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II. Cerebral Symptoms Due to Kinking, Tortuosity, and Compression of Carotid and Vertebral Arteries in the Neck

Recent advances in radiographic techniques including the use of improved contrast media now permit investigation of cases of cerebrovascular disease by arteriography without undue hazard to the patient. During the past 2-years such studies have been performed regularly on cases admitted to Detroit Receiving Hospital with a diagnosis of occlusive cerebrovascular disease, as well as in the investigation of patients with brain tumor, aneurysm, and focal convulsive disorder. The reason for carrying out arteriograms in cases of vascular disease of the brain has been an investigative one in order to obtain better understanding of the pathogenesis and treatment of these conditions.

Both vertebral and both carotid arteries and their intracrania! branches (panarteriography) have now been visualized in over 70 cases of occlusive cerebrovascular disease. In the course of this investigation we have been surprised to find how frequently marked tortuosity and coiling of the extracranial vessels in the neck (in both the carotid and vertebral arterial systems') were present in patients suffering from cerebrovascular symptoms. Similar coiling and tortuosity occasionally occur in the intracranial segments and branches of these arteries. In some cases stenosis of the vascular lumen was due to kinking of the vessels, and we believe that this may impair blood flow. with resulting symptoms.

Such a possibility is important from a therapeutic standpoint since reconstructive extracranial surgery is a relatively simple procedure.

The purpose of the present communication is to define: (1) the incidence and principal sites of this abnormality, (2) to discuss whether such kinking of the cervical arteries may be affected by posture of the head, and (3) whether

surgical therapy may restore normal anatomical continuity of the vessel and thereby render the brain less susceptible to ischemia.

Previous Reports

Kinks and Coils of the Carotid Artery.— In 1951, **Riser et al.[12] reported a case of carotid artery insufficiency with attacks of vertigo, headache, sweating, and nausea due, they believed, to redundant coiling of the carotid artery in the neck**. Treatment consisted of surgical suspension of the coiled artery to the underside of the sternocleidomastoid muscle after straightening the segment. The patient was observed for 41/2 months after operation, and no further attacks occurred.

In 1958, Gass[6] described 7 examples of "anatomical deviations" of the internal carotid artery in the neck and mentioned finding similar anomalies in 10 additional cases. He was of the opinion that such anomalies were developmental and did not result from pathological processes such as arteriosclerosis, hypertension, and inflammation. Surgical reconstruction was attempted in one patient of this series, and the kinking was demonstrated. The redundant coil was excised and end-to-end anastomosis was performed. Immediate restoration of flow was obtained, but 3 months later a repeat arteriogram showed complete obstruction at the site of anastomosis; fortunately no clinical manifestations resulted from occlusion. **The author concluded that subjects with kinks and coils of the carotid artery may develop symptoms of cerebral circulatory insufficiency under such circumstances as hypotension or change in position of the head.**

Smathers and Smathers described a severely coiled right internal carotid artery in a 37-year-old man which was rotated in the neck to such a degree that partial obstruction to flow existed in at least 2 separate segments. The coiled segment was resected and end-to-end anastomosis was performed. Repeat arteriogram one month later revealed complete obstruction at the site of the anastomosis, fortunately, without clinical manifestations.

Quattelbaum, Upson, and Neville reported 3 cases in which different types of end-to-end anastomosis were carried out for correction of coiling and kinking of the distal portion of the internal carotid artery in the neck. In 2 cases the common carotid was joined to the internal carotid after resection of the bifurcation with sacrifice of the external carotid. In the third case a portion of the common carotid was resected in order to reduce the kinking of the internal carotid. They were able to demonstrate during operative exposure that there was increased kinking as the head was turned toward the elongated and abnormal vessel.

Relation of Head Turning to Arterial Kinking and Stenosis.—Toole and Tucker [17] demonstrated in cadavers the frequent occurrence of obstruction of flow through the carotid and vertebral arteries when the head was rotated. Their experimental method was to perfuse the aorta under constant pressure and observe the outflow from these vessels at their exit from the skull after removing the brain and calvarium. **In 20 cadavers studied by this method it was found that in certain positions of the head (lateral rotation, extension) the rate of flow through one or both vertebrals was slowed to less than 10)% of control in 18 subjects,** while carotid flow was comparably affected in 17 cadavers. In an earlier publication relating to the effects of stimulation of the carotid sinus in man with electroencephalographic control, Toole pointed out that the "cerebral response" could be obtained only in 6 of 39 patients studied when the head was in the neutral position, but with the head rotated to 1 side or the other, a positive response was obtained in 11 additional cases.

