SIMULTANEOUS EMBOLISM IN BOTH ARMS

BY

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We read a good deal about arterial embolism of the limbs these days, but it must be a somewhat rare occurrence, for I have had to wait twenty-nine years as a surgeon at a hospital of two hundred mainly surgical beds to see my first case. But, as if to make up for this delay, my case when it did appear presented me with simultaneous embolism of both axillary arteries. How rare this is I have not been able to discover. The surgical literature at my disposal does not record a single case.

Case History

The patient, a railway worker aged 69, had a Syme's amputation of his right foot in 1915 after an accident on the line. The stump has served him well, his only complaint being the usual one with Syme's amputations—that the boot tended to work round in internal rotation so that the toe struck his other foot in walking.

Four months before admission to hospital an ulcer developed over the external malleolus. It was very painful and gradually extended in spite of rest in bed, "elastoplast" bandaging, ultraviolet irradiation, and a variety of dressings, including penicillin. On admission he was seen to be a healthy-looking man. His past health record was good: he had not attended his panel doctor for twenty years. His blood pressure was 130/80, his urine was free from sugar and albumin, and his Wassermann reaction was negative. The radial artery was thickened, however, and capillary circulation at the extremities was slower than normal. A diagnosis of arteriosclerotic circulatory defect was made, and he was advised to have the limb amputated below the knee.

On June 24, 1947, I amputated the leg 4 in. (10 cm.) below the tibial tubercle under thiopentone-gas-oxygen-ether given by Dr. John Moore. The reduced size of the lumen of the tibial arteries was noticed. Healing was retarded by the poor nutrition of the flaps. There was no inflammatory or febrile reaction, but when the stitches were removed on the twelfth day there was considerable failure of union. Healing proceeded very gradually. His general condition was good and he was free from pain. He got about the ward in a wheeled chair and later on crutches.

On Sept. 12, at 5.30 p.m., he went out on crutches to the toilet and while there felt tingling in both wrists and "came over faint." He walked back to his bed on his crutches with assistance. The tingling increased and numbness developed in his hands. A little later he complained of pain in both upper arms. When I saw him at 6.30 he was not greatly distressed, but said the use had gone out of both his arms. They were cold and blue. He could move his fingers slightly. No radial pulse could be felt on either side. Pulsation was present in the left brachial artery to about 1 in. (2.5 cm.) above the humeral condyles. There was no pulsation on the right below the axilla, where the third part of the axillary artery was pulsating.

At 7.30, using brachial plexus block analgesia, I exposed the site of the embolism in the right axillary artery and, controlling the pulsating artery by a loop of tape held up by a haemostat, incised the non-pulsating area immediately below. A clot some 3 in. (7.5 cm.) long was extracted and a small rubber catheter was passed down the artery and suction applied by a syringe; in this way several more pieces of clot were obtained, and a stream of 2% citrate solution was injected. The arm was then massaged in an upward direction and finally the artery was closed by a single continuous suture of No. 2 nylon with a very fine needle. The adventitia was stripped from the artery above the incision and the tape was then relaxed. Pulsation could immediately be felt at the elbow. Others in the theatre said they could feel the radial pulse. The wound was closed and dressed and the patient given a cup of tea and a

cigarette. After an interval of half an hour a similar proceeding was carried out at the lower end of the left brachial artery. A bifurcated clot which had filled the lower part of the brachial artery and the upper 2 in. (5 cm.) of both radial and ulnar arteries was extracted with forceps. Directly the artery was closed and the tape released full volume was restored in the radial pulse.

In view of the different fate which befell the arms I reviewed my technique to discover if possible what determined failure, on the right and success on the left. Differences of procedure were as follows:

Right	Left
Operation two hours earlier	Operation two hours later
Incision in non-pulsating artery	Incision in pulsating artery
Denudation of vessel not so satisfactorily performed	Very thorough denudation of vessel
Injection of heparin had not been given, through delay in obtaining it.	Injection of heparin before starting operation.

Whatever the cause, and I am inclined to blame the inadequate denervation of the right artery, the subsequent fate of the two limbs was strikingly different. By the next morning the left arm was of normal appearance and all movements of the wrist and fingers could be obtained, and with considerable power. Radial pulsation was of normal volume. Subsequent progress was uneventful. The wound healed by first intention and the arm has remained in full function ever since. The right arm was discoloured and cold from the elbow downwards. Pulsation could be felt in the brachial artery down to the elbow. Capillary circulation could be shown to about 2 in. (5 cm.) below the elbow. No radial pulse could be felt. Heparin was pushed, and the arm was kept exposed to cool air.

