Positional cerebral ischaemia

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SYNOPSIS Four patients are described in whom recurrent, dramatic transient clinical worsening accompanied elevation from the supine toward the sitting position, though postural hypotension was not present. In each patient occlusive cerebrovascular disease was documented angiographically. During an acute stroke syndrome, some patients' compensatory capacity may be so tenuous that postural changes may produce clinical worsening. Possible mechanisms are discussed.

'And for the patient to sit erect at the acme of a disease is a bad symptom in all acute diseases . . .' Hippocrates (Adams, 1939)

The order sheet of each patient admitted to a hospital contains a physician's notation regarding the level of permissible activity and position in bed. Despite this requisite, scanty information is available concerning optimal positioning in any given neurological illness.

Proper positioning of patients with cerebrovascular disease is controversial. Fisher *et al.* (1958) state 'In the upright posture, the cerebral circulation falls, and for this reason it is recommended that patients with a stroke as a result of ischemic infarction should remain horizontal in bed for 7-10 days initially . . .' This is in contrast with Carter's (1964) statement, 'The best position in bed is semi-recumbent if the patient is conscious, as the cerebral blood supply is little influenced by posture and comfort is of great importance'. Most other texts of cerebrovascular disease do not comment on position.

We would like to call attention to four patients with occlusive cerebrovascular disease in whom dramatic transient central nervous system deficits developed when the patients were elevated from the supine position. Postural sensitivity was present early in the stroke syndrome but remitted after several weeks; failure to recognize this might have had dire consequences.

CASE 1

A.W., a 56 year old female, had suffered from

classical migraine for many years. In April 1972, two days before admission, she noticed transient numbness of the right hand. On the afternoon of admission, after shopping, she developed right limb weakness. Her husband found her on the floor and witnessed a gradual increase of her right-sided weakness and a slow disorganization of her speech. As he continued to watch, she gradually improved. When initially examined in the Emergency Ward while she was seated, a severe right hemiplegia, paraphasic verbal errors, poor localization of right tactile stimuli, and right visual inattention were found. The blood pressure was 170/100 mmHg. She was placed supine and within one hour only mild right arm weakness and rare paraphasic errors remained. The blood pressure was 170/100 mmHg supine. Lumbar puncture was normal. The patient was maintained in a supine posture. Transfemoral carotid angiography revealed complete occlusion of the left internal carotid artery at the carotid bifurcation in the neck (Fig. 1). Collateral circulation from the external carotid artery filled the middle cerebral artery *via* the left ophthalmic artery (Fig. 2). Right-sided injection revealed moderate stenosis of the right internal carotid artery at the bifurcation with filling of the right and left cerebral hemispheres.

The next morning she sat up to wash and developed a severe aphasia and right hemiplegia. She was placed in the Trendelenburg position; three minutes later her status had improved with only minimal right hand weakness and occasional paraphasic errors remaining. In the subsequent six days, transient right hemiparesis and poor speech production would occur when she attempted to sit. There was no postural blood pressure change. Occasional transient right-sided weakness occurred even when supine. On day 11, she was allowed to sit

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FIG. 1. Case 1. Left internal artery occlusion. Common carotid injection demonstrating internal carotid artery (arrow ICA) occlusion at the bifurcation of the common carotid artery.

FIG. 2

FIG. 2 Case 1. Collateral circulation filling the carotid syphon. Common carotid arteriogram lateral intracranial view. The ophthalmic artery (single arrow) is filling the carotid syphon (double arrow). Middle cerebral branches are starting to opacify.

FIG. 3

FIG. 3 Case 2. Basilar artery occlusion. Vertebral angiogram, lateral intracranial view, oblique projection. The dye fills the basilar artery (single arrow) to the level of the anterior inferior cerebellar arteries (two connected arrows) and then does not opacify further.

FIG. 4 Case 3. Basilar artery occlusion. Left vertebral injection, intracranial AP view. The dye ends at the proximal end of the basilar artery (arrow BA).

gradually while carefully observed by her physicians. Subsequently, she gradually walked without difficulty. No neurological deficit remained by day 21. She remains well without central nervous system deficit three years later.

CASE 2

V.I., a 58 year old man, had been treated for mild hypertension for 25 years. Beginning in April 1973, he experienced transient feelings of dizziness with blurred vision. Occasionally, horizontal diplopia would accompany these feelings. The episodes occurred once or twice each week, until a week before admission to the hospital when they began to occur three or four times each day. After one of his usual dizzy spells he came to the Emergency Ward where the results of his initial examination were normal. Later, while being examined in the sitting position, he developed right facial weakness, right hemiparesis, and bilateral sixth nerve palsies. A respiratory arrest occurred. Moments later, after being placed supine,

spontaneous respirations resumed, and the only abnormal neurological signs were bilateral sixth nerve palsies, poor upward gaze, and extensor plantar responses. The blood pressure was 140/88 mmHg while supine and changed to 135/88 mmHg upon sitting. Lumbar puncture was normal. Intravenous heparin therapy was begun, and the patient was nursed in the supine position. Episodic light-headedness occurred, especially when the patient attempted to sit up, despite the absence of postural blood pressure change. On day 5, his nurse observed him as he became obtunded while sitting. He was sweating profusely, respirations were shallow with periods of apnoea, and the patient was unarousable. The blood pressure was 130/85 mmHg. Intravenous glucose was given, and the patient was placed in the Trendelenburg position. Within five minutes, his neurological signs had returned to baseline. (The