In an earlier communication[13] we have demonstrated by arteriography in living patients complaining of cerebrovascular symptoms that stenosis and temporary occlusion of the vertebral arteries by osleophvtic compression may occur during rotation of the neck in patients with cervical spondylosis.

Vertebra! Artery Compression in "Normal" Subjects During Rotation of the Head.

DeKleyn used a perfusion technique in cadavers and showed that with the head extended and turned to one side there was compression of the opposite vertebral artery with reduction of flow of the perfusion fluid. **This "physiological" obstruction to vertebral low on head turning occurs where the vertebral artery passes over the lateral mass of the atlas. Tatlow and Bammer.[15] using a radiographic technique, also demonstrated in cadavers that head turning results in compression of the vertebral arteries as described by DeKleyn.**

Case Material and Results

Arteriograms of all 4 vessels (bilateral, carotid, bilateral retrograde vertebral injections) of 71 cases admitted to the Detroit Receiving Hospital with symptoms and signs attributed to cerebrovascular disease were reviewed and the incidence of kinks and coils of the cerebral vessels noted. Bilateral carotid Arteriograms were carried out by the conventional percutaneous method. Retrograde vertebral injections were made either by percutaneous catheterization of the brachial artery or by percutaneous injection of the subclavian artery.[10,13]

Arteriograms of an additional 425 cases, in which either one or both carotid arteries were visualized, but the vertebral arteries were not, were reviewed separately. The arteriograms in this heterogeneous group of patients were made for the diagnostic evaluation of aneurysm, occlusive vascular disease, brain tumor, and convulsive disorder.

The vast majority of patients were in the sixth and seventh decades of life. There was no significant difference in the number of males and females. Hypertension was present in 60% of the cases.

Our criteria for including cases as examples of kinks and coils were that one or more major cerebral vessels should show efficient elongation and tortuosity so that the narrowing of the vascular lumen resulted or that anatomical abnormality was so gross that it might be assumed that the flow of blood through the vessel probably was disturbed. Cases with compression of the vertebral arteries at the lateral mass of the atlas ("physiological" compression) and by the osteophytes of cervical spondylosis were not included. However, in the selection of case reports, one case of cervical spondylosis is summarized to demonstrate the importance of head turning in the production of cerebral symptoms.

Incidence of Kinks and Coils.—**Of 71 cases diagnosed clinically as occlusive cerebrovascular disease in which panarteriography of the cerebral vessels was performed, one or both carotid or vertebral arteries were markedly tortuous, kinked, or rotated in 21 cases (30%). Tortuosity or kinking of the carotid arteries was present in 17 cases (24%), and abnormal tortuosity of 1 or both vertebral arteries was noted in 9 cases (13%). In 5 cases tortuosity and kinking of vertebral and carotid arteries was combined (7%). Almost all cases had advanced arteriosclerosis or atherosclerosis with associated plaques and stenosis or occlusion of vessels. Kinks and coils were present in 10 cases (4%) of the heterogeneous group of 425 cases in which carotid arteriography alone was performed. The incidence of kinking of extracranial cerebral vessels in 100 consecutive autopsies in patients over 50 years of age dying from any cause has been reported as 4%.[9]**

Relation to Head Turning.—**Head turning to either side during angiography** was carried out in the last 25 cases in which pan-arteriography has been performed. **By this means a greater number of instances of kinking were demonstrated, since significant kinking with stenosis often did not appear until the head was rotated. In all cases in which kinking was present with the head in the neutral position it was made worse by rotation of the head. In numerous cases we have seen evidence of compression pf the vertebral artery as it passes over the**

lateral mass of the atlas but we consider this to be "within normal limits."