A masseuse visited him several times a day to give light effleurage and the skin was kept as sterile as possible by rubbing with spirit. 125,000 units of penicillin were given thrice daily. The colour remained the blue of a bruise and no improvement in capillary circulation could be seen, but the patient said he could feel life in the forearm nearly to the wrist and could make some slight movement of one or two fingers. After nineteen days massive vesication appeared and I amputated at the lower third of the upper arm. He has made a good recovery, uses his left arm normally, and is busy trying to walk on his new artificial leg.

KETONURIA IN A CHILD WITH CIRRHOSIS OF THE LIVER

BY

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The interest of the following case lies in the fact that a child with cirrhosis of the liver had ketonuria as the chief sign. The cirrhosis was unsuspected during life. The initial illness, characterized by constipation and abdominal distension, was apparently not related to the liver disease.

Case Report

In November, 1945, a boy aged 2½ was admitted to the Southampton Children's Hospital with severe constipation and abdominal distension. The condition was diagnosed as Hirschsprung's disease. A barium enema shortly after admission revealed a mild degree of megacolon; another, two months later, showed no abnormality. During the next eighteen months the boy was seen regularly in the out-patient department. He remained in fair health, though persistent constipation necessitated the regular use of aperients and occasional enemata.

In January, 1948, when aged 4 years 10 months, he was re-admitted. He had been well until the day before admission, when he had had a convulsion. A further convulsion occurred a few hours later, this being followed by a third on the morning of admission, since when he had been comatose. On examination he was found to be unconscious, slightly cyanosed, and

breathing rapidly and deeply. There was slight neck rigidity. The abdomen was not distended, and rectal examination was negative. The urine contained a large quantity of acetone. Gerhardt's test for diacetic acid was also positive. Sugar was absent. Consciousness returned a few hours after admission. Next day a purulent nasal discharge, pharyngitis, and cough were noted. Radiographs of the chest and examination of the cerebrospinal fluid revealed no abnormality. The blood-sugar level was not estimated. A few days later, following a course of a sulphonamide, recovery appeared to be complete.

After leaving hospital he was noticed always to be listless and easily tired, though well enough to attend school for some weeks. In April, 1948, he was in bed for a week with pyrexia, vomiting, and diarrhoea. His parents remarked that at times he became excessively thirsty, and had an inordinate liking for salt. Apart from a distaste for sweet things, his appetite, though poor, was normal.

In July, 1948, he was again readmitted. A convulsion, preceded by severe abdominal pain, had begun a few hours previously, since when he had been unconscious. On examination he was found to be unconscious and slightly cyanosed, but was breathing normally. No other abnormal physical signs were elicited. The urine contained large quantities of acetone and diacetic acid. No sugar was detected. A sample of blood (taken on admission) showed a blood-sugar level of 35 mg. per 100 ml. A few hours after admission he was conscious and rational and was drinking glucose-water freely. His condition, however, deteriorated. During the night there were two convulsions, and vomiting became frequent. Twenty-four hours after admission he died in a third convulsion.

Post-mortem Findings.—Development normal; nutrition fair; no jaundice. **Lungs:**—Bulky and emphysematous; distended with air anteriorly and cover heart; some petechial haemorrhages on posterior surface; normal appearance on section. **Heart:**—Many large subpericardial haemorrhages. **Spleen:**—Much enlarged; weight, 4½ oz. (135 g.); Malpighian bodies prominent. **Liver:**—Small; weight, 1 lb. 4½ oz. (580 g.); surface coarsely roughened; on section, coarse cirrhosis and early nodular hypertrophy. **Pancreas:**—Normal. **Kidneys:**—Cortex swollen, showing toxic changes. **Colon and other organs:**—Normal.

Histology.—**Liver:**—Portal cirrhosis of nodular type; the portal tracts show excess of fibrous tissue, and heavy infiltration by lymphocytes and a few eosinophils; the liver lobules are very irregular in size and shape; the sinusoids are full of red cells, indicating acute passive congestion. **Pancreas:**—Normal. **Kidneys:**—Marked congestion of the medulla and cortex and of glomeruli; cloudy swelling of tubules.

Comment

It may be inferred that the first attack of convulsions, which ceased spontaneously and did not recur for five months, was hypoglycaemic in origin and was precipitated by a mild infection of the upper respiratory tract.

