intracellular and exchangeable bone sodium. It is not possible on the evidence to say if the pattern of distribution of sodium is related causally in any way to the depressive syndrome, although there is evidence that alterations in brain intracellular sodium can affect brain function. After recovery from depression the pattern of sodium distribution suggests that there may be constitutional differences between depressive and normal subjects.

Summary

Water and electrolyte distribution in patients suffering from severe depression was studied by means of a multiple isotope technique and total body counting. Each patient was tested twice, initially when severely depressed and later after clinical recovery.

Residual sodium, which includes intracellular and some bone sodium, was very significantly increased during depression. Total exchangeable sodium and extracellular sodium did not change significantly. Total body water, extracellular fluid, and extracellular chloride were all greater after recovery. Estimates of total body potassium and residual and extracellular potassium did not vary; nor did plasma concentrations of sodium, potassium, chloride, plasma water, and haematocrit.

The abnormal proportion of exchangeable sodium to total body potassium, even after clinical recovery, suggests that depressed patients may differ constitutionally from normal subjects in this respect.

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REFERENCES

- Arons, W. L., Nusimovich, B., Vanderlinde, R. J., and Thorn, G. W. (1958). *J. clin. Endocr.*, **18**, 611.
 Coppen, A. J. (1960). *J. Neurol. Neurosurg. Psychiat.*, **23**, 156.
 — and Shaw, D. M. (1962). Unpublished observations.
 — and Mangoni, A. (1962). *Brit. med. J.*, **2**, 295.
 Driver, M. V., and Eilenberg, M. D. (1960). *J. ment. Sci.*, **106**, 611.
 Flear, C. T. G., Cawley, R., Quinton, A., and Cooke, W. T. (1958). *Clin. Sci.*, **17**, 81.
 Gibbons, J. L. (1960). *Ibid.*, **19**, 133.
 — and McHugh, P. R. (1962). *J. psychiat. Res.*, **1**, 162.
 McMurrey, J. D., Boling, E. A., Davis, J. M., Parker, H. V., Magnus, I. C., Ball, M. R., and Moore, F. D. (1958). *Metabolism*, **7**, 651.
 Maycock, G., Terry, S. W., Vennart, J., and Wise, M. E. (1960). *Nature (Lond.)*, **188**, 355.
 Moore, F. D., McMurrey, J. D., Parker, H. V., and Magnus, I. C. (1956). *Metabolism*, **5**, 447.
 Nichols, G., and Nichols, N. (1956). *Ibid.*, **5**, 438.
 Persky, H., Grinker, R. R., Hamburg, D. A., Sabshin, M. A., Korshin, S. J., Basowitz, H., and Chevalier, J. A. (1956). *Arch. Neurol. Psychiat. (Chic.)*, **76**, 549.
 Remenchik, A. P., and Miller, C. E. (1962). *Whole Body Counting*. International Atomic Energy Agency, Vienna.
 Russell, G. F. M. (1960). *Clin. Sci.*, **19**, 327.
 Schales, O., and Schales, S. S. (1941). *J. biol. Chem.*, **140**, 879.
 Shagass, C., and Schwartz, M. (1962). *Arch. gen. Psychiat.*, **6**, 235.
 Staffurth, J. S., and Birchall, I. (1960). *Clin. Sci.*, **19**, 45.
 Veall, N., and Vetter, H. (1958). *Radioisotope Techniques in Clinical Research and Diagnosis*. Butterworths, London.
 Woodbury, D. M. (1958). *Pharmacol. Rev.*, **10**, 275.