Anatomical Sites of Kinking.—**The commonest site of severe kinking and tortuosity of the cerebral vessels is in the portion of the internal carotid artery below the base of the skull. The second commonest site is a few centimeters distal to the origin of the internal carotid artery from the bifurcation. The third commonest site is in the first portion of the vertebral artery. Other common sites include the second portion of the vertebral artery, usually a few centimeters below the normal tortuosity of this vessel as it passes around the lateral mass of the atlas.** Tortuosity with kinking of the intracranial vessels is less commonly seen, but we have noted it occasionally in the following sites: a complete loop of the infraclinoid portion of the internal carotid artery, severe kinking of the anterior cerebral artery, and marked tortuosity of the intracranial course of the vertebral artery.

Illustrative Cases

Of the 38 cases in which marked tortuosity or kinking of major cerebral arteries was present, 7 have been selected for presentation. Kinking was evidently the sole precipitating cause of cerebral vascular insufficiency in one case (Case 1).

CASE 1.—A 41-year-old female alcoholic was admitted to the hospital with the complaint of intermittent but progressive weakness of the left arm and leg with slurred speech for 6 months. She had suffered a mild left hemiparesis one year earlier, with rapid recovery. On examination, the blood pressure was 180/125, with a regular pulse. She was mentally dull and obtunded; there was a spastic weakness of left arm and leg with clonus at the ankle and increased tendon jerks on the affected side. The left plantar reflex was extensor. The right carotid pulse was diminished. EEG showed bilateral slowing (4-6 cps) in both sylvian regions but with right-sided preponderance. EKG showed evidence of left ventricular hypertrophy.

Bilateral carotid and retrograde subclavian arteriograms were performed. **The only abnormality found was a filling defect due to a sharply angulated right internal carotid artery at the level of the first cervical vertebra. At the point of kinking the lumen measured 1 mm.**

The right internal carotid artery was explored under general anesthesia by Dr. Herbert Robb. When the vessel was exposed the nature of the obstruction due to kinking was confirmed, and 1.5 cm. of the kinked vessel was excised and end-to-end anastomosis of the internal carotid artery was performed. After the repair good distal pulsation was present. The abnormal carotid artery was occluded for

14 minutes during the repair without any clinical or EEG change. The patient feels she has benefited by the procedure and there have been no further episodes of worsening. She has been followed for one year and her neurological status of spastic left hemiparesis remains the same. She was readmitted one year after operation and repeat arteriogram showed that normal anatomical continuity of the vessel has been successfully restored .

CASE 2.—A 61-year-old woman was admitted because of vomiting, weakness, vertigo, and ataxia. 0n examination the blood pressure was 110/80. There was left lateral and vertical nystagmus, paralysis of right lateral gaze, right facial weakness, ataxia of gait with deviation to the right, decreased tendon jerks on the right side and extensor plantar reflexes.

Bilateral carotid and vertebral arteriograms were performed. There was marked elongation, dilation, and tortuosity of both intracranial and extracranial vessels. The right carotid artery performed a complete loop in its cervical portion. **There was angulation with kinking of the left internal carotid artery a few centimeters from its origin and tortuosity of the intra- and extra-cranial portions of the distal carotid. there was also marked tortuosity of the second portion of both vertebral arteries, the left vertebral artery being extremely narrow throughout its entire course**. The basilar artery was elongated (the upper end of the basilar artery measuring 22 mm. above the sella turcica) and was irregularly narrowed in its midsection.

Comment: **The clinical diagnosis in this patient was vertebral-basilar artery insufficiency**. She was treated with daily doses of ephedrine and has made complete recovery. Unfortunately, arteriograms were not made before and after rotation of the head.

CASE 3.—A 67-year-old man was admitted to the hospital because of recurrent dizziness and syncope. During the 2 1/2 years prior to admission he had collapsed 4 times. The attacks occurred with or without loss of consciousness. He had also noted transient episodes of ataxia and diplopia. The blood pressure was 118/60; the left pupil was irregular and smaller than the right. Vertical and horizontal nystagmus was present and the gait was broad-based and ataxic. There was rigidity of all 4 limbs, and the left plantar reflex was extensor. **Marked cervical spondylosis was present.**

Bilateral carotid and retrograde vertebral arteriograms revealed several interesting features.

