reported by Super and colleagues, one had completed their family and another five did not request parental diagnosis. This contrasts with the 12 heterozygous couples detected through our antenatal screening programmes,^{2,3} all of whom have requested prenatal diagnosis.

It is impossible to measure the theoretical effectiveness of cascade testing by simple extrapolation. It is necessary to create models with family size distributions and alternative testing strategies. Our projections, using such modelling, suggest that less than 25% of heterozygous couples be detected by cascading to the second cousin level.⁴ In contrast, we have already shown that over 50% of heterozygous couples can be detected by either of the two major forms of antenatal screening.^{2,3}

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General practitioners should be involved

EDITOR,—Cascade screening for carriers of the cystic fibrous gene has the tempting appeal, particularly when done before conception, of detecting clusters of carriers in particular families.¹ Unfortunately, couple screening, in which couples of whom only one partner is a carrier are identified as being at low risk,² would undermine any cascade screening programme. Reasonably, in her commentary on the study of cascade screening¹ Theresa Marteau has brought to light various concerns about a cascade approach to screening. Other concerns exist—for instance, the impact on family and kinship networks of introducing the possibility of “family illness” through a cascade of screening initiated from an “affected” carrier. In other settings more distant relatives have been shown to be disinclined to undergo, or are uninformed about, carrier testing.³

The alternative situation, in which a carrier index subject blocks dissemination of genetic information to the extended family, also needs to be considered. This could later give rise to friction within the family—for instance, if a sibling later discovered her carrier status after the birth of an affected child. Other members of the family could be contacted without the confidentiality of the index subject being broken only if there was a complementary population screening programme based in general practice.

For an effective preconceptional programme the primary care team needs to be informed and involved. M Super and colleagues and Jean Livingstone and colleagues do not elaborate on the role of the primary care teams in their screening programmes.^{1,2} Unless the health professionals themselves appreciate the issues they are not able to help patients make an informed choice. Even when adequately informed, patients could take days, months, or even years to decide whether to be screened.

Screening programmes based in secondary care are unlikely to offer carrier screening at the time when patients feel able actively to seek it.⁴ This reinforces the argument that population based genetic screening and counselling should be easily available in general practice.⁵ This cannot happen, however, without adequate assessment of the

information and skills needed by the primary care team.

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Medical aspects of scuba diving

Standards for diabetic divers are workable

EDITOR,—The diving medical committees of the British Sub-Aqua Club, the Scottish Sub-Aqua Club, and the Sub-Aqua Association—organisations mentioned in J J W Sykes's review of scuba diving¹—have combined to form a national diving medical committee. This committee acts as the responsible body for amateur scuba diving medicine in Britain, providing medical advice for divers of whatever diving organisation and carrying out research into aspects of diving medicine.

The article questions whether standards for divers with diabetes will prove workable. These standards have been published and are available from the headquarters of the diving organisations mentioned above.² An annual record is kept of all diabetic divers (now about 60) together with details of their treatment, the diving they undertake during the year, and any possible side effects related to their diabetes. Seven hundred and two person dives have been logged in two years, with 220 being in the depth range 0-10 m, 252 at 11-20 m, 165 at 21-30 m, and 65 at over 30 m. Thirty one of these dives entailed decompression stops. Only two incidents of slight hypoglycaemia after a dive have been reported. So far the system has proved workable and has allowed many diabetic people to enjoy scuba diving safely. The diving medical committees in the Netherlands and Denmark have expressed strong interest in allowing diabetic people to dive under these rules.

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Decompression sickness may be due to paradoxical embolism

EDITOR,—J J W Sykes states that “the proposition [that paradoxical gas embolism across a patent foramen ovale is a mechanism for certain types of decompression illness] remains controversial”¹ and supports this statement with a single reference.² In this unreviewed letter, Cross *et al* stated that they were unable to confirm in a small non-blinded and uncontrolled study (19 patients) the results obtained in two considerably larger and better designed studies also quoted by Sykes. Each was published as a paper after peer review and showed an excess prevalence of shunts in divers who had neurological decompression illness. Since then many studies have been published after peer review to support the role of patent foramen ovale

in decompression illness, but the limit of five references in letters in the *BMJ* prevents me from citing them all.

It has been pointed out that contrast echocardiography does not have 100% accuracy for detecting shunts and that if Cross and colleagues had missed a single shunt that would explain their contrary findings.³ One of the affected divers in whom they had failed to show a shunt has since had a second contrast echocardiogram, which Cross and colleagues concede showed an obvious and large shunt. If this subject is moved from the no shunt to the shunt group, the findings accord with ours. It is unacceptable that an isolated incorrect observation should continue to be used to cast doubt on a theory that is supported by overwhelming evidence.

Sykes's scepticism is particularly surprising. He has no doubt that arterial gas embolism resulting from pulmonary barotrauma can cause neurological decompression illness. Why not paradoxical gas embolism? He was chairman of a meeting of the Diving Accident Working Group of the Medical Research Council Decompression Sickness Panel at which doubts were expressed about the first reports of association between shunts and decompression illness. A colleague and I therefore agreed to perform a blind controlled replication study at a centre remote from my own under supervision by members of the group and using subjects and equipment they provided. The results corresponded with those that we had previously published.^{4,5}

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Riding for people with disability

EDITOR,—J C Chawla mentions the growing interest in sporting activity for disabled people and the support provided by the medical profession.¹ I endorse all the benefits claimed for people with physical disabilities and those with learning disabilities, but I take issue with the comment that riding is not advisable for people who have difficulties in communication or behavioural disorders.

The Riding for the Disabled Association, now in its 25th year and a worldwide organisation, has wide experience of riders with learning difficulties. Indeed, in our census analysis of 1993 they accounted for about half of our 25 000 riders and carriage drivers (table). We find that riding is enormously beneficial to people with learning difficulties, whose confidence, coordination, and communication skills are greatly improved. In several cases the first verbal communication of people with autism has been with their horse. Improvement may be slow, but the relationships

Numbers of people with learning disabilities who rode or drove a carriage with Riding for the Disabled Association by age, 1993

	<18	≥18
Moderate learning difficulty	3443	2112
Severe learning difficulty	4013	3044
Autism	645	296
Mental illness	280	618
	8381	6070