ARTERIAL BRUITS IN ANAEMIA

BY

R. T. WALES, M.B., B.Ch.

Late Research Assistant, Adelaide Hospital, Dublin

E. A. MARTIN, M.D., F.R.C.P.I., M.R.C.P.

Physician, Adelaide Hospital and St. Patrick's Hospital, Dublin

Auscultation over arteries has in recent years acquired importance. A bruit over a limb may point to focal narrowing, due usually to atherosclerosis, or more rarely to an aneurysmal dilatation or to an arteriovenous communication. Peart and Rob (1960) found that 59 out of 103 cases of internal carotid artery stenosis had a systolic bruit over the bifurcation of the carotid artery. An identical bruit in this area may occasionally arise from stenosis of the origin of the external carotid artery (Matthews, 1961). A bruit over the subclavian artery may be an important pointer to a condition of stenosis leading to ischaemia in the arm or to brain-stem and cerebral ischaemia (Reivich *et al.*, 1961; North *et al.*, 1962). Forty-five per cent. of cases of renal artery stenosis leading to hypertension have a bruit over the affected renal artery (Brown *et al.*, 1960). Hunt *et al.* (1962) found a bruit in 18 out of 20 patients with non-atheromatous stenosing lesions of the renal arteries. A continuous bruit in the upper abdomen or flank is a feature of renal arteriovenous fistula (Abbott and Poutasse, 1961). Ranger and Spence (1962) described a case of stenosis of the superior mesenteric artery with a systolic bruit over the central abdomen and both groins. Forty-five per cent. of patients with aortic thrombosis have a murmur over the lower abdomen or lumbar vertebrae (Covey, 1955; Starer and Sutton, 1958).

Not all bruits are pathological. Osler (1880) and Still (1921) published papers on cephalic bruits in children. Wadia and Monckton (1957) found spontaneous intracranial bruits in 60% of healthy children aged 4-5 years, the incidence falling to 4% at 15-16 years. Chest and

neck bruits were found in only 15 out of 513 children below the age of 15 years as compared with intracranial bruits in about 260 of the children. However, spontaneous intracranial or carotid bruits were heard in only 3 out of 228 adults. Bruits could be produced in about 20% of adults by contralateral carotid compression. This may happen also after carotid ligation (Matthews, 1961) and presumably thrombosis.

The following case in which carotid bruits due to anaemia contributed to an erroneous clinical diagnosis of intracranial angioma and led to unnecessary carotid angiography suggested this investigation into the incidence and site of arterial bruits in anaemia.

A 41-year-old housewife had been complaining for 20 months of a throbbing sensation in the left ear. It was synchronous with her pulse and was aggravated by exertion, stooping, leaning her head forwards, noise, emotion, and alcohol. It was relieved by lying down, leaning her head backwards, and pressure on the left side of the neck. For a number of years she had some swelling of the legs. A systolic bruit was audible along the length of both carotid arteries. It was louder in the upper half of the neck and was sometimes more marked on the left. A faint bruit was present over the eyeballs but there was none elsewhere over the skull. No thrill was palpable. Firm pressure over the lower part of the sternomastoid muscle on the left side caused rapid ballooning of the internal jugular vein in the left carotid triangle. This sign was described by Wadia (1960) as a very rare finding in 100 normal individuals and negative in only 1 out of 17 individuals with intracranial angiomas. The sign was negative on the right. There were no other positive findings except some ankle oedema. At a

subsequent examination a variable systolic murmur was heard in all the valve areas of the heart. This did not appear to be conducted upwards to the neck. Bilateral carotid angiography showed normal arteries in the neck and no evidence of an intracranial vascular anomaly. Haemoglobin was 8.5 g./100 ml. (57%). After treatment of the anaemia with iron the throbbing sensation disappeared, but re-examination to see if the bruits have altered has not been possible.

Material and Methods

Twenty patients with haemoglobin levels of 8.9 g./100 ml. (60%) and less were examined. They were in-patients or out-patients of the Adelaide Hospital and the Meath Hospital, Dublin. Patients with acute haemorrhage were excluded. Sixteen were suffering from hypochromic anaemia, three had pernicious anaemia, and one aplastic anaemia. Twenty patients matching the anaemic patients in age and sex but with haemoglobin levels above 11.5 g./100 ml. in females and 13.5 g./100 ml. in males were selected from the records of the pathology department of the Adelaide Hospital. These were examined as control cases when they were found not to have a condition in which a hyperdynamic circulation might be expected—for example, acute haemorrhage, thyrotoxicosis, and pregnancy.

Each group comprised 5 males and 15 females. There was a close parallel in age. The range was 21–82 for the anaemic series and 20–81 for the controls. Each group contained four patients aged 20–29, one aged 30–39, four aged 40–49, one aged 50–59, four aged 60–69, five aged 70–79, and one aged 80–89. The average age of both series was 54 years. Haemoglobin levels were 5.4–8.8 (mean 7.4) g./100 ml. in one series and 11.5–13.8 (mean 12.4) g./100 ml. for the other.

