Medical Memoranda

Severe Anaemia as a Cause of Cranial Bruit in Adults

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Cranial bruits in adults have not so far been reported as a result of severe anaemia. Over the past nine months I have noted the presence of cranial bruits in adults suffering from severe anaemia due to various causes—from pernicious anaemia to anaemia resulting from acute blood loss in bleeding peptic ulcers. I report the first three cases.

CASE REPORTS

Case 1.—A man aged 20 was admitted to hospital on 22 February 1967 complaining of severe headache, which had come on suddenly while at work two days previously. On the day of admission he complained to his general practitioner of headache and weakness, and was referred to us. He had been suffering from indigestion and upper abdominal pain for about four years. He had been taking milk of magnesia, which relieved his pain. On the day before admission he had one black motion; his bowels had not opened on the day of admission. He did not complain of nausea or vomiting. There was nothing relevant in his medical history. He worked for the Electricity Board as a fitter. On examination he was extremely pale. The cardiovascular and respiratory systems were normal. He had epigastric tenderness and examination per rectum showed black stool. C.N.S. examination revealed only the presence of extensive cranial bruits over the frontal area, the eyeballs, and the mastoid regions. Haemoglobin was 30%. Lumbar puncture showed a clear C.S.F. which was normal chemically and microscopically. He was transfused with 7 pints (4 litres) of blood, after which no bruit could be heard on his cranium and his haemoglobin had risen to 78%. Barium-meal examination later showed a posterior duodenal ulcer. He was discharged on 28 February. At follow-up in the outpatient department he was asymptomatic and the cranial bruits could no longer be heard.

Case 2.—A married woman aged 63 was admitted to hospital as an emergency with haematemesis. On the night of admission she had severe epigastric pain and vomited dark fluid and blood clots several times. She felt faint and dizzy but did not actually become unconscious. On admission she was not shocked and did not look anaemic. B.P. was 180/90. The only physical sign was the presence of black stool on rectal examination. On admission the haemoglobin was 71%. Four days after admission she felt ill and again became sick. She vomited about $\frac{1}{2}$ pint (280 ml.) of blood. She looked pale and was sweating. B.P. was 120/80. She complained of a hissing noise in her ears synchronous with her pulse. Auscultation of her right and left mastoid regions revealed the presence of bruits. Her haemoglobin was then 46%. She was transfused with 4 pints (2.3 litres) of blood, at the end of which the noise had gone and the bruit was inaudible. Her haemoglobin at the end of the transfusion was 80%. She made an uneventful recovery. Barium-meal examination later showed the presence of a duodenal ulcer, and she was treated conservatively.

Case 3.—A married woman aged 58 was admitted to hospital on 10 March 1967 complaining of weakness and shortness of breath. She had been unwell for two to three years but there had been a quick deterioration of her health for three weeks before admission. She had also lost a great deal of weight recently. She had long suffered from indigestion and her motions had been black for two years before admission, but she had been on iron tablets during that time. On examination she looked extremely pale. B.P. was 120/60. There were no signs of failure. A systolic murmur was heard all over the precordium, loudest at the apex. Examination per rectum showed black stool. There were loud bruits over both orbits and frontal, temporal, and mastoid regions. The only other C.N.S. signs were loss of vibration sense in both legs. Her haemoglobin was 22%. She was transfused with 5 pints (2.8 litres) of packed cell blood and her haemoglobin rose to 72%. By then she had lost all but faint bruits over the mastoid regions. She had a stormy course in the ward because she bled several times from the gastrointestinal tract. Finally she was operated on and a posterior duodenal ulcer was found. A Polya gastrectomy was performed

and she made excellent postoperative recovery. When seen by me one week postoperatively her haemoglobin was 95% and no bruit could be heard in the cranium.

Though these three cases of anaemia were all due to blood loss, I have heard the cranial bruits in three cases of pernicious anaemia with haemoglobin of under 40% and in several cases of iron-deficiency anaemia. In all these cases the cranial bruits disappeared when appropriate treatment was given to raise the level of haemoglobin in the blood.

COMMENT

Cranial bruits can be heard in normal children before the fontanelles close. Dalsgaard-Nielsen (1939) found that 19% of healthy children he examined between the age of 1 and $1\frac{1}{2}$ years had cranial bruits. He was convinced that the intracranial sound was derived from the venous system. Among the common causes of cranial bruits mentioned by Krenchel (1965), cranial arteriovenous fistula and racemose angioma of the brain seemed to be most common: 80% of patients with arteriovenous fistula and 11% of patients with racemose angioma have cranial bruits. Graf (1945) mentioned a few other conditions that give rise to cranial bruits—for example, carotid artery stenosis, intrathoracic aneurysm. In children with congenital heart disease the cardiac murmur can be transmitted to the cranium and heard as cranial bruit (Dalsgaard-Nielsen, 1939). To the list Elliot (1960) adds aneurysm of the posterior fossa, Paget's disease of the skull, arterial hypertension in young children, and aortic stenosis.

Severe anaemia in adults seems to be a fairly common cause of cranial bruit in general medical wards. Some of these patients can hear a murmur arising from the cranium, and their symptom is put down as tinnitus, which is quite well recognized in severe anaemia. The cause of the cranial bruit in anaemia seems to be multifactorial. In anaemia the body meets the oxygen consumption by raising the cardiac output and increasing its utilization (Nielsen, 1934). It has been shown that there is an increase of about 130% in the cardiac output if the haemoglobin is 30%. The increase in cardiac output is provided by an increased stroke volume, and to a less extent by a tachycardia. This leads to an increased cerebral flow, which has been shown quite clearly by Heyman *et al.* (1952).

Whitaker (1956) has shown that in anaemia there is generalized vasodilatation with the exception of a selective vasoconstriction in the renal vessels. The viscosity of the blood decreases in anaemia. Now the origin of the cranial bruit is most probably due to turbulent flow in the cerebral arteries. The development of turbulence was shown by Reynolds to be determined by the expression $\frac{rVd}{\eta}$, which is called the Reynolds number; r =radius of tube, V =velocity of flow, d =density, and η =viscosity. In anaemia r and V are increased and η is decreased—all contributing towards the production of increased turbulence in cerebral vessels and consequently an audible cranial bruit.

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