Significance of intracranial bruits in neonates, infants, and young children

The presence of an intracranial bruit (ICB) was first recorded by Fisher (1834). Reports suggest several possible causes of ICBs in infants and children. Local factors include cerebral arteriovenous fistulae and meningitis (Allen and Mustian, 1962; Mace et al., 1968). General factors that have been incriminated are anaemia, pyrexia, hyperthyroidism, and exercise (Allen and Mustian, 1962; Ford, 1966).

In studies confined to children, ICBs have been heard in 5–29% of otherwise normal children (Moore and Baumann, 1969). On closer analysis, some workers have found an equal incidence in young infants and older children (Hughes and Todd, 1953), while others report a lower incidence in infants (Mace et al., 1968).

Furthermore, controversy also exists regarding the frequency of ICBs with associated cardiac murmurs (Hughes and Todd, 1953; Farahmand et al., 1964).

The present study was therefore undertaken to analyse the aetiology and significance of ICBs in the age range one day to 3 years, as well as the relationship of ICBs to cardiac murmurs which was felt to have some relevance in the diagnosis of cerebral arteriovenous fistulae in children presenting with cardiac failure.

Patients and methods

Initially 76 children aged one day to 3 years chosen randomly from a children’s hospital and neonatal section of a maternity hospital were examined. In each case the patient was auscultated for the presence of an ICB as well as a cardiac murmur. General factors which could possibly predispose to ICBs were noted, particularly anaemia. All the patients were normothermic.

The results showed that it was necessary to increase the numbers of subjects with cardiac murmurs, particularly the very young infants. A further group of patients with cardiac murmurs and associated cardiac abnormalities, also chosen randomly, was therefore examined.

The patients were divided into two main groups. Group A consisted of 56 subjects who were normal neonates, premature infants, or children with medical conditions—such as respiratory distress syndrome, gastroenteritis, cerebral damage, nutritional disorders, and neonatal jaundice—but who had no cardiac murmurs. Group B consisted of 102 subjects with cardiac murmurs.

The patients in each group were further subdivided into three age ranges: 1 day to 1 month (36 in group A, 38 in group B), > 1 month to 4 months (7 in group A, 31 in group B), and > 4 months to 3 years (13 in group A, 33 in group B) (Figure).

Using the bell and diaphragm of the stethoscope, and with the patient quiet and at rest, the skull was auscultated over the vertex, the temporal areas, and the occiput. ICBs were classified into three grades according to the classification of Mace et al. (1968): grade I, soft systolic bruit heard only between respirations after careful auscultation; grade II, systolic bruits easily heard but obliterated by respiratory sounds; and grade III, pansystolic or continuous bruits heard above the sounds of respiration. Carotid artery compression was not applied.
Discussion

The results show that ICBs occur considerably less often in neonates, even in the presence of significant cardiac murmurs. This observation agrees well with the investigation of Mace et al. (1968) who found ICBs in only 2 (0.8%) of 250 control patients aged 0 to 5 months compared with an incidence of 20% (41 of 213) in children aged 6 to 36 months. Even in the presence of purulent meningitis the incidence of ICBs was lower in the age group 0–5 months (40%) than in the age group 6–36 months (82%). Similarly, Hope and Izukawa (1973) reporting on a series of 14 cases of intracranial arteriovenous fistulae comment that auscultation of 100 unselected neonates showed that cranial murmurs are rare, only one ICB having been heard. These and our findings may be contrasted with those of Hughes and Todd (1953) who found an almost equal incidence of ICBs in the age group 0–12 months (13.8%) and the age group 12–36 months (15.4%). However, these latter authors have no detailed breakdown of the ages of their positive cases in the crucial age group 0–4 months. The significance of our observations regarding the very young infant has not been appreciated, although the rising incidence of ICBs with age has been previously documented. The inference can be made that in an infant under the age of 4 months, an ICB is unlikely to be heard in the absence of a local lesion. If present, in association with cardiac murmurs, the ICB is usually grade I or II in intensity. A grade III ICB and associated signs and symptoms of cardiac failure in such a young infant, is we consider, strongly suggestive of a cerebral arteriovenous fistula. The corollary—namely that in the presence of an intracranial AV-fistula an ICB is very likely to be heard—was shown to be true by Cunliffe (1974), who found that ICBs were present in 12 out of 14 patients aged 0–4 months with an intracranial AV-fistula, and similarly by Hope and Izukawa (1973) in 6 of 7 infants in their own series. Likewise, we have diagnosed 3 cases of cerebral AV-fistula before cardiac catheterisation based on the observation that in addition to signs of cardiac failure each of them had a grade III ICB. These infants presented at ages 2 days, 6 weeks, and 3 months. In contradistinction, an ICB in an older infant or child does not have the same significance, where factors other than a local lesion may be the cause. The incidence of 4 out of 13 (31%) found in our control group (4 months–3 years) agrees closely with the previously reported figures (Hughes and Todd, 1953; Mace et al., 1968; Moore and Baumann, 1969). A general factor such as anaemia may be incriminated (Ford, 1966) as was found in 2 of the 4 patients with ICBs referred to above.
Short reports

