prognosis (untreated, 25 per cent of men with this disorder may be expected to have died by the age of 50 years)[2] and often clinical findings (tendon xanthomata).[3] As physicians with a special interest in abnormalities of lipid metabolism we know that many people with familial hypercholesterolaemia remain undiagnosed both in the community and in hospital. We hope that the booklet will help to remedy this situation and so help people with this condition to receive the appropriate dietary advice, genetic counselling and where necessary, drug treatment. Appropriate references are available in original papers and reviews.[1-3]

It is readily acknowledged that as for many other conditions (for example maturity onset diabetes or non-insulin dependent diabetes) there is no conclusive evidence that treatment will prolong life. We are presently involved in a joint research project (also sponsored by the Simon Broome Heart Research Trust) aimed at assessing our clinical impression that life expectancy has substantially improved since the fairly widespread introduction of drug therapy which is capable of achieving effective long-term cholesterol reduction. Our strong advocacy of active therapy is also based on sound theoretical grounds and strong circumstantial evidence: numerous epidemiological studies (within-country longitudinal studies as well as cross-cultural comparisons) show that low cholesterol levels are associated with reduced rates of coronary heart disease without an increase in other conditions.[4] Animal studies show the potential reversibility of atherosclerosis associated with hypercholesterolaemia.[5] The clinical trials quoted by Professor McCormick and Dr Skrabanek, and several other trials which they did not mention, have no relevance to patients with familial hypercholesterolaemia since the great majority of volunteers did not have this condition.

Only three of the trials included appreciable numbers of volunteers with raised levels of cholesterol, some of whom might have been expected to have familial hypercholesterolaemia.

In the Oslo trial dietary advice to lower cholesterol levels and advice to stop smoking were associated with an appreciable reduction in coronary heart disease without any increase in the number of deaths from cancer or other causes.[6] In the multiple risk factor intervention trial mortality from all causes was higher in the intervention group than in the control group.[7] This trial is difficult to interpret because there is so little difference between risk factor changes in the intervention and control groups. However, it is of interest to note that when the hypertensive patients were excluded, the overall results were remarkably similar to the Oslo findings, leading to the suggestion of an adverse effect associated with the hypotensive drug therapy.[8] The Lipid Research Clinic's trial showed substantial reductions of coronary heart disease in association with the lowering of cholesterol levels by cholestyramine therapy.[9] The difference in total mortality between the active-therapy group and the placebo group was less impressive because of seven deaths from accidents and violence. It is hard to imagine the latter being due to anything other than chance. There was no increase in deaths from cancer in the active-therapy group. Apart from the World Health Organization trial[10] (in which increased mortality seems likely to have been associated with a specific effect of the drug clofibrate) the suggestion that the lowering of cholesterol levels might cause cancer is based on the controversial premise that very low cholesterol levels increase the risk of cancer. Even on treatment, cholesterol levels in patients with familial hypercholesterolaemia tend to remain higher than average and therefore they are not at increased risk of cancer as a result of the treatment.

Conclusive proof that lowering cholesterol levels decreases mortality from familial hypercholesterolaemia could only come from a large randomized clinical trial of patients with this condition which would pose ethical problems. Evidence that cholestyramine therapy retards the progression of coronary artery disease in familial hypercholesterolaemia patients was published last year.[11,12] Indeed the evidence in favour of energetic therapy in familial hypercholesterolaemia is now stronger than that in favour of treating non-insulin dependent diabetes, a condition for which few would encourage withholding therapy.

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