The process of determining population needs is also in a primitive form and must be encouraged to evolve. The first contracts will of necessity be based on "met demand" as a proxy for "need." The more refined definition of need, based on epidemiological principles, will evolve over time.

Survival of the fittest seems to be a fundamental principle of the white paper's philosophy. The competition should, however, be between provider units and not between providers and commissioning authorities; if commissioning authorities are vested with real powers in making informed choices and monitoring performance then there are real opportunities for incorporating measures of clinical effectiveness within the contract framework outlined in the white paper.

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Steroid aerosols and cataract formation

SIR,—Dr Martin B Allen and colleagues suggest a link between cataract formation and inhaled steroids in asthmatic patients.[1] We have come to similar conclusions as a result of a clinicopathological study of lens capsules from patients taking steroids.[2,3] Twelve patients were identified whose cataracts may have been related to inhaled beclomethasone dipropionate; eight of these had not received any other steroid treatment. The age range was from 56 to 79 years. Histological changes in the lens capsules were identical with those in patients taking systemic steroid.[4] We agree that this relation requires further study.

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Interstitial laser hyperthermia

SIR,—Dr A C Steger and colleagues reported using a bare quartz fibre and laser energy to produce interstitial hyperthermia.[1] We agree that this is an encouraging development but it needs further evaluation.

We dispute the fact that this is a hyperthermic treatment because Matthewson *et al* reported temperatures of up to 100°C close to the fibre tip, which cause cavitation and charring.[2] For hyperthermic damage to occur to malignant cells they need to be maintained at temperatures greater than 42·5°C for longer than 670 seconds, but temperatures greater than 45°C damage normal cells.

We are currently investigating the use of an artificial sapphire probe with a neodymium yttrium aluminium garnet laser to produce hyperthermia. This has the advantage that the tip is not damaged by tissue contact, and there is no charring of the tissues because the temperatures produced are lower. A power of 3 W applied to the liver produces temperatures of 42-44°C 5 mm away from the probe. Theoretically this could be used to treat tumours up to 1 cm in diameter.

Initial animal experiments to study the effect of this probe on subcutaneous and intrahepatic tumours have shown that tumour necrosis can be produced, but even in a controlled situation treating small tumours it is difficult to produce total tumour necrosis. This may be improved by applying several probes for longer exposures.

In view of these findings more experimental work is required before this technique is widely used in clinical practice.

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Effect of homoeopathic treatment on fibrositis

SIR,—Homoeopathic physicians, no doubt, will see the positive outcome of treating fibrositis with homoeopathic *Rhus toxicodendron* 6x[1] as a vindication of their clinical practice. However, caution is advised. We would like to offer two points of constructive criticism—the one related to the crossover study design, in which the carryover effect of the homoeopathic medicines could have clouded the outcome, and the other regarding standardisation of the pharmaceutical product used in the study.

Firstly, what was the run in period before introducing the homoeopathic remedy in the first positively treated group? Equally important would be knowing the washout period in the crossover group. Homoeopaths have argued over the centuries that homoeopathy has a long carryover effect.[2] There is no general agreement as to its length, but it certainly does not relate to any pharmacological activity such as the half life of the homoeopathic ingredients used.

Secondly, the question arises whether this clinical study can be repeated. Although some details were given of the preparation of the homoeopathic remedy, starting from a mother tincture provided by a homoeopathic pharmaceutical manufacturer, these details are insufficient. What is the precise nature of the starting materials of this mother tincture? This is something that our own research team suggests is particularly important. The standardisation of homoeopathic medicines must be called into question. This is relevant because of the likely review of product licences for homoeopathic medicines to be undertaken by the Medicines Commission in Britain, by the Food and Drug Administration in the United States, and by the European Commission in Strasbourg. Reference to the individual national pharmacopoeias shows that there is no generally recognised standard for the mother tincture. The Blackie Foundation Trust is attempting to investigate this question by sponsoring a PhD student in the pharmacology department of Professor Paul Turner. We have had considerable difficulty in recruiting a suitable student in part because of the controversial nature of homoeopathy and the adverse criticisms that it has received as a result of recent publications.[3]

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Is abdominal aortic aneurysm familial?

SIR,—The results of Mr Jack Collin and Ms Jackie Walton suggest that abdominal aortic aneurysm is a familial disease.[1] The case for a familial tendency in this disease is now very strong, and the mode of inheritance has been investigated by Tilson and Seashore.[2] They concluded that in over half of the cases in families where two or more first degree relatives were affected the pattern of inheritance was consistent with a dominant X linked gene. A study in animals has also indicated that the disease is most commonly inherited in this way.[3] Therefore, we agree with Mr Collin and Ms Walton that a screening programme aimed at male first degree relatives of patients with aneurysm is likely to be productive, but we believe it important to know whether the aneurysms that they found were sizable and whether they were treated surgically. Confirmation on these points would strengthen the case for such a screening programme.

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AUTHORS' REPLY,—Drs H J Scott and T R Cheatle are correct in stating that the case for abdominal aortic aneurysm being familial is now strong,[1] but they would be ill advised to give much credence to the suggestion that a dominant X linked gene is responsible for the disease in humans.[2]

If we assume a gene prevalence of 5%,[3] 9·5% of women would have the gene on one X chromosome and 0·25% on both X chromosomes. If such a gene were dominant abdominal aortic aneurysm would be almost twice as common in women as in men. If, on the other hand, the gene was expressed only in the absence of a normal X chromosome the disease would be 20 times more common in men than women. The relative risk of death from abdominal aortic aneurysm in men declines progressively from 11 times that in women at age 60-64 to three times at age 85-89.[4] We suggest, therefore, that the gene for abdominal aortic aneurysm is autosomal but the consequences of possessing it are, like so many other human ills, delayed in women.

The four patients on whom we performed