A total of 150 injuries were x-rayed and glass fragments were seen on 29 films. In 16 cases these had not been suspected clinically. Most fragments were removed immediately but the few which could not be found did not complicate wound healing and some subsequently worked their way out.

It is not departmental policy1 to give prophylactic antibiotics for minor wounds and these were given for two particularly dirty lacerations only, both of which healed well. The remaining 181 children with uncomplicated injuries were followed up after two days to assess healing and only 6 needed antibiotics—an infection rate of 3.3%.

Tetanus toxoid was needed by 103 children; 10 received this as part of their preschool booster and 7 as part of their primary course, with humotot if necessary.

Discussion

One quarter of the injuries involved breakage of architectural glass. This could be prevented2 3 if more stringent safety glass regulations were to stipulate that at least the vulnerable lower half of glass doors, french windows, and ordinary windows4 5 adjacent to play areas should be constructed of safety glass. A less expensive measure would be the application of plastic safety film. All glass doors should be marked with transfers to indicate their position. Glass should not be used in doors at the bottom of staircases.

The use of unbreakable materials for 'pop' bottles would greatly reduce injuries. The public should be encouraged to dispose of glass receptacles safely in bottle banks or skips. Many accidents at home, school, and in play areas could be avoided if supervising adults were more aware of the dangers of glass.

The importance of radiological examination of glass injuries is indicated by the finding of glass fragments on one in five of the films. It is particularly important that a radiograph should be taken when there is a glass injury to the eye as failure to do so may lead to diagnostic errors with medicolegal consequences. The low incidence of infection in uncomplicated wounds suggests that antibiotic prophylaxis is unnecessary.

I thank Dr C M Illingworth for allowing me to study patients under her care.

References


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corrective operation which can be performed not only for patients with a ventricular septal defect but also for those with an intact septum.

In this report we describe the successful anatomic correction of an 18 hour old neonate with complete transposition of the great arteries and intact ventricular and atrial septa. The diagnosis was successfully made without recourse to cardiac catheterisation and angiography by using cross sectional echocardiography. Early, non-invasive quantitative investigation of myocardial function suggests a satisfactory result.

Case report

A 14 hour old neonate was referred to this hospital because of cyanosis and a clinical diagnosis of transposition of the great arteries. The child was born of a diabetic mother, weighed 4-4 kg, and had a body surface area of 0-25 m². Clinical examination showed a deeply cyanosed infant with normal peripheral pulses. There were two components to the second heart sound and no murmurs. Respiratory rate was 60–70 per minute and there was mild subcostal recession.

The infant's standard surface electrocardiogram and chest radiograph were normal. Complete transposition of the great arteries was positively diagnosed by cross sectional echocardiography, using an Advanced Technology Laboratories mechanical sector scanner with a 5 MHz transducer. Any additional lesions were excluded and the usual origin of the coronary arteries for a heart with transposition was identified.

Because the infant was developing a metabolic acidosis, it was decided to perform immediate anatomic correction. The normal procedure would have been to perform cardiac catheterisation and balloon atrial septostomy, the latter palliative procedure allowing corrective intra-atrial surgery (Mustard's or Senning's operation) to be performed later in infancy.

Operation. The patient was surface cooled to 24°C under general anaesthesia and the heart was exposed by median sternotomy. Cardiopulmonary bypass was instituted, the baby was cooled to 15°C, and the circulation was stopped.

Surface examination of the heart and great vessels further confirmed the diagnosis. The aorta and pulmonary artery were completely freed from each other and from surrounding connective tissue. A patent ductus arteriosus was divided and oversewn. The two great vessels were transected about 4 mm above the valve commissures, the coronary arteries were then excised with a button of aortic wall around the ostia, after which an ellipse was excised from the edge of the posterior (putative aorta) at the site of two anterior valve sinuses. The coronary arteries together with the button of aortic wall were then sutured into place. The cut end of the distal aorta was then rerouted posterior to the bifurcation of the pulmonary artery and was anastomosed to the root of the posterior vessel, thereby establishing left ventricular aortic continuity. By further mobilisation of the right and left pulmonary arteries and dividing the pericardial folds it was possible to anastomose the pulmonary artery to the root of the anterior vessel directly, thereby establishing right ventricular to pulmonary artery continuity. Cardiopulmonary bypass was re-instigated, the heart was immediately defibrillated, and bypass was stopped without event.