The incidence of ICBs in the cardiac group (23 out of 70) was significantly higher than the control group 4 out of 56 ($\chi^2 = 12.03$, $P<0.001$), when only cardiaics with murmurs of intensity grade III/VI or more were considered. Again age was a factor in determining the prevalence of ICBs in association with cardiac murmurs. Thus 4 out of 40 (10%) infants aged 1 day to 4 months had ICBs compared with 19 out of 30 (63%) children aged 4 months to 3 years ($P<0.01$ (Fisher) $\chi^2 = 22.10$, $P<0.0005$). Since most cases with an ICB in this study had associated cardiac murmurs grade III to V/VI, conduction from the chest along the carotid and vertebral vessels might explain the phenomenon.

The conclusion of this study, in conjunction with some of the reported findings, suggests that further investigation is indicated in infants under the age of 4 months with an ICB of grade III intensity. A computerised axial tomographic scan with contrast injection would be appropriate. If cardiac failure is also present, the first choice of diagnostic procedure might be cerebral angiography to confirm the presence of an intracranial arteriovenous fistula.

Summary

In a control group an intracranial bruit (ICB) was heard in 4 of 13 children aged between 4 months and 3 years, but in none of 43 younger infants between the ages of one day and 4 months. In a group of 70 infants with cardiac murmurs of intensity grade III/VI or more, 19 of 30 aged between 4 months and 3 years had an ICB, compared with 4 of 40 younger infants aged between one day and 4 months ($P < 0.0005$). It is concluded that in infants under the age of 4 months, even in the presence of a loud cardiac murmur, an ICB is rarely heard. The presence of an ICB, with or without signs of cardiac failure, strongly suggests an intracranial arteriovenous fistula.

References


MORRIS COHEN and SOLOMON E. LEVIN

Department of Paediatrics, Transvaal Memorial Hospital for Children, and the University of the Witwatersrand, Johannesburg.

Correspondence to Professor S. E. Levin, Department of Paediatrics, Transvaal Memorial Hospital for Children, Joubert Street Extension, Johannesburg 0001, South Africa.

Propranolol as an anti hypertensive agent in children

The anti hypertensive effect of propranolol, a $\beta$-adrenergic blocking agent, is well established in adults (Holland and Kaplan, 1976), but there is little information on it in children. This report describes the anti hypertensive effect of oral propranolol in children with hypertension.

Materials and methods

Nine patients with hypertension were studied. Their diagnoses are given in the Table.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Table</th>
</tr>
</thead>
</table>
| Plasmas renin activity was measured with the patient on an ad lib. salt diet, off all treatment with drugs, and in the supine position (normal values are 0–2.6 ng/ml per hour). Blood pressure (BP) was measured in the supine position using a cuff which covered two-thirds of the upper arm. Diastolic BP was recorded as the muffling of the Korotkoff sounds. To test exercise-induced tachycardia, patients were asked to run on the spot for one minute. Heart rate was measured before and after exercise.

Propranolol was started at a dose of 0.5 mg/kg per day divided 3 or 4 times a day. Dosage was increased until BP responded or side effects developed. Patients were on the drug 3–7 days before the effect of the treatment was assessed.

Two patients were on no other drug during the study; 2 received diuretic plus $\alpha$-methyldopa, and 3 received diuretic plus hydralazine. The dosage of