1. Numerous atheromatous plaques were visualized in both subclavian arteries.

2. Stenosis was present at the origin of both vertebral arteries due to atheromatous plaques.

3. Radiolucent lines due to areas of kinking were seen in both vertebral arteries at the level of the atlas and axis.

4. The intracranial portion (part 4) of the right vertebral artery was angulated.

5. The entire vertebral-basilar system including the right subclavian artery was filled by injection of the contrast material into the left subclavian artery.

6. Rotation of the head to the left produced severe kinking and stenosis of the right carotid artery . The vessel filled normally with the head in the neutral position or rotated to the right . **There was increased displacement of both vertebra arteries by osteophytes when the head was turned to the ipsilateral side. A kink appeared in the first portion of the left vertebral artery when the head was turned to the left.**

Comment: Although it cannot be entirely proven without necropsy examination of the small cerebral vessels, it seems reasonable to conclude that this patient's transient symptoms were due to vascular insufficiency resulting, at least in part, from structural abnormalities of the extracranial vessels, particularly when the head was rotated. His blood pressure was low on admission and fluctuated during his period of hospitalization, which, no doubt, also contributed to his episodic vascular disturbance.

CASE 4.—A 33-year-old man was admitted to hospital for investigation of convulsive disorder. Six months earlier he began to have generalized seizures preceded by masticatory movements and shortness of breath. After the first seizure the left side of his body had felt numb and had remained so for 3 weeks. In later seizures, his left leg would remain numb for a few hours to several days after each attack. On examination, his blood pressure was 114/84. General medical and neurological examinations were normal. Electroencephalogram and pneumoencephalogram were normal. **Right carotid arteriogram revealed striking tortuosity with looping of the distal cervical portion of the internal carotid artery.**

Comment: :This case is cited as a presumed example of the developmental type of anomalous vessel, since there was no evidence of arteriosclerosis and he was not hypertensive. Whether or not this finding was coincidental or related in any way to his convulsive disorder is a matter of speculation.

Case 5 .—A 56-year-old woman was admitted because of headache and weakness of the right leg, followed by coma, right hemiparesis, and aphasia. She had been known to be hypertensive for the past 7 years and weighed 250 lbs. She had been placed on a reducing diet and digitalis, and her blood pressure had been reduced to normal levels. On admission the blood pressure was 104/80 and she weighed 144 lbs. Examination of the heart and lungs was normal. She regained consciousness and the following day she gradually recovered from her hemiparesis and dysphasia. **Carotid arteriograms revealed tortuosity and kinking of the left internal carotid artery** . Surgical exploration of the vessel was considered but was not performed because of uncertainty of the relation of the symptoms to this Arteriographic abnormality.

She was followed in the clinic and 3 months later was readmitted because of subjective complaints. On examination, no neurological abnormality was noted. Repeat left carotid arteriogram showed less kinking and tortuosity . After discharge she was again followed in the clinic and readmitted 9 months after her first admission because of headache, weakness, and anxiety. Neurological examination was again normal, and repeat arteriogram showed a normal carotid artery without evidence of the kinking noted on the first admission .

Comment: Serial arteriograms showed progressive improvement of the kinking rather than worsening as we anticipated. In retrospect, it is fortunate that surgery was not performed. Whether the reduction in blood pressure played any role or whether the artery spontaneously displaced itself to a more favorable position is uncertain. The position of the head and the relationship of the artery were the same in the 3 serial arteriograms.

CASE 6.—A 64-year-old man had been well until 3 months prior to admission, when he complained of severe occipital headache. He was examined by a physician and told that he suffered from hypertension, but treatment was not prescribed. **Two weeks before admission he had frequent episodes of feeling faint or "light-headed."** On the day of admission, as he left his employment as a factory worker, he was seen to stagger and fall to the ground. Oxygen was administered as an emergency measure and he was brought to the hospital.

On examination, the blood pressure was 200/100 and the pulse was 72 per minute and regular. The heart was moderately enlarged to the left. He was disoriented but could give his name and recognize a few members of his family. There was bilateral ptosis, paralysis of upward gaze, and weakness of the lower portion of the right side of the face. His gait was ataxic and he tended to fall to the right. Both grasp and sucking reflexes were present, and there was a tendency to catatonic posturing of both upper limbs.

Panarteriography was interpreted as follows:

1. Diffuse atherosclerosis with stenosis was present, due to plaques in both internal carotid arteries, both subclavian arteries, both axillary arteries, and of the left anterior cerebral artery .