FIG. 5 Case 3. Vertebral angiography lateral view. Basilar artery (BA) opacified at its distal portion without rostral filling.

blood sugar was 10.6 mmol/l before glucose was given). On the 14th hospital day, transfemoral vertebral angiography revealed total occlusion of the basilar artery at the level of the anterior inferior cerebellar arteries (Fig. 3). On day 21 he was gradually allowed to assume the erect position and later to walk. He has been maintained on warfarin and remains neurologically normal two years later.

CASE 3

H.H., a 65 year old man, had an uncomplicated myocardial infarction 20 years previously and was well except for leg claudication. A year before admission a transient period of diplopia and dizziness was attributed to 'the flu'. Two weeks before admission, an episode of diplopia lasted five minutes. A week later he suddenly fell to the ground without warning and was able to rise without deficit. During the week before admission, three episodes of transient vertigo with diplopia occurred. On the morning of admission, he rose to urinate, felt dizzy, and noticed that he had difficulty in using his right arm. Initial evaluation in the Emergency Ward revealed a left Horner's syndrome, left horizontal rotatory nystagmus, and right limb ataxia. Lumbar puncture was normal except for 31 red blood cells per mm³ on microscopic examination. The blood pressure was 130/60 mmHg. He was nursed in a supine position and noticed occasional fluctuations in his dizziness and right arm clumsiness. On day 5 transfemoral vertebral angiography revealed complete occlusion of the proximal basilar artery with reflux from the left vertebral artery into the right vertebral artery. Both posterior inferior cerebellar arteries were opacified (Figs 4 and 5). Intravenous heparin therapy was instituted. Five days later, after sitting for 10 minutes to eat, he slumped forward unresponsive. One minute later, he vomited. He was placed supine and five minutes later his neurological examination had returned to baseline. The blood pressure was 125/70 mmHg sitting and supine. A thigh haematoma developed; heparin was discontinued and transfusions were given. On the 15th hospital day, while dangling his feet with a nurse in attendance, he became dysarthric. On the 17th day he was seen by the examiner (LRC) while sitting eating breakfast. Neurological examination was normal except for a left Horner's syndrome and left-sided nystagmus. Five minutes later, while still talking to the examiner, he gradually developed left gaze palsy, left intranuclear ophthalmoplegia, right hemiplegia, and garbled speech. The blood pressure was 130/70 mmHg sitting. After three minutes supine, results of the examination returned to baseline. On the 21st hospital day, he was allowed to sit with a

physician in attendance. He was gradually mobilized and allowed to walk. Six months after his hospitalization, neurological examination is normal except for a left Horner's syndrome and minimal gait ataxia. He has been maintained on 300 mg aspirin three times a day.

CASE 4

B.W., a 67 year old female, had been treated for mild hypertension for 14 years. In November 1972, she noticed dysarthria and right-sided weakness on

FIG. 6 Case 4. Pericallosal artery occlusion. Common carotid angiogram lateral intracranial view. There is mild irregularity of the carotid siphon. The pericallosal branch of the anterior cerebral artery is occluded (arrow) for a short segment.

awakening. Examination revealed dysarthria, without an aphasic content, and a slight right hemiparesis. No visual or sensory abnormalities were present. Deep tendon reflexes were increased bilaterally and were greater on the right. There were bilateral extensor plantar reflexes and 'pseudobulbar' speech. Right limb weakness gradually improved over a one week period.

During the subsequent two years, abdominal pain led to the discovery of an abdominal aortic aneurysm, and congestive heart failure was recognized. For two weeks before admission to the hospital in December 1974, the family noted irritability and frequent yawning. Two days before admission, dysarthria worsened transiently. One day before admission, while walking, she suddenly became mute and leaned on the refrigerator with her weak right side. Laughing was her only verbal response to exhortation by the family. She went to bed and shortly thereafter seemed able to talk normally and use all of her limbs. On the day of admission, she again became mute while walking in the kitchen. In the Emergency Ward, examination while supine was normal except for mild dysarthria. The blood pressure was 200/130 mmHg. She sat up on the examining table and 15 minutes later was mute and was unable to repeat phrases, read, or follow spoken commands. The right lower extremity was completely paralysed. After 30 seconds in the supine position, she became

able to speak and obey commands. The right leg weakness returned to 90% of normal. Her blood pressure while supine was 230/125 mmHg. On repositioning the patient in the sitting position, she again became mute with right leg paralysis, the deficit rapidly clearing when supine. Before cerebral angiography, the left common carotid artery was manually compressed for three minutes (to determine if the patient could tolerate carotid compression after carotid arteriography by direct puncture). Mutism and right hemiparesis occurred, clearing within one minute of cessation of the compression. Transfemoral carotid angiography revealed an irregular stenosis of the left internal carotid artery in the syphon with severe stenosis of the left anterior cerebral artery in its early pericallosal course (Fig. 6). In the radiology department, elevation of the head by 10° was accompanied by transient clinical worsening.