The low level of the blood sugar was no doubt due to the liver disease (Hartmann and Jaudon, 1937), and was probably the cause of death. Hypoglycaemia has been observed in the last stages of cirrhosis (Peters and van Slyke, 1946; Halbertsma, 1932). In this case, however, the absence of ascites, jaundice, and wasting shows that the disease was hardly in the last stage. Moreover, there had been recovery from the previous attack. Peters *et al.* point out that the hypoglycaemia of liver disease characteristically appears at the end of a considerable period after meals, and that often it is preceded by alimentary hyperglycaemia.

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Medical Memoranda

Spontaneous Subarachnoid Haemorrhage in Infancy due to Angioma of the Cerebellum

The rarity of spontaneous subarachnoid haemorrhage in infancy and the nature of the pathology of this case prompt its presentation for publication.

CASE HISTORY

The patient, a male infant, was the only child of healthy parents and had been born at full term by a normal labour. The neonatal course was uneventful, and he progressed well in all respects up to the age of 3½ months. Then suddenly one afternoon, while being dressed by his mother, he had a convulsion and became semicomatose. He remained in this state, punctuated by recurring generalized fits, until his admission to hospital some hours later. There was no history of any antecedent illness, and the family history revealed nothing of note.

On admission his temperature was 104° F. (40° C.). His condition was that of a resistant stupor. The head was held turned towards the right shoulder and there was conjugate deviation of the eyes to the right. A capillary naevus 3 cm. in diameter was noticed over the left occipital region, and a small capillary naevus was present on the right upper eyelid. The fontanelle was not bulging, but it felt spongy. The head circumference was 17 in. (42.5 cm.). The pupils were of normal shape and reacted to light; no abnormality was seen in the fundi. The head deviation made the assessment of any nuchal rigidity difficult, but there was no spasticity or obvious paralysis of any of the limbs. The heart rate was markedly irregular, tachycardia and bradycardia alternating in an inconstant manner. No other abnormality was found. Lumbar puncture produced 6 ml. of evenly blood-stained C.S.F.; the supernatant fluid was yellow.

Thiopentone per rectum and chloral hydrate were given to control the fits, and 500 mg. of ascorbic acid and 10 mg. of "synkavit" were injected intramuscularly every six hours. Oesophageal feeding was used. No significant change in his condition occurred until two days later, when oedema of the eyelids, right-sided facial spasm, and spasticity of all limbs were noted. A lumbar puncture, done four days after the onset of the illness, again showed a blood-stained C.S.F.

The next day saw considerable improvement in the clinical condition. The fits had ceased, the temperature was almost normal, and the patient was able to take feeds from a bottle. By the ninth day the palpebral oedema had subsided, the deviation of head and eyes and spasticity had relaxed, the temperature was normal, and he was taking notice and feeding eagerly.

This satisfactory state continued for three days. Then his temperature suddenly rose to 104° F. (40° C.), subsequently reaching 107° F. (41.7° C.). No abnormal physical signs were found except for a slight reddening of the left ear-drum. Microscopical examination of the urine and an x-ray film of the chest showed no abnormality. Lumbar puncture produced a clear C.S.F. with a high chloride content (860 mg. per 100 ml.).

A left myringotomy was performed, but no pus was obtained. Hyperpyrexia (107° F.: 41.7° C.) persisted until his death in coma two days later.

Post-mortem Examination.—There was some pooling of blood in the subarachnoid space in the posterior fossa, together with blood-pigment staining. A venous plexus was present over the surface of the left cerebellar hemisphere and one of the vessels had ruptured, the aperture being filled with blood clot. In the substance of the left cerebellar hemisphere there was a vascular neoplasm 3 cm. in diameter, with haemorrhage into it and into the surrounding brain tissue. The left choroid plexus was thrombosed. No tumour was found in any other part of the nervous system and no cysts or tumours in any other organ. The middle ears were healthy and no other disease process was found.

Histological Examination (Professor J. Gough).—"The sections of cerebellum show a haemangioblastoma. In parts there are cellular tissues with ill-developed blood spaces; elsewhere there are cavernous spaces. There is surrounding gliosis and evidence of old and recent haemorrhage, resulting in the formation of haemosiderin and haematoidin. Intense macrophage activity is present."

This case is interesting in that the subarachnoid bleeding occurred abruptly in an apparently healthy infant aged 3½ months, and in the association between two capillary naevi in the head area and a venous plexus over the surface of the left cerebellar hemisphere with a haemangioblastoma in its substance. The sequence of events is thought to have been, first, rupture of one of the veins of the plexus overlying the cerebellum, producing subarachnoid haemorrhage, then an