2. Both vertebral arteries were diffusely narrow and there was bilateral vertebral artery compression by cervical osteophytes . When the head was rotated to the left there was complete occlusion of the right vertebral artery . The right carotid artery was elongated and tortuous and it became kinked and stenotic when the head was turned to the left .

Comment: This patient had symptoms of diffuse cerebral arterial insufficiency with clinical evidence of focal ischemia of the brain stem. Arteriography revealed atherosclerosis of the cerebral vessels with stenosis. Both vertebral arteries were presumed to be narrow as a developmental anomaly. In addition, 2 other mechanisms were demonstrated which might further decrease cerebral blood flow, namely, compression of both vertebral arteries and kinking of the right carotid artery with rotation of the head.

CASE 7.—A 32-year-old truck driver entered the hospital because of temporary paralysis and dimness of vision. The day prior to admission he was leaning out of the open door of his cab with his head maximally rotated to the left while he was backing up the truck. Suddenly he noted his vision was becoming blurred and dim; he became weak and collapsed from the truck to the ground temporarily paralyzed but without loss of consciousness ("drop attack" of Kremer[8]). He was assisted to his home and rested overnight in bed. When admitted to hospital next day he was still weak in all 4 limbs; his gait was ataxic, and he complained of tingling (paresthesias) over the left side of his trunk and left limbs.

On examination, the general medical status was normal. The neurological examination revealed nystagmus on right lateral gaze, bilateral ptosis, and miosis of the right pupil. Muscle strength was decreased in all 4 extremities. There was mild ataxia on performing the finger-to-nose and heel-to-shin tests. Both biceps jerks were decreased, and the right plantar reflex was extensor. There was no objective sensory disorder demonstrable.

In 2 days the abnormal cranial nerve signs disappeared. All other neurological signs with the exception of weakness and ataxia cleared after 6 days. At the end of 2 weeks examination was normal. However, when his head was passively rotated to either side he developed vertigo, weakness, and lateral nystagmus.

Radiographs of the cervical spine revealed cervical spondylosis with encroachment of the intervertebral foramina by osteophytes. Retrograde transbrachial vertebral arteriograms revealed the following:

1. With the head rotated to the right during injection of the contrast media the right vertebral artery became compressed and narrowed to 2 mm. at the level of C5-C6 and to 3 mm. at C4-C5. When the head was rotated to the left, the right vertebral artery appeared normal

2. When the head was rotated to the left, the left vertebral artery became compressed and stenosed. At the level of C6-C7 the diameter of the lumen was 2 mm.; at C5-C6 it was 2.5 mm at C4-C5 it was 1.5 mm.: at C3-C4 it was2 mm in diameter. When the head was rotated to the right the left vertebral artery appeared normal.

Comment: **We cite this case as an ex-ample of the production of neurological symptoms by vertebral artery compression in the neck**. The subject was young and free of hypertension, heart disease, and diffuse vascular disease. The only abnormality demonstrated by arteriography was vertebral artery compression, which was most severe in the left vertebral artery when the head was rotated to the left. It should be remembered that normally the right vertebral artery becomes compressed on turning the head to the left at the level of the lateral mass of the atlas.[3,13,15] In this patient, rotation of the head to the left would result in severe stenosis or occlusion of both vertebral arteries. He was treated by fusion of the cervical spine. Only 6 months have elapsed since operation but he has been free of symptoms during this interval.

General Comment

The routine investigation by panarteriography of a large series of patients suffering from cerebrovascular disease has revealed that arteriosclerosis and atherosclerosis of cerebral vessels is commonly a diffuse process usually involving more than one vessel and often involving the entire cerebrovascular system. **Associated spondylotic compression of the vertebral artery may also reduce cerebral blood flow or may he the only cause' of cerebral ischemia. Furthermore, arteriosclerosis often results in tortuosity and kinking of one or more major cerebral vessels. Practically all of the large extracranial and intracranial vessels may be involved by this process. We have observed its presence in all 4 portions of the vertebral artery but more commonly in the carotid artery. The commonest site is in the distal portion of the internal carotid artery just before it enters the skull**. The intracranial portion of the internal carotid artery and the anterior cerebral artery may show tortuosity, but we doubt that such intracranial tortuosity causes symptoms other than by compression of the

brain and cranial nerves. **When extracranial vessels become severely tortuous and coiled, this may result in kinking and stenosis. Particularly during rotation of the neck, kinking of the vessel may result in transient occlusion or severe stenosis. We have described one case (Case 1) in which kinking of the vessel appeared to be the sole factor responsible for cerebral ischemia, and 5 other cases where kinking of extracranial vessels may well have contributed to cerebral ischemia**. We wish to emphasize, however, as exemplified by Case 4, that we have encountered several cases where kinking was present without producing any symptoms whatsoever.

