

CEREBRAL VASCULAR ACCIDENTS

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This article does not aim at presenting any new material. Its purpose is to indicate the clinical findings in the chief varieties of Cerebral Vascular Accidents viz., haemorrhage, thrombosis, and embolism.

It is important to realise from the outset that cerebral thrombosis and cerebral haemorrhage are not as was previously thought almost antagonistic; that a differential diagnosis between the two conditions for the purpose of applying different forms of treatment is not absolutely essential. Cerebral thrombosis and haemorrhage are not mutually exclusive the reason being that the underlying pathological condition is the same, viz., a degenerative process in the vascular wall which gives rise indifferently to either condition. In practice it is found that in the same patient while thrombosis is the cause of the initial strokes, haemorrhage is the cause of the final and fatal one. Consequently cerebral haemorrhage is seen more often in the autopsy room whereas cerebral thrombosis is more commonly met with in the hospital wards.

Cases of cerebral accidents present certain clinical findings, depending on the site and extent of the lesion. When the Middle Cerebral Artery, the commonest site for these lesions, is affected, hemiplegia is the result. This is accompanied by flaccidity which is later replaced by muscular hyper-tonus as voluntary movements start returning. The return of movements is in the order of face — leg — arm. In both upper and lower limbs the movements of the proximal joints are the first to return. Hemiplegia may be associated with aphasia, and sometimes with hemianopia. Pontine apoplexies are characterised by a double hemiplegia and bilateral loss of sensibility; cerebellar apoplexies by incoordination and vomiting. Consciousness may or may not be lost. Conjugate deviation of the eyes is a feature common to all apoplexies. The pupils are often unequal and may be contracted or

dilated widely. Respiration is hurried and laboured; the blood pressure tends to rise provided that the heart is able to cope with the need of a higher B.P. The pulse is full and rapid and the body temperature is usually slightly elevated. The urine may show traces of sugar in the first 24 hrs. after the accident. There may be a mild or moderate leucocytosis and a transient hyperglycaemia may occur.

CASE REPORT I.

C.C., Greengrocer, aged 42, was in hospital on the 13th June 1948 when he developed a partial hemiplegia on the right side.

HISTORY:— The patient was admitted to hospital on the 6th June with severe pain in epigastrium. Patient had vomited once. There was profuse sweating and some dyspnoea. There was rigidity over the upper half of the abdomen and slight enlargement of the liver. The pulse was 110/min. regular and full. The arterial walls were tortuous (Radial, Temporal, Retinal). The B.P. was 140/120. There was no displacement or enlargement of the heart, no murmurs, but the heart sounds were muffled. The possibility of a perforated gastric ulcer was excluded especially by the E. C. G. which showed the Pardee curve of a recent coronary thrombosis. In the course of a few days the B. P. fell to 95/70; after treatment the B. P. was restored to within normal limits. On the 13th June, i.e. seven days after the coronary thrombosis, the patient developed a cerebral accident. Prodromal symptoms were absent. The onset was sudden.

Physical Examination: Patient had facial paralysis on right side, complete paralysis of right upper limb, and slight paresis of right lower limb. There was also complete aphasia.

Consciousness was not lost, but respiration was hurried and stertorous. There was no vomiting and the temperature was 100°F. The B.P. was 80/65. By the 28th June the patient was able to pronounce a few consonants but no whole words; there were almost full movements of the lower limb, and slight movements at the right shoulder joint were now possible. On the 4th July the patient was able to pronounce words, and the return of movements in the upper limb had spread to the elbow.

This case of Cerebral Vascular Accident can be attributed to two causes:

- I. A cerebral thrombosis occurring soon after the coronary infarction. This fits in with the generalised arteriosclerosis from which the patient was found to be suffering and also with the conditions under which the accident occurred — Physical and Mental Rest.
- II. A cerebral embolus originating from an intraventricular thrombus following the infarction of the heart for which the patient was initially admitted to hospital.

The return of movements followed the typical pattern, with the upper limb recovering last, and the proximal joints recovering before the distal. On the other hand the B.P. readings differed from the classical case, in that the B.P. tends to rise provided the heart is capable of responding to the needs of an increased B.P. In this case the recent involvement of the myocardium was probably responsible for the failure of the heart to meet the emergency.

CASE REPORT II.

Mrs. V.P., aged 33, was admitted to hospital on the 15th July 1948 with hemiplegia on the left side.

HISTORY: The patient suffered from Rheumatic fever 16 years before the accident, followed by a second milder attack 8 years later. Patient complained for years of dyspnoea on exertion. There were no prodromal symptoms ushering in the cerebral accident; the onset was instantaneous, the patient falling to the ground; she did not lose

consciousness completely, but was in a stuporous condition. The patient vomited and complained of frontal headache.

Physical Examination: There was complete paralysis on the left side. The reflexes were exaggerated on the affected side, but the plantar response was flexor. The pupils were unequal, the left being greater than the right; they reacted normally to accommodation and light. There were no sensory changes. The sphincters were normal. Pyschically the patient was clear and calm. The heart on auscultation presented a low pitched early diastolic and presystolic murmur at the apex, with accentuation of the pulmonary second sound. The pulse was 60/min. The B.P. 140/100. Respirations 24/min. The urine was turbid, with a specific gravity of 1020, acid in reaction and containing sugar.

By the 25th July the patient had partially recovered movements at the left shoulder and elbow joints; the return of movements in the lower limb was imperceptible. By the end of July wrist movements appeared, while the lower limb started to recover.

From the signs of onset and the history and from the absence of signs of arteriosclerosis, this case of cerebral accident was diagnosed as one of cerebral embolism. The commonest cause of such cases is a detached fragment from an auricular clot in cases of fibrillation. In this particular case there were no signs of fibrillation and the origin of the embolus was presumably from an auricular clot accompanying the mitral lesion.

An interesting point about this case is the course the embolus took passing from the arch of the Aorta into the Innominate and thence into the Right Common Carotid rather than taking the more usual path via the Left Carotid. This explains the absence of aphasia. The B.P. reading after the accident agreed with the usual findings, as did the transient appearance of glucose in the urine.

A case report of cerebral haemorrhage has not been included with the above because, as already stated, these cases usually die soon after onset,