CAROTID SINUS SYNCOPE SECONDARY TO LIGATION OF CAROTID VESSELS FOR INTRACRANIAL ARTERIO-VENOUS ANEURYSM

REPORT OF A CASE WITH SURGICAL CURE, ELECTROENCEPHALOGRAPHIC AND ELECTROCARDIOGRAPHIC STUDIES


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As a direct result of the present war, numerous freak injuries of the nervous system and/or the vessels directly supplying the brain and spinal cord have been observed.

The following case report is that of a soldier who received severe injuries to the head and body with the subsequent development of an intracranial arterio-venous aneurysm which was controlled by ligation of the carotid vessels. As a result of this operative procedure, which was aimed particularly towards the saving of the only remaining eye, opportunity was afforded for the study of the pathogenesis of a typical example of the syndrome of carotid sinus syncope of the cardiodepressor type. In addition, controlled serial electroencephalographic studies were carried out with some rather unusual findings.

Weiss, Ferris and co-workers\(^1\)\(^-\)\(^3\) have described the three types of syncope due to sensitive carotid sinus from the clinical standpoint, as follows: (1) Cardiodepressor, in which there is an appreciable slowing of pulse rate or even asystole, without essential change in the blood pressure; (2) the vasodilator type, in which syncope is associated with fall in blood pressure; and (3) the central type, in which syncope occurs without appreciable change either in blood pressure or pulse rate. Forster, Roseman and Gibbs\(^2\) have recently described the electroencephalographic pattern in all three types of the carotid sinus syncope syndrome. Yeager\(^4\) was able to study the electroencephalographic pattern before and after ligation of the carotid arteries in a case of intracranial saccular aneurysm and noted large slow waves in the electroencephalogram immediately following ligation. The examples referred to above are somewhat amplified in the following case report.

REPORT OF CASE

History. H.W., a single 28-year-old private, was admitted to the Walter Reed General Hospital on 29 September 1943. His past history was irrelevant except for the fact that in 1940, while in civilian life, he was accidentally shot, during a brawl, in the right lower quadrant of his abdomen with perforation of the bowel in several places and with subsequent development of a severe peritonitis. However, he made a slow but complete recovery from this accident and was in good health until his induction in the Army on 4 September 1942. He was
sent to Africa on 26 January 1943 as a mechanic in the Air Forces and was helping in the preparations for the forthcoming Sicilian campaign. On 22 July 1943, the patient was aiding in the loading of motorcycles and jeeps into air transports. He was driving a motorcycle up a ramp to one of the transports when he struck some obstruction and both he and the motorcycle overturned in such a fashion that the patient fell on his back and the cycle struck him on his face and head. The patient stated that he was unconscious for a period of two days and was "crazy in the head" for another week. He was admitted to a general hospital within a few hours following injury where it was noted that the right eye showed a marked degree of edema and ecchymosis of the lids, with chemosis of the conjunctiva and cornea, and no light perception. There was a laceration from 10 to 1 o'clock along the corneal region with some haze under the conjunctiva. There was subcutaneous emphysema about that eye and face, and tenderness all along the zygoma and maxillary bones on the right. Fresh blood was present in both nares and in the right auricular canal. The right ear drum was perforated. The patient was unconscious and in shock. X-ray studies revealed: Fractures of the base of the skull, right mastoid process, the right ramus and condyle of the mandible, the right malar bone extending across the orbital plate and the right clavicle at the junction of the outer and middle one-thirds.

The patient began to drain cerebrospinal fluid from both ears and nares. He was treated for shock by means of whole blood, plasma, immobilization of the accessible fractures, and by the judicious use of sedation. By 24 July he was well enough to be transferred to another general hospital, at which time he was drowsy but well oriented. On 26 July a complete paralysis of the left sixth and seventh cranial nerves was noted. Hearing in both ears was markedly impaired and there was a bluish discoloration and perforation of the left ear drum. He was placed on sulfadiazine on 26 July and this drug was continued in four-gm. daily doses until 4 September. Cerebrospinal fluid continued to drain from both nostrils until 1 September 1943. By 1 September 1943 hearing in both ears had returned to normal and he was able to be up and around. His course during the first two months following injury was marked only by minor, comparatively rare, headaches. There were no other gross neurologic signs. Blood pressure was 120–130 systolic and 70–80 diastolic without much variation from these limits. The general medical examination otherwise was not remarkable.

**Clinical Findings at Walter Reed General Hospital.** On admission 29 September 1943 the nose and nasal accessory sinuses were found to be clear both on physical examination and by x-ray. The ear canals were normal and the drums appeared normal. Hearing in the right ear was 15/15 and in the left 12/15. Vision in the right eye was nil and in the left eye was 20/20—Jaeger-1. The visual field of the left eye was normal. The right eye showed a shrunken phthisical globe. There was paralysis of the left external rectus muscle and complete paralysis of the left face, of infranuclear type. The corneal reflex and the left fundus were normal. Lumbar puncture revealed initial pressure of 150 mm. of water, with clear colorless fluid containing no cells, negative globulin, total protein of 22 mg. per 100 cc. of spinal fluid, and negative Wassermann and colloidal gold reactions.