The Pathological Basis for Kinking and Tortuosity.—**We believe that 3 mechanisms may play a part in the production of tortuosity and kinking of the major cerebral vessels: (1) arteriosclerosis (particularly of the medial type) with loss of elastic fibers, dilatation, elongation, and tortuosity; (2) hypertension; (3) developmental anomalies.**

Arteriosclerosis: It is known that arteriosclerosis is the commonest cause of tortuosity of major cerebral vessels and it is well recognized in general pathology that arteriosclerosis may result in tortuosity, elongation, and aneurysm formation of large vessels. Penny-Brown and Foley,[4] and Fang and Palmer have described arteriosclerotic changes in the basilar artery with tortuosity and aneurysm formation. We have made similar observations in cases of our own.[10] We agree with Quattelbaum and co-workers that **loss of elastic tissue due to arteriosclerosis is the most important factor in the pathogenesis of this type of tortuosity, kinking, and coiling. The resultant dilatation of cerebral vessels results in widening of the intracranial vessels as well as the extracranial vessels which can readily be recognized in arteriograms.** In one case, not included in the case reports, biopsy of a tortuous superficial temporal artery confirmed the presence of severe medial arteriosclerosis with loss of elastic fibers.

Hypertension: In their discussion of arteriosclerotic aneurysms of the basilar artery, Penny-Brown and Foley[4] stressed the frequent association of arterial hypertension. The same frequent association with hypertension was found in the present series and in one case (Case 5) reduction of hypertension alone may have resulted in improvement of marked tortuosity and kinking of the internal carotid artery over a period of 9 months.

Developmental Anomalies: Gass has presented cogent arguments in favor of the developmental nature of kinks and coils of the major cerebral vessels. **In 2 cases in this series hypertension, diabetes, and arteriosclerosis were**

absent and the patients were young. Some of our cases have led us to conclude that tortuosity of the internal carotid artery occasionally may be due to a developmental anomaly.

Boldrey and associates have discussed the possible role of compression of the internal carotid artery during rotation of the neck by the lateral process of the atlas in producing thrombosis of the internal carotid artery. These authors remarked that when the head is rotated laterally the opposite internal carotid artery is compressed against the lateral process of the atlas. This is particularly likely to occur if adhesions attach the artery to the lateral process, which was shown to be present at operative exploration in one of their cases. Boldrey and associates stated that "occlusion can occur without a thrombosis and has been observed in arteriograms when the lateral view has shown a block in the upper cervical region, yet filling has been demonstrated in the anterior-posterior view with the head turned to the normal anatomical position." **They were of the opinion that compression of the internal carotid artery may occur during sleep, especially with the head turned sharply to side, and that this may contribute to the development of the so-called hypotensive stroke where there is transitory inadequacy of circulation to the cerebrum without actual permanent occlusion demonstrable in subsequent angiograms.**

From our own Arteriographic findings in the living patient we conclude that during rotation of the head in normal individuals there may be (1) compression of the contralateral vertebral artery against the lateral mass of the atlas and (2) compression of the contralateral internal carotid artery against the lateral process of the atlas. We believe that kinking and tortuosity as the sole cause of symptoms is rare. However, in subjects with cerebrovascular disease due to either hypertension, arteriosclerosis, cervical spondylosis, or developmental anomaly, the following additional factors may contribute to cerebral ischemia during rotation of the head:

3. Kinking and stenosis of a tortuous internal carotid artery in its cervical portion. In 25 cases of cerebrovascular disease in which arteriograms of the right carotid artery were performed with the head in the neutral and left and right lateral positions, kinking during rotation of the head was demonstrated in 3 cases, with either ipsilateral or contralateral head turning.