Cases and controls were first examined in the supine position after 15 minutes' rest. The pulse rate and blood-pressure were recorded and auscultation was carried out over (1) the four valvular areas of the praecordium; (2) each supraclavicular triangle; (3) each carotid artery at (a) the upper border of the thyroid cartilage and (b) behind the angle of the jaw on each side; (4) each eyeball; (5) each maxilla; (6) the abdominal wall—1½ in. (3.8 cm.) lateral to and 1 in. (2.5 cm.) above the umbilicus on each side; (7) the xiphisternum; (8) each femoral artery just below the inguinal ligament; and (9) each loin, with the patient prone.

The presence of a murmur was recorded with + or ++, the latter denoting that it was loud. Where no murmur was heard or where there was any doubt a negative was recorded. Wadia's (1960) test of firm pressure over the lower end of each sternomastoid muscle was then carried out and the presence or absence of ballooning of the internal jugular vein in the carotid triangle noted. When possible, the patient was directed to sit up from the supine position, touch the toes, and lie down again 10 times. The pulse rate was recorded again and auscultation carried out as before.

Each individual was questioned regarding the presence or absence of noise in the head synchronous with the pulse.

Re-examination of the anaemic patients 6 to 12 months later was possible in only 11 patients for reasons which included failure to trace, refusal, death, and emigration. Only 5 of the 11 examined had haemoglobin levels above 11.5 g. in females and 13.5 g. in males.

Results

Table I records the incidence of bruits in anaemic patients and controls.

Table II shows an analysis of bruits audible in the neck and aortic area in anaemic patients.

The bruits in all areas were systolic in time and were usually low-pitched and blowing in quality, but about one-quarter of those heard over the supraclavicular triangle were higher-pitched and harsh. In four cases the bruits in one or more areas of the neck were loud. An aortic systolic murmur was noted in only 4 of the 12 cases with neck murmurs. In these the neck murmurs were louder than the aortic murmurs. These two observations suggest that the bruit in the neck is not due to upward conduction from the aortic valve. In a number of cases the effect of altering the posture of the head was tested and found to produce no appreciable change. Considerable pressure, sufficient to occlude the carotid artery, was usually required to stop a bruit.

TABLE I.—Incidence and Situation of Bruits

Site of Auscultation	Anaemic Patients	Controls
Pulmonary area	10	0
Aortic area	4	0
Mitral area	9	3
Tricuspid area	1	0
Right supraclavicular	8	1
Left	7	1
Upper border of thyroid cartilage { Right	10	1
Left	11	2
Behind angle of jaw { Right	6	1
Left	7	0
Eyeball { Right	0	0
Left	0	0
Maxilla { Right	0	0
Left	0	0
Lateral to umbilicus { Right	0	0
Left	1	0
Below xiphisternum	0	0
„ inguinal ligament { Right	1	0
Left	1	0
Loin { Right	0	0
Left	0	0
Total bruits heard	76	9
No. of patients' bruits heard	12	3

TABLE II.—Analysis of Bruits in Aortic Area and Neck in Anaemic Patients

Case No.	Age	Aortic Area	Supraclavicular		Thyroid Cartilage		Angle of Jaw	
			Right	Left	Right	Left	Right	Left
1	76	+			+	+	+	+
2	34		+					
4	23				++	++	++	+
6	54				+	++	+	+
7	72	+	+	+	++	+	++	+
10	64		+	+	+	+		
12	26	+	+	+		++		+
15	21		+	+	+		+	
16	78		+	+	+	+		
17	82		+	+	+		+	+
18	45	+	+	+	+	+		
19	49				+	+		
12 cases		4	8	7	10	11	6	7

++ = Loud bruit.

Resting pulse rates were higher in the anaemic patients (range 68–108, mean 84) than in the controls (range 60–88, mean 71). This difference was maintained after exercise, the range in the 15 exercised anaemic patients being 80–150 (mean 106) and in the 20 controls 76–100 (mean 88). Three anaemic patients had no bruits before or after exercise, and in a further five the bruits were unchanged. In Case 2 a right supraclavicular bruit disappeared. Cases 8 and 9 developed bilateral neck bruits; in four others neck bruits became more widespread. In Case 6 a right thyroid bruit became louder. These changes are detailed in Table III. In one patient bilateral femoral bruits disappeared. The findings in the controls, on exercise, were unchanged in 19 of the 20. The remaining patient was a man of 56 in whom bilateral femoral bruits developed and the bruits over the supraclavicular triangle and carotid artery became louder.

