

encourage reattachment—for example, expanding gases such as sulphurhexafluoride, or longer acting materials such as silicone oil.¹² Sometimes vitrectomy will be the primary treatment.

Typical premonitory symptoms or a profound reduction in visual acuity should alert a general practitioner or an optician to the possibility of impending or actual retinal detachment. Neither, however, can be expected to make the diagnosis with certainty. Retinal breaks are usually in the periphery of the retina and hard to see, and even with good pupillary dilatation viewing the retina of the myopic fundus with a direct ophthalmoscope is extremely unreliable. A simple confrontation field test may, however, show a field defect, and if the macula is detached visual acuity will be profoundly depressed. The patient must be referred promptly to hospital (within 24 hours), and the hospital must respond quickly. The examination demands binocular indirect ophthalmoscopy, scleral depression, and three mirror gonioscopy. The unaffected eye must also be examined for lesions that may need prophylactic treatment. These examination skills will often be beyond the scope of the ophthalmologist in the early stages of training. Prompt diagnosis and treatment are essential in managing retinal detachment.

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close. The polygenic theory also predicts that the offspring of affected parents would be at a similar risk to siblings. The data are now available to test the theory.²⁻¹¹

Most of the information is on the commoner parental lesions—aortic stenosis, patent ductus arteriosus, coarctation of the aorta, atrial and ventricular septal defects, pulmonary stenosis, and tetralogy of Fallot. The two largest studies found that 2.5-5.0% of children also had heart defects, a percentage marginally higher than for siblings.^{3, 4} Other reports give recurrences of up to 16%, but a bias in ascertainment is likely. Geographical, racial, and environmental factors were not apparent, and only 30-60% of parents and children had the same lesion.^{3, 4, 9, 11, 12} This poses questions about pathogenesis and whether the heritability of particular heart lesions should be considered in isolation or as part of well defined groups.¹² Similar questions have been raised in other species, particularly the dog, in which the prevalence and distribution of congenital heart disease are similar to those in man.

Pure dog breeds can be developed at some genetic loss, and for some breeds this has meant an increase in the incidence of congenital heart disease. In the Keeshond the incidence is 62.5 for every 1000 live births; Fallot's tetralogy is the commonest lesion, but the range stretches from simple absence of papillary muscle to end stage tetralogy. The conclusions from embryological¹³ and breeding studies¹⁴ were that the pattern of transmission was best explained by a polygenic mechanism and that a disturbance in growth rate of the conotruncal region with secondary haemodynamic changes could explain the range of defects.

Whether the same always holds for man is not certain, but there is evidence from twin studies that altered haemodynamics in utero are one important cause of congenital heart lesions.¹⁵ Other questions remain unanswered. The first is to determine the risk of transmission by studying live births and then antenatal tests¹⁶ and fetal¹⁷ and neonatal deaths. The second is whether the sex of the parents and the severity of their lesion will be important; such factors influence the risk of recurrence for other malformations. The last is to recognise those families that have a higher than average risk of recurrence for heart defects and to identify the factors responsible. One thing is sure—even for families with clusters of affected members the distribution of defects does not usually fulfil the criteria for strict Mendelian inheritance.

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The heritability of congenital heart disease

Six in every 1000 children born alive have a structural heart defect. In some it is part of a syndrome, which may result from chromosomal imbalance. Alcohol, some drugs, and rubella in pregnancy may be responsible for the defect, and the prevalence is also higher in children whose mothers have phenylketonuria or insulin dependent diabetes. Single mutant genes are seldom responsible, and for 80% of heart malformations there is no individual explanation. In this group there is a small increased risk to siblings and second degree relatives, although the affected relatives may not have the same lesion. The best explanation is that two or more genes of small effect, not necessarily individually deleterious, influence and disturb growth and development. In practice the figures derived from the polygenic theory of inheritance¹ and those from family studies turn out to be remarkably

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Called to account

Consultants who are cavalier about patients' complaints had better watch out; they could find themselves called to account before a parliamentary select committee. On 24 March Dr James Thirkettle, a consultant physician at Crawley Hospital in East Sussex, will appear before the Select Committee on the Parliamentary Commissioner for Administration, which shadows the ombudsman. Dr Thirkettle will be questioned on why a patient's family was not informed of his sudden and fatal deterioration in time to visit him while he was still conscious and why over the next six months he "fobbed off" written and telephoned requests for an explanation.