4. Kinking and stenosis of the first portion of the vertebral artery, usually when the head is rotated to the opposite side.

5.Compressive stenosis of the second portion of the vertebral artery by the osteophytes of cervical spondylosis when the head is turned to the ipsilateral side, but occasionally when the head is rotated to the opposite side. Theoretically, such kinks may damage the intima and become the site of fibrin and platelet deposition with the liability of displacement during head turning with embolization of distal vessels.

Therapeutic Considerations in Kinks and Coils of Cerebral Vessels.—Surgical Considerations: We wish to emphasize at the outset that we consider reconstruction of coiled vessels found in arteriograms to be unjustified unless there is marked stenosis present at the point of kinking and there is an obvious relation to symptoms. If the neurological symptoms can reasonably be assumed to be due to the kinking and Stenosis of the vessel, then surgery may be justified (as in Case 1). If osteophytic compression of the vertebral arteries is severe and symptoms of intermittent ischemia are present, we have recommended surgical fusion of the neck or decompression of the transverse foramina to prevent further episodes.[13]

Our Arteriographic investigation of subjects with cerebrovascular symptoms has frequently revealed a diffuse process of atherosclerosis, arteriosclerosis, and vertebral artery compression similar to those reported in necropsy studies by Hutchinson and Yates.[7] In the majority of our cases the following factors have excluded surgical intervention: (1) diffuse vascular disease (e.g., Cases 3 and 6), (2) inaccessibility of diseased vessel (e.g., basilar artery atherosclerosis), (3) poor general condition (e.g., associated myocradial infarct), (4) irreversible cerebral infarction.

Medical Considerations: From the above it is evident that in most cases the therapeutic approach remains a medical problem. Management of the blood pressure is an important consideration. It is probable that the normal anatomical coiling- of the internal carotid arteries at the siphon and of the vertebral arteries as they pass around the atlas serve to damp down the transmitted pulse to the brain.[2] Probably hypertension increases the tortuosity of the major cerebral vessels and early treatment of hypertension may prevent the development of tortuosity of cerebral vessels.

Extreme caution should be exercised in the use of hypotensive agents in subjects with cerebrovascular disease, however, since symptoms of cerebral ischemia may only appear when the blood pressure is lowered. In some of our cases the prevention of postural hypotension by the daily use of ephedrine and weight reduction has abolished attacks of cerebral ischemia. Cardiac output should be maintained at maximum efficiency for symptoms may appear in association with myocardial infarction, heart failure, or blood loss. Instruction regarding limitation of rotation of the neck and the temporary use of a neck collar appears to have

been helpful in those cases in which symptoms were related to rotation of the neck or in which symptoms could be provoked by passive rotation of the neck.

Conclusions

Tortuosity and kinking of at least one major cerebral vessel (with stenosis and the liability of reduction of blood flow through the involved vessel), particularly during rotation of the head, were noted in the panarteriograms (both carotid and both vertebral arteries) of approximately one quarter of a series of 71 cases with symptoms of cerebrovascular disease.

Tortuosity and kinking of major cerebral vessel s are commonly found m the neck and less frequently within the skull. The common sites are the internal carotid artery before it enters the skull and the first and second portions of the vertebral artery. Tortuosity and kinking appear to be due to hypertension and arteriosclerosis, but In some instances coiling of major cerebral vessels results from a developmental defect.

Rarely do kinking and tortuosity of cerebral vessels alone give rise to symptoms of cerebral ischemia. It is occasionally found in arteriograms as an incidental finding. In cases where there is diffuse cerebrovascular disease, such as atherosclerotic plaques with stenosis and vertebral artery compression by osteophytes, kinking and tortuosity of major cerebral vessels may further impair the cerebral circulation and play a role in the production of symptoms.

Arteriograms during rotation of the head show that when the head is turned stenosis of vessels may occur due to the following mechanisms: compression of the vertebral artery against the lateral mass of the atlas, compression of the internal carotid artery against the lateral process of the atlas, kinking of a tortuous internal carotid artery in its cervical portion, kinking of the first portion of the vertebral artery, and compression of the vertebral artery by the osteophytes of cervical spondylosis.

CEREBRO VASCULAR DISEASE IN MAN

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