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## *Whiplash Internal Carotid Artery Occlusion and Hemiplegia*

*Harry A. Gair\**

**W**HEN AN APPARENTLY normal healthy individual, engaged in a business, trade or profession, within a week following an automobile collision in which his neck experienced the hypermotility and torsional force known as "whiplash," suffers a "stroke" and is left permanently hemiplegic, it would be naive to suppose that those affected by such an affliction would meekly ascribe it all to pure coincidence, and not concentrate their fullest attention on the possible connection between the two events.

We deal here with the following medical history: The automobile collision in which the injured was a passenger occurred on June 29, 1958. There was no period of unconsciousness. The only immediate effects were a sense of malaise, slight but persisting headache, and emotional upset. In the five days that followed, the injured followed his usual professional activities, but complained of headaches and of a sensation of numbness on the right side of his face. As the days passed, he noticed flashes in front of his eyes and experienced a fleeting episode of blindness. His headaches became worse, despite pills given to him by his doctor, and he cancelled his social engagements. During the morning of July 4, 1958, he was with his family at his country club swimming pool. A friend suggested that since he was in his bathing suit that a dip in the pool might make him feel better. He jumped in, swam the length of the pool, came out, walked to his chair and collapsed. He was immediately removed by ambulance to a nearby hospital.

The hospital records disclosed that upon admission he seemed oriented and alert. He had left hemiplegia but, during the next 24 hours, he seemed to be improving to the stage of a flaccid hemiparesis. He had good motion in his left extremities. On July 5th, however, he retrogressed, developed a severe left hemiplegia and became obtunded. An emergency right carotid

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arteriogram was performed which revealed a thrombosed right internal carotid artery at the base of the skull.

Spinal tap was within normal limits, with a normal spinal fluid protein and normal manometries. Neurological examination revealed a complete right cerebral syndrome, including a left hemiparesis, left homonymous hemianopsia and left hemisensory syndrome.

His blood pressure on admission was 132/92. A few days later it was 130/80. There was a left Babinski sign. The pupils were round, regular, equal and reacted sluggishly to light. There was no papilledema.

The patient's permanent residuals several years later were a left hemiparesis; he walked with the use of a brace, but he still had no use of the left upper extremity. On occasion, he had episodes of cerebral ischemia manifested by transient dizziness, weakness, nausea, and hemianopsia.

Prior to the accident there were no signs or symptoms related to cerebrovascular insufficiency. There was no evidence of any arteriosclerosis in any organ.

The plaintiff's medical position was that the disability was traumatic in origin. The medical route between the trauma and the disability is charted in the following medical brief.

### **Thrombosis of Cerebral Arteries and of the Great Vessels of the Neck Have Been Caused by Manipulation of the Neck.**

Whiplash forces, about which we are concerned in this article, may exert greater trauma than that incurred through manipulation of the neck, a standard maneuver in chiropractic treatment. Further, it is not only the vertebral and basilar arteries which suffer from such trauma, as will be shown.

The Bulletin of Johns Hopkins Hospital reported two cases in which sudden rotation of the head was followed by damage to the brain stem and cerebellum. In one case post-mortem examination revealed thrombosis of the basilar artery. The clinical features of the other case were consistent with obstruction of the basilar artery. However, this patient survived, but with persisting disability. The Bulletin referred to two previously reported similar cases, in which post-mortem disclosed thrombosis of the basilar artery. The conclusion was expressed that excessive rotation of the head results in damage to the vertebral arteries

with interruption of the blood flow into the basilar system and thrombosis due to stasis.<sup>1</sup>

In a recent article entitled, *Complications of Head and Neck Manipulations*, reporting on injuries sustained by two patients, one of whom died, from sudden head movements, Smith and Estridge raise the question of why *permanent* symptoms result when the head and neck have been rotated, causing the vertebral artery to be *momentarily* compressed. They explain:

Trauma to an artery, one not necessarily diseased, occurring as a result of stretching or compression, may produce spasm. Even after removal of the irritant, the constriction of the artery may persist, with reduction of the blood supply to the irrigated parts or stagnation of blood flow within the involved vessel. The latter could result in thrombosis, and if collateral circulation were inadequate, infarction of the brain stem would ensue. . . .<sup>2</sup>

The "constriction of the artery" mentioned in this article as the persisting cause for reduction of blood supply, appears in the literature as a "kinking" process.<sup>3</sup>

#### **The Relation Between Traumatic Thrombosis of the Carotid Artery and Hemiplegia**

The symptoms arising from thrombosis of the internal carotid artery may simulate severe cranio-cerebral injury,<sup>4</sup> and Sedzimir warns against the error of attributing to concomitant head injuries the symptoms and signs which develop as a result of traumatic thrombosis in the internal carotid artery of the neck.<sup>5</sup> Hemiplegia is sometimes seen in closed head injuries and is then probably due to traumatic thrombosis of the internal carotid or middle cerebral arteries according to Symonds.<sup>6</sup> Further evidence that hemiplegia may result from cervical trauma to the great vessels of the neck can be found in the literature documenting the effects of cervical trauma to the brain stem.<sup>7</sup>

<sup>1</sup> Ford and Clark, Thrombosis of the Basilar Artery, 98 Bull. Johns Hopkins Hosp. 37 (1956).