Wadia's test was negative in all cases and controls.

None of the anaemic patients had throbbing in the head or ears at the time of examination, but five admitted to this symptom at some time in their illness.

Only three (Cases 1, 2, and 12) of the five patients who had recovered from the anaemia at follow-up had had bruits when first examined at rest. In all three the neck murmurs had disappeared. The aortic murmur in Case 12 had disappeared but that of Case 1 persisted.

TABLE III.—*Anaemic Cases in Which Exercise Altered the Findings*

Case No.	Age	Aortic Area	Supraclavicular		Thyroid Cartilage		Angle of Jaw	
			Right	Left	Right	Left	Right	Left
2	34							
6	54		+	+	++	++	+	+
8	45				+	+	+	+
9	65		+	+	+	+		
10	64		+	+	+	+	+	+
12	26	+	+	+	+	++	+	++
18	45	+	+	+	+	+	+	+
7 cases		2	5	5	6	6	5	5

Discussion

Bruits can sometimes be induced by firm manipulation of the stethoscope bell over superficial arteries such as the carotid and femorals. Such manipulation was scrupulously avoided in this series.

The arterial bruits occurred almost exclusively in the neck. In the case described in some detail a bruit was also heard over the eyeballs. Error is therefore most likely where intracranial and particularly cerebrovascular disorder is in question. In only 1 of the 12 anaemic patients with neck bruits was the bruit unilateral, and in this patient it was heard over the right supraclavicular triangle. In the other 11 cases bruits were heard in an average of four of the six areas of auscultation in the neck. However, in individual cases auscultation at one level could be misleading. Bruits at the angle of the jaw were unilateral in three patients but in only one at the level of the thyroid cartilage. These are the levels at which the bruit is heard in lesions of the internal carotid artery, and in any cerebrovascular disorder auscultation in these areas is routine. Our experience indicates that where a bruit is heard auscultation should be carried out at other levels and on both sides of the neck. Sometimes the effect of exercise might be tried. Bilateral bruits would suggest anaemia, aortic-valve disease, widespread atherosclerosis, or thyrotoxicosis.

Bruits in anaemia, like cardiac murmurs, are probably related to the rapid flow which is associated with the increased cardiac output (Sharpey-Schafer, 1944) and decreased blood viscosity (Lewis and Drury, 1923). Both of these would contribute to the development of turbulence. The more severe the anaemia the higher the incidence of bruits one might expect. However, in our small series there is no evidence of a direct relationship between the presence or absence of bruits and age, haemoglobin level, and pulse rate. The high incidence in the neck might be related to the more intermittent movement of blood in the large vessels near the aortic valve than peripherally, to the increased cerebral blood flow in anaemia (Schmidt, 1950), and also to the superficial situation of the blood-vessels in the neck. Since there is no higher incidence in the right side, the degree of branching of vessels seems unlikely to be exerting much effect.

Hamburger (1931) records the case of a young woman aged 23 with a haemoglobin of 40% in whom a loud

systolic murmur was audible throughout the head. He emphasized how anaemia might also increase the normal cephalic bruits of childhood. Mackenzie (1955) mentions a similar case and quotes the case of his own and two of Cairns (1942) in which severe nose bleeds followed head injury and in which intracranial bruits developed. Traumatic carotid aneurysm opening into the sphenoidal sinus was present in Cairns's patients, while in the third no clear explanation was found. In these circumstances, knowledge that a bruit might be due to anaemia could be of importance. Meadows (1960) states that a bruit over the usual listening-points of eyeballs, temples, and suboccipital region is not uncommon in anaemia, particularly if the haemoglobin is below 50%. Bruits in such circumstances are also mentioned by Sears (1938) and by Wadia and Monckton (1957). Our experience suggests that bruits in the neck are more common than intracranial ones. Meadows lists, in addition to anaemia and lesions of the carotid artery, the following causes of cephalic bruits: intracranial and orbital angiomas, carotid-cavernous fistulous aneurysms, some cases of intracranial meningioma and glomus tumours, Paget's disease of the skull, and thyrotoxicosis.