According to the ombudsman's report, Dr Thirkettle told him that a response would have led to "protracted correspondence" and that he did not have time to go through the clinical notes. But the invitation to appear before the committee is not one he will be able to decline. Select committees have the power to summon individuals to appear before them, and anyone who refuses will be in contempt of Parliament.

In December the committee summoned Mr G D Wood, a consultant dental surgeon at Arrowe Park Hospital, the Wirral, who on three occasions took more than four months to reply to patients' complaints. In two of the cases, when he eventually replied, he merely said: "I have no comment to make on the content of the letters sent by the respective authors." Mr Wood told the committee he fully accepted the ombudsman's criticism of him and recognised that he had failed to respond speedily to the complaints. He gave an undertaking that he would do everything in his power to ensure that nothing similar occurred in the future.

Parliamentary authorities believe the power to summon a consultant has been used only once before, some years ago. But committee members say it is likely to be exercised again in the future. The committee cannot impose any sanction on the consultant; its chief weapon is embarrassment. The cases it examines are chosen from the ombudsman's reports; its main role is to try to find out where things went wrong and to prevent a recurrence.

The ombudsman and the committee are constrained by terms of reference which allow them to investigate cases of alleged maladministration but not mistakes of clinical judgment. Administrative mistakes were made in both these cases. In one case relatives were not told of the serious deterioration in the patient's condition. In the other dental treatment was carried out on mentally handicapped children without proper parental consent. But had the consultants apologised or explained—or, at the very least replied—they would probably not have found themselves subjected to an embarrassing grilling before a parliamentary committee whose proceedings are open to the public and the press.

In both these cases district health authority administrators

investigating the complaints cited difficulties in calling the consultant, an employee of the regional health authority, to account. When it comes into force later this year the Hospital Complaints Procedure Act 1985 will ensure that complaints are dealt with at a high level and will lay down a code of practice which should make the procedure for handling complaints more efficient. But for the consultant who thumbs his nose at the idea that he should be accountable to the consumers of his services the threat of a parliamentary appearance seems a useful sanction.

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Social skills training

One of the few consistent findings in patients with mental health problems is that they have poor social skills. We all need these skills to relate to each other and get through our lives. Deficiency in them is an important factor in patients with schizophrenia, mental handicap, depression, social anxiety, addictions, and psychosexual disorders as well as in those who have the particular problems of childhood, adolescence, and old age.¹ People experiencing a wide range of problems—such as loneliness, divorce, and disease—also tend to have poor social skills.^{2,3} Poor social skills are important too in those who are not ill or under any particular strain—for instance, ineffective communication skills, which are not uncommon in doctors and nurses and affect the outcome of treatment.^{4,6} It is against this background that social skills training has developed and been used widely for both helping patients and training professionals.¹

The idea of social skills training is simple. Within the constraints of a person's biological endowment social skills are learnt—either by direct experience or vicariously through models. Good skills make people better able to cope, and bad skills lead to problems. As skills are learnt they can be taught to those that lack them. The first comprehensive theoretical model of social skills was put forward by Argyle and Kendon⁷ and developed by Argyle and others.⁸ The model draws on an analogy with serial motor skills and consists of three main sequential stages—perception, cognitive translation, and performance—and a feedback loop from the environment that completes the cycle. According to this model, all social behaviour is constantly monitored and adjusted.

The idea of social skills training is educational rather than medical: instead of a diagnosis being made and treatment prescribed deficiencies in knowledge and performance are assessed and training provided. Firstly, a skill is broken down into its verbal and non-verbal elements. Secondly, the skill is demonstrated by one or more models. Thirdly, the trainee practises the new skill in role play. Fourthly, the trainee is coached and given video feedback on his performance and reinforcement for improvements. Finally, the trainee practises the new skill at work or socially and keeps a record of the results.

The most broadbased social skills training includes modules on observation, listening, non-verbal communication, asking questions, self disclosure, conversation management, expressing feelings, use of social routines and linguistic devices, and more general themes such as self presentation.⁸ More specifically programmes include assertion training,