<sup>2</sup> 182 J. A. M. A. 528 (1962).

<sup>3</sup> Derrick, Carotid Kinking as Cause of Stroke, 25 Circulation 849 (1962).

<sup>4</sup> 9 J. Neurosurgery 495 (1952).

<sup>5</sup> Sedzimir, Head Injury as a Cause of Internal Carotid Thrombosis, 18 J. Neurol. Neurosurg. Psychiat. 293 (1955).

<sup>6</sup> Brock, Injuries of the Brain and Spinal Cord 89 (1960).

<sup>7</sup> Smith and Estridge, *op. cit. supra* note 2, Shapiro and Torres, Brain Injuries Complicating Whiplash Injuries, 43 Minnesota Med. 473 (1960), Textbook of Medicine 1545 (Cecil and Loeb, editors, 10th ed. 1959).

### **Trauma to the Carotid Artery Can Cause Its Occlusion and Hemiplegia**

Before proceeding to ascertain if the particular trauma of whiplash to the carotid artery is known to be a producing cause for hemiplegia, we will establish the medical fact that trauma per se to that artery is a competent producing cause for it.

Minor head injury or injury to the neck may precipitate thrombotic process. Dr. Clark Millikan, consultant in Neurology, Mayo Clinic, has observed "the picture of a stroke"<sup>8</sup> as a result of trauma to the carotid artery and its occlusion after head injury, the site of the injury being immaterial since it is the trauma to the artery which occludes it and causes the stroke. The injury to the neck may appear to be trivial and still be the cause of damage to the carotid artery severe enough to be followed by a spreading thrombosis and hemiplegia as was observed by Northcroft and Morgan.<sup>9</sup>

Sedzimir explains how trauma affects the carotid artery:

It would appear that the distal segment of the internal carotid artery moves with the brain, to which it is tethered by its terminal branches, while the more proximal end remains immobile at the point where the artery leaves the cavernous sinus.

At the latter point, the artery is subject to shearing strains. Such strains, more so, if the artery is sclerotic, may well be the precipitating factor in the formation of a thrombosis.

There are two sites of a thrombosis . . . one near the point of origin of the vessel, and the other in the intracranial portion, close to its termination.

The commonest site of internal carotid thrombosis appears to be in the neck.<sup>10</sup>

The whiplash forces to which we will direct our attention are exerted at the more proximal portions of the carotid artery.

### **Hyperflexion, Hyperextension and Rotational or Torsional Forces (Whiplash) Are a Competent Producing Cause for Thrombosis of the Internal Carotid Artery**

The neck or cervical spine is extremely vulnerable to injury. Stability has been sacrificed for flexibility<sup>11</sup> and the mobility of

<sup>8</sup> 8 Neurology 18 (1958).

<sup>9</sup> A Fatal Case of Traumatic Thrombosis of the Internal Carotid Artery, 32 British Journal of Surgery 105 (1944).

<sup>10</sup> Sedzimir, *op. cit. supra* note 5 at 295.

<sup>11</sup> Jackson, Preface—Symposium—Disorders of the Cervical Spine, 24 Clinical Orthopedics 9 (1962).

the neck makes it easily vulnerable to whiplash injuries.<sup>12</sup> Mc-Gehee describes what happens as the result of the inertia of the relatively heavy skull supported upon the slender column of the cervical spine in a rear end collision:

The body is suddenly thrust or accelerated forward, and the head, which can be regarded as a semi-movable object attached to a relatively fixed base, is left in the original position because of inertia. This hyperextends the neck and may injure the anterior structures.

The force then stops and the body decelerates, but the head is then violently thrown forward as a result of its momentum, plus the reflex action of the neck muscles. Hyperflexion is produced and the posterior structures of the neck are stretched.<sup>13</sup>

Frankel further describes:

The various forces that may be transmitted to the neck are compression, distension, bending, shearing, and torsion.