On or about 1 October 1943 the patient first noted a buzzing sound in his left ear. This sound was likened to a swishing noise, which was synchronous with his pulse beat, and varied in intensity from time to time, depending on his state of activity. Examination on 11 October revealed a slight proptosis of the left eye, and a definite bruit, which was synchronous with the pulse, could be heard by placing the stethoscope on the left temple and over the left eye. Obliteration of the left common carotid pulse by pressure caused immediate disappearance of the subjective noise and the objective bruit. A diagnosis of arterio-venous aneurysm of the left internal carotid artery and cavernous sinus was made. Because of the possible loss of vision in the one remaining eye, and because of the subjective noise, it was decided that ligation of the left common carotid artery was indicated.

**Operation 1.** Following prolonged periods of digital compression of the left common carotid artery, the patient was taken to the operating room on 15 October 1943 and a preliminary tarsorrhaphy of the left eye was performed. The left common carotid artery was then exposed for a distance of about two inches. A rubber band was placed about the vessel and by
traction it could be easily occluded. This immediately stopped the buzzing sound in the patient’s head and produced no untoward symptoms or signs. There was no change in blood pressure or pulse. The vessel was held occluded for thirty minutes and the patient tolerated this well. Thereupon, a tantalum band, 5 mm. in width, was placed about the common carotid artery and compressed until all pulsation distal to the band ceased. One end of this band was made to overlap the other shorter one in order to prevent expansion. Following this there was no subjective swishing sound in the head or objective bruit, and the superficial temporal pulse was obliterated. There were no abnormal neurologic signs.

Course. The postoperative course was uneventful.

Operation II. On 20 October 1943 a simple enucleation of the right eye was done. Following section of the optic nerve there was a severe gush of blood which, however, was controlled by the use of pressure packs.

Course. The postoperative course was not remarkable until 22 October 1943, when the patient again complained of the buzzing sound in his left ear. An audible bruit could not be heard. Because the subjective buzzing became more severe and proptosis again became notable, the patient was again taken to the operating room.

Operation III. On 30 October 1943, under local anesthesia, the old wound was revised. The common carotid artery at the site of previous ligation was imbedded in scar tissue. The tantalum band was exposed and found to have completely obliterated the common carotid artery. The common carotid artery was then exposed at its point of bifurcation and the internal carotid artery mobilized. The latter was compressed and the subjective noise disappeared. Ten cc. of thorotrast were then injected into the internal carotid artery, and x-rays showed an area of increased density on the left side of the sella turcica. A tantalum band was then placed about the internal carotid artery approximately 1 cm. distal to the bifurcation. Immediately all noises in the patient’s head stopped.

Course. For two hours following operation the patient had a right Babinski and increased right ankle and knee jerk. He was otherwise asymptomatic.

On 4 November the patient had an intermittent buzzing in his right ear, which appeared from time to time but in a period of 5 or 6 months gradually became non-extant. He frequently complained of burning and tearing of the left eye and this eye on numerous occasions showed marked conjunctival injection. His course was otherwise asymptomatic until 15 December 1943. On this date he noticed that upon sudden turning of his head to the right his heart would skip one or two beats and he would feel faint. This was inconstant but occurred two or three times per day. On 16 January 1944 he had a focal convulsion. This came on while he was in the sitting posture and immediately after he had suddenly turned his head to the right to address another patient. He fell to the floor unconscious and had convulsive movements of his right extremities lasting for 10 seconds. He had numerous similar episodes subsequently. There was no previous history of syncope or convulsions and no family history of the same.

Operation IV. On 28 February 1944, following preliminary simultaneous electroencephalographic and electrocardiographic studies carried on during massage of the left carotid sinus, the left carotid bifurcation was excised in toto. The previous scar along the anterior border of the left sternocleidomastoid muscle was excised. The section was carried downward until the tantalum clip on the common carotid artery was identified. The internal and external carotid arteries were then dissected free. The tantalum clip on the internal carotid artery was seen to have completely occluded the vessel, thus demonstrating that a tantalum band was an adequate and a safe method of occluding large vessels. The dissection was carried distally along both external and internal carotid arteries. The external carotid artery was then doubly ligated and cut. The internal carotid artery distal to the tantalum clip, and an equal distance from the bifurcation, was likewise doubly ligated and sectioned. The common carotid artery was then cut, no ligature being necessary because of the previous tantalum clip. However, another clip was put on as a safeguard. The bifurcation of the common carotid, its two branches, the carotid body, surrounding nerve fibers, scar tissue and tantalum clip on the internal carotid artery, were then removed in one piece (Fig. 1). The vagus nerve and sympa-
thetic chain were identified. Because of the left facial paralysis and proptosis of the eye, and hence the inability of the patient to close his left eye (in spite of a previous Fuchs' tarsorrhaphy), the sympathetic chain was then crushed, in order to produce a Horner's syndrome.