During the next 24 hours she was stable while supine. Five days later despite the supine posture, she developed mutism and right leg paralysis. Though alert, she could follow no verbal or written commands or mimic any hand motions. The right shoulder shrug was weak and a strong right grasp reflex was present. She could nod correctly to yes-no questions but could utter only a few syllables. An EMI scan 10 days later revealed an infarction in the paramedian left frontal lobe in the territory of the left anterior cerebral artery (Fig. 7). EEG showed slow activity in the left frontal region. A fixed speech disorder with right leg paralysis remained six months later.

DISCUSSION

Each of the subjects of this report had arteriographically documented occlusive cerebrovascular disease. Occlusion of the basilar artery had developed in two patients; one patient had an occlusion of the carotid bifurcation, and another patient had severe stenosis of the anterior cerebral artery with infarction. We have not seen postural cerebral ischaemia in patients with disease of small penetrating blood vessels. It has always occurred with major vessel occlusive disease.

Transient ischaemic attacks and a fluctuating clinical course were features of each case. Striking, dramatic central nervous system deficits occurred while the patients were erect. In the patients with posterior circulation disease, these consisted of respiratory arrest, stupor, and bilateral brain-stem signs. In the two patients

FIG. 7 Case 4. Computerized axial tomography. There is an area in the left frontal region near the midline with absorptive values close to cerebrospinal fluid. The ventricles are slightly enlarged.

with occlusion in the carotid circulation, altered speech and hemiplegia developed. Dramatic improvement was noted when the patients were placed supine or in the Trendelenburg position. Postural hypotension was absent in three patients and minimal in one patient (230/125 went to 200/130 mmHg). All patients were maintained essentially supine for three weeks, after which they were slowly and carefully mobilized. One patient developed cerebral infarction, the other three patients escaped without significant neurological deficits.

The cerebral circulation is a dynamic system containing within itself multiple reservoirs of possible collateral circulation. Anatomical studies dating back to Thomas Willis (Vander Ecken and Adams 1953), arteriographic data (Mount and Taveras 1957), and experimental studies (Meyer *et al.*, 1954; Meyer and Denny-Brown, 1957; Meyer, 1961) establish the availability and rapid development of collateral circulation after focal ischaemia.

Focal ischaemia gives rise to metabolic alterations which secondarily allow normal blood vessels to augment and regulate blood flow to the ischaemic area according to its needs—a compensatory mechanism. Cerebral blood flow studies in animals (Waltz, 1968) and in patients with stroke (Meyer, 1961; Bloor *et al.*, 1966; Fieschi *et al.*, 1968; Paulson, 1969; Fieschi, 1971), document the frequent failure of autoregulation of vessels in ischaemic regions. It may be impossible without direct measurement to predict whether local blood flow will augment or decrease with alterations in blood pressure or paCO_2 .

Scheinberg and Stead (1949) found a 21% decrement in cerebral blood flow accompanied an alteration in posture from supine to 65° in 20 normal subjects. Meyer *et al.* (1956) exposed patients with carotid and basilar circulation insufficiency to rapid tilting to 70° while being monitored electroencephalographically. Twenty-one of 23 patients with carotid occlusive disease developed EEG changes on tilting and two of these patients had clinical symptoms accompanying the tilt. These changes were attributed solely to the fall in blood pressure accompanying tilting, though in some patients blood pressure changes were modest. In our patients, assumption of the erect posture had a deleterious effect

out of proportion to alteration in blood pressure (minor, transient, or sporadic blood pressure changes could have been missed during clinical study). Might assuming the erect posture in some patients with a very tenuous collateral circulation produce alteration in local cerebral blood flow without any concomitant drop in systematic blood pressure?

Change in posture from the supine to erect position has been known to alter paCO_2 and pH by improving ventilation. Blood gas determinations were not measured in our patients at times of postural change or cerebral ischaemia. However, in patient 4, a 10° alteration in position, though accompanied by clinical worsening, could scarcely have led to acute changes in paCO_2 . In addition, none of our patients had dyspnoea, cyanosis, congestive heart failure, or clinical respiratory disease.

Our experience, herein reported, has led us to conclude that, in some patients with occlusive cerebrovascular disease, position in bed may be critical. Positional worsening should be sought for especially in patients with fluctuating signs. These patients should be maintained either supine or in the Trendelenburg position, and, after an undefined period best determined by trial and error, the patient should be gradually mobilized with caution and supervision.

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