Where the car is struck from the side . . . such forces are likely to be responsible for a more disabling type of injury than is usually found in the direct-forward or backward-forward movement of the head. . . . If the head and neck are turned at the time of impact, or if the impact comes from the side, severe torsional forces may then be transmitted.<sup>14</sup>

Shapiro and Torres, writing on *Brain Injuries Complicating Whiplash Injuries*, discuss three possible phenomena which can produce brain injury. It may be purely mechanical due to the brain being brought into sudden contact with the skull as a result of the oscillation of the head and neck. The injury may be produced by the acceleration or deceleration influences on the brain tissue. The third explanation is on a circulatory basis due to a vascular insufficiency arising from involvement of the vertebral artery.<sup>15</sup> Gurdjian and Webster, in discussing the factors in the mechanism of whiplash injuries, state:

Since head and neck injuries commonly occur together, thrombosis of the internal carotid artery is possible after direct blows to the neck or lower jaw, whiplash injuries of the neck or head, or accidents which twist or stretch the neck.<sup>16</sup>

<sup>12</sup> Hadley, *The Spine* 151 (1956).

<sup>13</sup> *Whiplash Injury of the Cervical Spine*, 1 *Orthopedics* 105 (1959).

<sup>14</sup> 4 *Defense Law Journal* 93 (1958).

<sup>15</sup> Shapiro and Torres, *op. cit. supra* note 7.

<sup>16</sup> *Head Injuries* 233 (1958).

The internal carotid artery in the neck is protected from *external* violence by the neighboring bony structures and soft tissues. The transverse processes of the upper cervical vertebral artery are behind it, the pharynx lies medially, and the sternomastoid muscle covers it on the anterolateral aspect.<sup>17</sup> As has been described above, the whiplash type of trauma causes sudden hyperextension of the neck with lateral flexion to the opposite side. This sudden stretching of the internal carotid artery over the transverse process of the third cervical vertebra is what causes the intimal tear of the arterial wall.<sup>18</sup> Thus, we can see how, although the internal carotid artery is protected from direct blow, the stresses and strains produced in the neck from this type of whiplash injury are responsible for its occlusion and the resulting thrombosis and hemiplegia. In 1952, The Journal of Neurosurgery reported a thrombosis of the internal carotid artery due to a:

Sudden wrenching of the neck with no blow directly to the artery, because there was no contusion or hematoma.<sup>19</sup>

What needs to be further stressed, is the fact that not only can the interference with the cerebral blood supply be produced by occlusion of the extra-cranial arteries, but that such occlusion is often localized to those arteries without the thrombosis extending inside the skull.

DeBakey refers to this pathology as "extra-cranial segmental occlusive disease":<sup>20</sup>

Cerebrovascular insufficiency produced by extra-cranial arterial occlusive disease has been known as a distinct clinical entity for more than a century. Only since the last decade, however, has this form of arterial occlusion been recognized as often being a well-localized process, with proximal and distal arterial beds relatively normal.<sup>21</sup>

Stewart, in referring to vertebral artery occlusion as the extra-cranial source, writes that infarction of the brain is *common* without occlusion of the *intracranial* vessels.<sup>22</sup>

<sup>17</sup> 43 British Journal of Surgery 215 (1955-1956).

<sup>18</sup> *Ibid.*

<sup>19</sup> Schneider and Lemmen, Traumatic Internal Carotid Artery Thrombosis Secondary to Nonpenetrating Injuries to the Neck, 9 Journal of Neurosurgery 495 (1952).

<sup>20</sup> Surgical Treatment of Cerebrovascular Insufficiency, 30 Modern Medicine 110 (1962).

<sup>21</sup> *Ibid.*

<sup>22</sup> Current Concepts of the "Barre Syndrome," 24 Clinical Orthopedics 45 (1962).

Occlusion of the common or internal carotid artery by thrombosis or embolism was formerly considered to be a rare occurrence. Since the advent of angiography, it has been shown that this is not true.<sup>23</sup> Diagnosis of the condition both in the cervical portion of the internal carotid artery and in its intracranial portion is now relatively simple.<sup>24</sup> Dr. Tyler of the Harvard Medical School, working under a research grant from the National Institute of Health, U. S. Public Medical Services, points out that one of the recent major advances in the understanding of cerebrovascular disease has been the realization that many 'strokes' are secondary to lesions occurring extracranially in the carotid and vertebral circulations and he refers to the large number of *clinically* unrecognized carotid occlusions.<sup>25</sup> In this article he also refers to a suggestion by Fields that lesions contributing to cerebral insufficiency may be extracranial in approximately 25% of patients.<sup>26</sup>

#### **Assuming the Presence of Atherosclerosis, the Occlusive Thrombosis Usually Requires a Triggering Mechanism**

As previously stated, the medical history of this patient revealed no symptoms referable to arteriosclerosis, nor hypertension, nor were there any findings indicating such underlying conditions.

