connected with the structural anomaly of chromosome 18 in this case (Stewart et al., 1970). A causal relation between the GH deficiency and the deletion of chromosome material is suggestive, but the association could be coincidental. Deficiency of a pituitary hormone has not been hitherto reported as a feature of any of the chromosomal syndromes, though a probably impaired GH response was found in one of 12 patients with an autosomal aberration recently reported by Ruvalcaba, Thuline, and Kelley (1972). Apparently, our patient has deficient secretion of GH, but, in addition some disturbance of the peripheral growth mechanism independent of GH deficiency (such as in 45,X syndrome), since no acceleration of growth was seen during a year of adequate GH substitution therapy.

**Summary**

A boy with mild mental retardation, short stature, and minor somatic anomalies had deletion of a short arm of chromosome 18, due to a translocation. Immunoglobulin IgA was absent in blood plasma and intestinal mucosa. Growth hormone was deficient after repeated insulin and arginine testing, and nitrogen retention test. There was no acceleration of growth after growth hormone treatment for 12 months.

**References**


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**Short reports**

**Cardiac failure due to acute bacterial endocarditis treated with peritoneal dialysis and aortic valve replacement**

Active bacterial endocarditis of the aortic valve is often fatal due to cardiac failure caused by leaflet perforation and weakening of ventricular muscle from infiltrating myocarditis. So far 61 adult cases treated by valve replacement with a 70% complete cure have been reported (Sarot, Weber, and Schechter, 1970). Peritoneal dialysis has occasionally been used to prepare these patients for surgery when cardiac failure proved intractable. We successfully treated a child with diuretic-resistant cardiac failure caused by active bacterial endocarditis by peritoneal dialysis and aortic valve replacement.

**Case report**

A previously healthy 12-year-old boy developed fever, splenomegaly, aortic insufficiency, and congestive cardiac failure. 3 blood cultures grew penicillin-resistant *Staphylococcus aureus* and he was treated with 18 days of intravenous oxacillin, after which 4 blood cultures were sterile and he was afebrile. 3 months after the onset of this illness he was transferred to the Hospital for Sick Children, Toronto, because of intractable cardiac failure.

On admission he was pale, orthopnoeic, and in marked cardiac failure; pulses were bounding and blood pressure was 114/55 mmHg. There was a grade 1/4 ejection systolic murmur and a grade 3/4 early diastolic murmur at the lower left sternal border. His liver was palpable 6 cm below the right costal margin and tender. ECG showed marked left ventricular hypertrophy and ischaemic changes over the left precordial leads. Chest x-ray showed massive cardiomegaly with a 70% cardiothoracic ratio. There was no clinical evidence of active endocarditis; there was no fever or splenomegaly, and 5 blood cultures were negative. He responded initially to digoxin, frusemide, and spironolactone, but 5 days after admission his cardiac failure became rapidly and progressively more severe; cardiomegaly increased, urine output decreased, and persistent hypotonia and hypochloraemia developed. An intravenous 'cocktail' of frusemide, acetazolamide, aminophylline, and an intramuscular injection of mercuhydrin failed to produce diuresis. Aortic valve replacement became a matter of urgency.

Because he was refractory to conventional decongestive measures, and because his electrolyte disturbance persisted, the patient underwent peritoneal dialysis with a commonly available dialysis solution† to which 5 mEq/l. of KC1 had been added. 500 cm² of this

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*Hoechst Pharmaceuticals, Don Mills, Ontario.

†Dianeal–Baster Laboratories of Canada Ltd., Malton, Ontario.
solution were infused, left in the peritoneal cavity for 30 minutes, and withdrawn. The infusion was repeated 37 times in 24 hours until 3825 cm$^2$ excess fluid had been removed. Before dialysis serum Na was 122, K 5·2, Cl 80 mEq/l; blood urea was 64 mg/100 ml and Hb 12 g/100 ml. After dialysis serum Na was 130, K 3·3, Cl 88 mEq/l; blood urea was 42 mg/100 ml and Hb 14·2 g/100 ml.

Cardiac catheterization, carried out immediately after dialysis showed severe aortic incompetence with a left ventricular end-diastolic pressure of 57, equal to the aortic diastolic pressure. There was no mitral incompetence. Before dialysis serum Na was 122 and Hb 14·2 g/100 ml.

A 12-year-old boy developed intractable cardiac failure caused by bacterial endocarditis rupturing an aortic valve cusp. The cardiac failure was resistant to intensive drug therapy and the patient was prepared for aortic valve replacement by peritoneal dialysis.

We thank Drs. J. D. Keith and R. Fowler for permission to publish this case. D.P. was supported by the Ontario Heart Foundation and Wellcome Trust.

**Discussion**

Aortic valve replacement is a well recognized treatment for active primary endocarditis (Sarot et al., 1970; Manhas et al., 1970). The current mortality from medical treatment is about 30%: the main cause of death being uncontrolled cardiac failure. Severe heart failure after intensive diuretic treatment is often associated with low serum sodium and chloride, possibly due to inappropriate secretion of antidiuretic hormone, and will not respond to further diuretic drugs. The usual methods of correction such as water restriction and acidifying regimens are slow, and their use is limited by side effects. Peritoneal dialysis in a poor-risk patient made cardiac catheterization and valve replacement possible by rapidly changing serum electrolyte levels and reducing total body water.

This technique has been used to prepare adult patients with intractable congestive heart failure for cardiac catheterization and operation (Cairns et al., 1968), but we know of only one other report (Nors et al., 1966) of its use in treating children. Nors et al. (1966) used peritoneal dialysis in 7 infants to remove excess sodium and water and relieve pulmonary oedema. The improvement, though transient, provided the few hours required for a remedial surgical procedure, or the respite necessary in self-limited heart disease.

**Summary**

A 12-year-old boy developed intractable cardiac failure caused by bacterial endocarditis rupturing an aortic valve cusp. The cardiac failure was resistant to intensive drug therapy and the patient was prepared for aortic valve replacement by peritoneal dialysis.

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