*Course.* The patient made an uneventful recovery. The left eyeball was sunken and the left pupil was smaller.

Numerous plastic operations were performed on his face and eyes. The right eye socket was fitted with a well emplaced prosthesis. Several Fuchs' tarsorrhaphies were performed on the external canthus of the left eye, the last one on 19 February 1944. As a result of the latter procedure, plus the left Horner's syndrome, the patient was able to close the left eye completely. On 17 January a plastic repair of the left side of the face was carried out by a sling of tantalum wire anchored to the left temporal fascia. During October and November 1943, the fractures of the mandible were reduced and immobilized, with resultant firm consolidation at sites of fracture, with no deformity and with relatively little disability or loss of function about the jaw.

He was sent on a convalescent sick leave of thirty days starting 15 March 1944. During the entire time that he was home and subsequently until the period of observation ceased, about 1 December 1944, the patient had no further convulsions and no episodes of faintness or skipped beat. He noted, however, that his tolerance to alcohol was quite small and that he would become drunk very easily and have very marked hangovers. Accordingly, he cut down on his alcoholic intake, gained much weight, and improved both from a physical and mental standpoint.

**ELECTROENCEPHALOGRAPHIC STUDIES**

Numerous control tracings, beginning on 1 October 1943 (using the Grass 6-channel electroencephalograph), were obtained. A total of 55 EEGs...
were made. Several control simultaneous electroencephalographic and electrocardiographic tracings were made with the patient in a recumbent posture and while alternately massaging each carotid sinus, and the clinical responses and changes in the EEG and EKG, if any, were noted. Prior to the ligation of the common carotid on 15 October 1943 there was no evidence of any slowing in the pulse rate, change in blood pressure, or in the electroencephalogram, upon massage of either carotid sinus. The control EEG tracings showed a moderately dysrhythmic record (Fig. 2). The dominant rhythm

![Control EEG](image)

**Fig. 2.** Control EEG. Note high-voltage 25–30 per second activity in frontal, parietal and temporal areas. (LF) left frontal; (LM) left parietal, (LO) left occipital, (LT) left temporal, (R) similar areas on right side. (All EEGs were made on the Grass 6-channel electroencephalograph, using the “standard ground pick-up” with the ear leads as the “indifferent” electrodes.)

was an excellent 10–10.5 per second but there was much 25–30 per second activity, most marked in the frontal, parietal and temporal leads plus frequent bursts of high-voltage 6–8 per second activity in all leads, with numerous positive spikes. There was no evidence of any focal pathology. There was a moderate “build-up” with overventilation, as noted by the appearance of large high-voltage 2–4 per second waves within 1½ minutes after the onset of overbreathing. These persisted for 20 seconds following three minutes of this procedure.

Frequent electroencephalograms were taken immediately following ligation of the left common carotid artery on 15 October 1943 (Fig. 3A, B, C, D,
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10-16-43
90/72

H.W.
10-28-43
Nº 1748

D
E). The record taken 1½ hours after ligation (when the buzzing had disappeared) showed only a generalized decrease in amplitude. Four and one-half hours postoperatively, although the generalized decrease in amplitude still persisted, the fast activity noted in the control electroencephalogram seemed to be decreasing in quantity and in amplitude. By 16 October 1943 (Fig. 3C) the dominant rhythm was an excellent 9 per second and no appreciable fast activity was seen. On 22 October 1943 a low-voltage fast activity of 18–25 per second began to reappear (Fig. 3D) and it is of interest that it was on this date that the patient noted the recurrence of the buzzing in his left ear. This high-voltage fast activity became more marked in subsequent days (Fig. 3E).

Two hours after ligation of the left internal carotid artery on 30 October 1943 the record showed predominantly high-voltage fast activity, again most marked anteriorly (Fig. 4A). Some 8 per second activity was seen in the occipitals. In both parietal regions a rare burst of high-voltage 3 per second waves was noted. There was no evidence of any focal discharges, although at this time a right Babinski was present. Five hours postoperatively the EEG showed much 14 per second activity, with frequent single discharges of high-voltage 3 per second waves without any focal discharges,
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and in spite of the fact that the patient was conscious and wide awake (Fig. 4B). Ten hours postoperatively the dominant rhythm was a good 8½ per second in the occipital regions and there was much 25–30 per second activity in both frontal and parietal regions (Fig. 4C). The 3 per second waves had disappeared. By 3 November the fast activity had again completely disappeared and the dominant rhythm was now a good 8–8½ per second (Fig. 4D).