Millikan has pointed out that "ocular signs and symptoms definitely related to cerebrovascular insufficiency were present in 88% of patients with occlusive carotid disease."<sup>27</sup> However, even assuming the presence of atherosclerosis, the most that can be said is that the blood vessel channel (the vascular lumen) narrows, blood flow is diminished, and its margin of safety is reduced. As the conference, headed by Dr. Millikan, reported in 1958:

It is remarkable how severe atherosclerosis may be in the cerebral arteries without giving rise to clinical manifestation or to pathologic change in brain tissue. . . .

It is relatively uncommon for arteriosclerosis itself to

<sup>23</sup> Textbook of Medicine, *op. cit. supra* note 7 at 1543.

<sup>24</sup> 11 Neurology 852 (1961).

<sup>25</sup> Tyler, Modern Concepts of the Pathogenesis and Diagnosis of Cerebrovascular Accidents, 44 Med. Clin. of No. Amer. 1215 (1960).

<sup>26</sup> *Ibid.*

<sup>27</sup> Cerebral Vascular Diseases 8 (Millikan, editor, 1961).



block vessels completely. For the most part, vascular occlusion is a combination of arteriosclerosis plus thrombosis.<sup>28</sup>

Sherry demonstrates that, in a number of cases, thrombi (clots) forming on top of or adjacent to atherosclerotic plaques are not found in pathological study where cerebral vascular insufficiency syndromes have developed.<sup>29</sup> In these cases, Sherry concludes that there must be some fresh thrombotic event as the triggering mechanism. He explains that the formation of the fiber bases for the plaque itself is not a static process and can readily be enhanced by certain stimuli which predispose to further thrombosis and rapidly forming micro thrombi.<sup>30</sup> In short, Sherry concludes that the presence of a slowly developing atheromatous plaque is not a sufficient explanation for acute vascular insufficiency without some acute event to trigger its development,<sup>31</sup> although the plaque is probably the most important local mechanism for the site of thrombus formation "which may be the site of trauma."

Finally, in a study of 30 autopsies reported in the *British Journal of Surgery*, it was found that almost every case presented a variable degree of atheroma (marked arteriosclerosis) at the bifurcation of the common carotid arteries. The conclusion was that "the presence of atheroma at the site of injury is probably coincidental."<sup>32</sup>

### **The Time Factor and the Severity of the Trauma Are Consistent With the Conclusion of Traumatic Causation**

This patient's case has been definitely diagnosed as a thrombosis of the right internal carotid artery. From the facts, described earlier, it will be recalled that the patient suffered a stroke within a week of the accident and that he was not unconscious after the collision.

The occurrence of the stroke within a week is within the recognized time limits for traumatic causation which range from

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<sup>28</sup> Millikan, *op. cit. supra* note 8 at 412.

<sup>29</sup> Sherry, *op. cit. supra* note 27 at 60.

<sup>30</sup> *Ibid* at 61.

<sup>31</sup> *Ibid* at 64.

<sup>32</sup> Northcroft and Morgan, *op. cit. supra* note 9.

"several hours or days,"<sup>33</sup> "24-48 hours,"<sup>34</sup> to "weeks."<sup>35</sup> Gurdjian and Webster write:

" . . . where the paralysis occurs soon after the injury and is due to a thrombosis of the internal carotid artery or other major vessels, the probable cause is then a contusion of the vascular intima caused by hyperextension by kinking, or by an actual blow to the vessel by a fracture deformation."<sup>36</sup>

The fact that the plaintiff was not unconscious after the collision only establishes that there was no serious direct head injury. Gurdjian and Webster maintain that especially where there is no history of unconsciousness after the head injury one should consider the possibility of a traumatic thrombosis of the internal carotid artery in the neck.<sup>37</sup>

Trial lawyers accustomed to strife and controversy will find in what has been written only additional evidence of their never ending burden of preparing for the duties of advocacy. Many of us must battle against the inertia of the mind which makes it adhere to ideas and methods which have had values in the past, and to resist the intrusion of the unaccustomed. This is not peculiar to the laity. The resistance to new formulations, the rejection of seemingly new deductions from different orientations to known facts is found in medicine and allied science. Even where medicine becomes well aware of the etiologies of diseased states and adapts its therapeutic procedures to them, such awareness and knowledge is often repudiated in the courts.

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<sup>33</sup> Gurdjian and Webster, *op. cit. supra* note 16 at 169.

<sup>34</sup> *Ibid* at 364.

<sup>35</sup> Millikan, *op. cit. supra* note 8.

<sup>36</sup> Gurdjian and Webster, *op. cit. supra* note 34.

<sup>37</sup> *Ibid* at 170.