Bilateral femoral murmurs were present in a patient, a man aged 54, with a haemoglobin of 6.3 g./100 ml. He had no ischaemic symptoms or signs in the legs. A left abdominal bruit was present in a man aged 62; his blood-pressure was 140/90. The bruit disappeared with a rise in the haemoglobin from 8.4 to 14.3 g./100 ml. It is interesting to speculate whether anaemia could rarely initiate focal renal ischaemia and hypertension just as it may produce angina and claudication. Correction of anaemia might then relieve the hypertension.

The occurrence of cervical bruits in three of the control series demands comment. The patients were aged 26, 56, and 78. None had symptoms of cerebrovascular disease or atherosclerosis elsewhere. Extensive experience of negative results of auscultation over the carotid triangle in neurological patients suggests that this incidence is a chance finding. We have occasionally noted bruits in patients outside the series, particularly in the supraclavicular area. Present methods, such as palpation of arteries and fundus examination, are often unreliable in the assessment of the presence or absence of significant atherosclerosis. Routine auscultation over some major arteries might be a worth-while addition.

No false positives to Wadia's test were elicited in the series, but experience of the case described in detail suggests that caution is required in the interpretation of a positive result.

Summary

Twenty anaemic patients and 20 controls with normal haemoglobin levels were examined for arterial bruits. Bruits occurred in the neck in about half the anaemic patients but were rare in the other regions. They were frequently bilateral and increased by exercise. Follow-up after recovery from the anaemia was possible in only five patients. Bruits had almost entirely disappeared in the three patients in whom they had been present. Bruits in the neck were heard in a small number of control patients with normal haemoglobin. The relevance of these findings to diagnosis is reviewed with particular reference to cerebrovascular disease.

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REFERENCES

- Abbott, C. E. B., and Poutasse, E. F. (1961). *Cleveland Clin. Quart.*, **28**, 283.
- Brown, J. J., Owen, K., Peart, W. S., Robertson, J. I. S., and Sutton, D. (1960). *Brit. med. J.*, **2**, 327.
- Cairns, H. (1942). *Lisboa méd.*, **19**, 375.
- Covey, G. W. (1955). *Neb. St. med. J.*, **40**, 205.
- Hamburger, L. P. (1931). *Amer. J. med. Sci.*, **181**, 756.
- Hunt, J. C., Harrison, E. G., jun., Kincaid, O. W., Bernatz, P. E., and Davis, G. D. (1962). *Proc. Mayo Clin.*, **37**, 181.
- Lewis, T., and Drury, A. N. (1923). *Heart*, **10**, 301.
- Mackenzie, I. (1955). *Brain*, **78**, 350.
- Matthews, W. B. (1961). *J. Neurol. Neurosurg. Psychiat.*, **24**, 161.
- Meadows, S. P. (1960). *Med. Annu.*, p. 297.
- North, R. R., Fields, W. S., de Bakey, M. E., and Crawford, E. S. (1962). *Neurology (Minneapolis)*, **12**, 810.
- Osler, W. (1880). *Boston med. surg. J.*, **103**, 29.
- Peart, W. S., and Rob, C. (1960). *Lancet*, **2**, 219.
- Ranger, I., and Spence, M. P. (1962). *Brit. med. J.*, **2**, 95.
- Reivich, M., Holling, H. E., Roberts, B., and Toole, J. F. (1961). *New Engl. J. Med.*, **265**, 878.
- Schmidt, C. F. (1950). In *The Cerebral Circulation in Health and Disease*, edited by R. F. Pitts, p. 57. Thomas, Springfield.
- Sears, W. G. (1938). *Guy's Hosp. Rep.*, **88**, 308.
- Sharpey-Schafer, E. P. (1944). *Clin. Sci.*, **5**, 125.
- Starer, F., and Sutton, D. (1958). *Brit. med. J.*, **1**, 1255.
- Still, G. F. (1921). *Brit. J. Childh. Dis.*, **18**, 173.
- Wadia, N. H. (1960). *Brain*, **83**, 425.
- and Monckton, G. (1957). *Ibid.*, **80**, 492.