On 4 November the patient had begun to complain of an intermittent buzzing sensation in his right ear and it is of interest that whenever the buzzing was present the fast activity reappeared in the electroencephalogram (Fig. 4E, F, G and Fig. 5). By 13 December 1943 the buzzing was still intermittently present, but less marked, and it was noted that the fast activity appeared only after overventilation. The “build-up” with overventilation was now quite marked. From 15 December 1943 to the time of the block excision of the carotid bifurcation on 28 February 1944, the buzzing had completely disappeared and the fast activity in the electroencephalogram was only slightly in evidence in both frontal and parietal regions, if at all. Following operation on 28 February 1944 and subsequently, the buzzing in the right ear was intermittently present again, and usually corresponding to the time in which the buzzing was present fast activity was noted. The more intense the buzzing sound, the higher the voltage of fast activity (Fig. 5A, B).
Fig. 5. EEGs following block excision of carotid bifurcation. Note variation in appearance of fast activity in anterior electrodes, occurring simultaneously with the presence of subjective buzzing sound in right ear (A, B).
Although between 1 October 1943 and 15 December 1943 numerous combined electroencephalographic and electrocardiographic studies were made while alternately massaging either carotid sinus with the patient in a recumbent posture, no evidence of a sensitive carotid sinus of either a central, cardiac or depressor type was noted. On 25 January 1944, following the onset of syncope, this procedure was repeated. As a control study the patient was asked to quickly turn his head to the right. Immediately following this there were one or two skipped beats, but no essential change in the electroencephalogram (Fig. 6). The left carotid sinus was then massaged. Within one second, a period of asystole lasting 20 seconds occurred (Fig. 7A, B). Two seconds after onset of massage, large high-voltage 3–4 per second waves appeared in the electroencephalogram, lasting about two seconds. At the same time the blood pressure fell to zero and coma supervened with the appearance of larger and slower EEG waves. Fourteen seconds after onset of massage, the patient had a right-sided convulsion which lasted 22 seconds (Fig. 7B, C). Following cessation of massage and pressure on the left carotid sinus, the patient recovered.
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A

B

H.W.
Fig. 7. Simultaneous EEG, EKG and blood pressure recordings while massaging and compressing the left carotid sinus. Note: Onset of asystole one second after onset of massage, lasting 20 seconds (A, B); appearance of high-voltage 3–4 per second waves in EEG within two seconds (A), fall of blood pressure to zero (A), followed by coma and convulsion (B); return to resting state after cessation of massage (C, D). (Each strip represents a 10-second period; they run consecutively and continuously from right to left.)
Fig. 8. Simultaneous EEG, EKG and blood pressure recordings while compressing the right carotid sinus region. Note: Relatively slight slowing in pulse rate and no change in blood pressure (A, B); appearance of high-voltage slow waves in EEG within 13 seconds after compression (A, B), followed by coma and convulsion (B); rapid return to normal state after release of pressure (C).

As a corollary a similar procedure was carried out while compressing the right carotid sinus region (Fig. 8). There was relatively little slowing in the pulse rate and no notable change in the blood pressure. However, within 13 seconds following the onset of pressure, large slow waves appeared (Fig. 8A) followed by coma and left-sided convulsion (Fig. 8B), with a rapid return to the normal state after release of pressure (Fig. 8B, C). This phenomenon was apparently due to cerebral ischemia from compression of the remaining carotid vessels.

Following excision of the left carotid body (Fig. 1) on 28 February 1944 this procedure was repeated on numerous occasions until 1 August 1944, with no essential change in the EEG, EKG, blood pressure or in clinical responses. Thus, the sensitive carotid sinus had been completely excised. Responses similar to those previously noted were always evinced on the right side.
SUMMARY

An unusual case of a sensitive carotid sinus syncope developing after ligation of the common carotid and internal carotid arteries is presented. The patient originally had multiple severe injuries about the head, with the resultant production of an arterio-venous aneurysm of the left internal carotid artery and cavernous sinus. An unusual opportunity for follow-up was afforded by the use of the electroencephalogram and the electrocardiogram. The control electroencephalogram was marked by the presence of high-voltage fast activity which disappeared with the abeyance of the subjective noises in the head and reappeared with the intermittent presence of the swishing sounds heard by the patient. The sensitive carotid sinus appeared on the side of the ligated carotid vessels some two months after operation and is attributed to scar-tissue formation, with resultant tension stimulation of the left carotid sinus each time the head was turned to the right. Electroencephalographic and electrocardiographic tracings of the syndrome are recorded. Cure of the sensitive left carotid sinus occurred following complete excision of the carotid bifurcation.

REFERENCES