The patient was discharged well from hospital 14 days after operation, without need for any drugs.

Postoperative echocardiography. An M mode echocardiogram and simultaneous electrocardiogram, phonocardiogram, and apexcardiogram performed 28 days after surgery showed almost normal left ventricular function for age. There was paradoxical movement of the interventricular septum but the size of the left ventricle was normal. The shortening fraction was 30%, the mean velocity of circumferential fibre shortening was 1-2 cm/sec⁴, and the left ventricular dimension/apex cardiogram loop was almost 'square'.

Discussion

The ideal of anatomic correction for complete transposition of the great arteries has been pursued by surgeons since 1954 but it was not until 1975 that Jatene et al reported the first successful operation. Yacoub and co-workers have reported the application of this technique to patients with simple transposition of the great arteries in whom the left ventricle has been 'prepared' by banding or shunting the pulmonary artery or both, to prepare the morphological left ventricle to sustain sufficient pressure to overcome systemic vascular resistance. Preparation of the left ventricle by these techniques has not so far gained universal acceptance, and in any event submits the patient to the disadvantages of a palliative operation followed by corrective surgery later in life. The unique contribution of Lecompte et al was to describe a technique of re-establishing continuity of both the aorta and the pulmonary arteries without the use of foreign material.
The precise diagnosis of complete transposition by cross sectional echocardiography should, in most cases, obviate the need for cardiac catheterisation and angiography, provided that the operation is performed immediately so that balloon atrial septostomy is not required.

It follows, therefore, that the ideal patient for anatomic correction has simple transposition of the great arteries (the most common type) and has had no previous palliative or preparatory operation. Surgery should be performed in the early neonatal period while fetal pulmonary vascular resistance remains raised, before its fall in the first few days of life.

Because of the most satisfactory early and intermediate results of interatrial correction of transposition we are not inclined to submit our young patients to preparation of the 'systemic ventricle' by surgical means. We feel that the neonate seen early in life is ideally suited for anatomic correction, providing the operative risk is not considered too high.

Conclusion

We present the successful correction by anatomic repair of transposition of the great arteries and intact ventricular septum in an 18 hours old neonate. It is our opinion that this is the correct approach in the treatment of patients with this cardiac anomaly should an alternative to infra-atrial repair be sought.

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Glue sniffing and cerebral infarction

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**Summary**

A 12 year old boy who was a habitual glue sniffer developed a dense hemiparesis in association with an episode of glue sniffing. Occlusion of the right middle cerebral artery was found. It is postulated that the stroke was precipitated by an episode of vascular spasm.

Glue sniffing is a practice which is increasing in prevalence, particularly among adolescent boys in lower socioeconomic groups. Recognised acute sequelae of glue sniffing include death from asphyxiation, inhalation of vomit, cardiac arrhythmias, coma, and convulsions. We report the case of a boy who sustained a dense hemiparesis from cerebral infarction while glue sniffing.

**Case report**

A 12 year old Caucasian boy, found by his parents in his bedroom unable to speak or move his right arm or leg, suffered a series of convulsions and was admitted to hospital. He subsequently admitted to glue sniffing for the previous two years and immediately preceding the incident had been sniffing a plastic modelling glue, Britfix (the main component of which is trichlorethylene), from a plastic bag held over his mouth and nose. He had become euphoric and had twice fallen off his bunk bed. There was a past history of severe behavioural problems, involving violence to other children and a disruptive tendency in the classroom, for which he attended a residential school.

On examination he was drowsy and aphasic but responded purposefully to commands. He had a dense right hemiparesis, a facial palsy, and an extensor plantar response on the right. There was a soft mid systolic murmur radiating to the carotids. Blood pressure and pulse were normal. Computed tomography on the day of admission to hospital was normal; but a tomogram performed four days later showed substantial infarction in the area of the left middle cerebral artery. Left carotid angiography showed an occlusion of the left middle cerebral artery. There was also a congenital abnormality in