OCCURRENCE OF RESPIRATORY SYNCYTIAL VIRUS IN ACUTE RESPIRATORY DISEASES IN INFANCY

BY

J. DOUGLAS ANDREW,* M.B., Ch.B., M.R.C.P.Ed., D.C.H.

P. S. GARDNER, M.D., Dip.Bact.

Senior Registrar

Consultant Virologist

From the Departments of Microbiology and Child Health, Royal Victoria Infirmary, Newcastle upon Tyne

In 1959 a comprehensive study was carried out in Newcastle to correlate clinical, bacteriological, and virological findings in acute respiratory infection in children (Gardner *et al.*, 1960). One striking feature of this investigation was the failure to obtain evidence of virus infection in acute bronchiolitis.

Continuing observation in the succeeding years, 1960–2, has not thrown further light on this finding, but during this period workers in America (Beem *et al.*, 1960; Chanock *et al.*, 1961; Reilly *et al.*, 1961) have shown that the respiratory syncytial (R.S.) virus, originally isolated in 1956 as a coryza agent in chimpanzees (Morris *et al.*, 1956) and soon after from infants by Chanock *et al.* (1957), is an important causal agent of acute respiratory disease in children. In Britain so far there have been only two reports of the occurrence of this virus in association with respiratory disease (Peacock and Clarke, 1961; Holzel *et al.*, 1963).

We therefore describe briefly our findings in a study of acute bronchiolitis and pneumonia from December, 1962, to March, 1963.

Investigation

This survey was limited to children suffering from acute bronchiolitis and pneumonia, as previously defined by Gardner *et al.* (1960). Children entering the wards of three hospitals in Newcastle upon Tyne—the Royal Victoria Infirmary, the Walkergate Hospital, and, in February and March, the Fleming Memorial Hospital—were included. All hospitals drew patients from the same area of Newcastle and the adjacent communities, admission to a particular hospital being determined primarily by availability of beds. Because of the distances involved and occasionally from difficulties with the transport of specimens, it was not possible to include every child suffering from these conditions.

Whenever feasible, unfrozen cough swabs, broken into Hanks basic salt solution, were brought as quickly as possible to the virus laboratory for the attempted isolation of R.S. virus. Arrangements were made, too, for examination of frozen specimens for the established respiratory viruses which can be safely transported in this way. All specimens were dealt with in the virus laboratory of one hospital; this gave us the opportunity to see if widely

scattered hospitals could form a basis for a satisfactory investigation of the labile R.S. virus in one virus laboratory situated at a distance from some of the participating hospitals.

Methods

All unfrozen specimens were inoculated on to "Bristol" line of HeLa cells if available (Peacock and Clarke, 1961), or Hep 2 cells. Frozen specimens were examined on fertile hens' eggs, monkey-kidney cells, and HeLa or Hep 2 cells. "Bristol" HeLa cells were grown and maintained in rabbit serum, while Hep 2 cells were grown and maintained in calf serum. During the latter part of the investigation suitable supplies of rabbit serum became difficult to obtain and most of the work was performed on Hep 2 cells grown and maintained in calf serum. Tissue-culture cells were maintained for at least 28 days before they were discarded as negative.

R.S. virus was suspected when the characteristic degeneration showing giant cells and syncytia was produced and when this phenomenon could be adequately passaged. Confirmation was obtained by the performance of a neutralization test for the R.S. virus. This test consists of standing a mixture of R.S. virus and antiserum at room temperature for an hour and then transferring the mixture into Hep 2 tissue-culture cells. R.S. virus was also confirmed by staining within the tubes, using Giemsa stain, for syncytia, giant cells, and inclusions. Techniques used for the isolation of other viruses were based on the methods previously described (Gardner *et al.*, 1960).

Results

Specimens were submitted from 43 children under the age of 2 years (20 boys and 23 girls).

There were 13 isolations of virus: R.S. virus from 10 patients and E.C.H.O. (type 11), para-influenza (type 3), and adenovirus (type 7) from one patient each. R.S. virus was found only in late January, February, and early March, and at this time Hep 2 cells were in use for attempted virus isolation from unfrozen specimens. During December and much of January, when "Bristol" HeLa cells were in use, no R.S. viruses were isolated, but it is difficult to say whether virus was absent in December and January or whether Hep 2 cells are a more suitable cell line. This problem is being further investigated. Table I shows the

*Now Consultant Paediatrician, General Hospital, Bishop Auckland.