SYMPOSIUM ON VASCULAR SURGERY

COMBINED VASCULAR AND NERVE INJURIES OF WARFARE*

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IT WOULD APPEAR that in this war the incidence of vascular injuries and of concomitant nerve injuries would probably surpass that of any other war. In addition to ordinary wounds produced by machine gun, rifle bullets and shrapnel, a great many multiple injuries are being produced by fragmentation of land mines, grenades and aerial bombs, as will be shown in a review of individual cases. Land mines and grenades may produce as many as fifty small individual wounds scattered over the body without causing death. This naturally increases the chance of vascular and nerve injuries. Of more significance to the study of major wounds to blood vessels and nerves is the fact that improved methods for the control of shock and infection have preserved a greater number of such injuries for subsequent observation.

Nerve lesions leading to motor and sensory paralysis of the upper and lower extremities are so obvious, because of their disabling consequences, that vascular injuries, at least in their early stages, are frequently overlooked. Conversely, vascular injuries in which a large pulsating hematoma is present may mask injuries to neural structures. With this in mind, it is of extreme importance that patients having nerve lesions be examined with great care lest an aneurysm or an arteriovenous fistula, or other blood vessel injury be missed. Only by a careful palpation, inspection, and auscultation of every wound can this be accomplished.

It is well to recall that all large blood vessels, with a few exceptions, are accompanied by peripheral nervous tissue. This is to be remembered especially in wounds of the neck where the great vessels lie in such close proximity to important cranial and cervical nerves. The same is likewise true in the brachial, femoral and popliteal regions, and while aneurysms of a traumatic nature are more frequently found at those points, it is nevertheless true that any vessel, regardless of its size or location, may be the seat of a disabling vascular injury.

It is not the purpose of this paper to review the common signs and symptoms of aneurysms, or of the ordinary manifestations of nerve injury or nerve section. Reports in the past which have dealt with aneurysms,

*Read before the Southern Surgical Association, December 7–9, 1943, New Orleans, La.

have in the main neglected, or have failed to note concomitant nerve lesions. For this reason cases are presented here to show the frequent combination of such injuries.

In the inception of any vascular injury such as an aneurysm or fistula, external or internal bleeding may be severe and the immediate care, quite correctly, is directed to the control of the hemorrhage. In many instances, therefore, the appraisal of damage to neural tissue is delayed. The appearance of the external wound seen days or weeks after the original trauma is of little value in determining the state of the blood vessel or nerve injury. In many injuries hemorrhage may be slight, even though a major vessel is involved. In the absence of severe laceration, bleeding may be controlled by the pressure of the soft tissues and thus pulsating hematoma or false aneurysm will be formed.

The history will usually suggest the extent of the nervous tissue damage because even during the acute stage of the injury the patient, if conscious, is usually immediately cognizant of sensory and motor loss, whereas, the presence of an aneurysm or a fistula may be discovered only at a much later date. For instance, paralysis of a peripheral nerve recognized by the patient at the time of the wound might indicate severe trauma or even section of that nerve; whereas, if it came on at a later date in the presence of an aneurysm, it most certainly would be due to pressure, a fact of considerable prognostic importance.

In the presence of a large number of casualties and under battle conditions such as front line hospitals are now working, a diagnosis, though incomplete, or even incorrect, may be carried by a wounded soldier for some time. This does not imply that proper treatment has not been given, but it does entail upon those who are working in the Zone of the Interior, or in general hospitals distant to the battlefield, the necessity of particularly careful examination, uninfluenced by any previous diagnosis. Moreover, it is a well known fact that a vascular lesion may develop slowly to the point of recognition. For example, an arteriovenous fistula, surely present from the onset of injury, may not show the characteristic signs until some time later when edema and tissue hemorrhage have been absorbed. This is substantiated by the fact that vascular lesions were discovered in five patients in this general hospital upon whom only neurologic diagnoses had been made previously.

The neurologic sequelae of gunshot wounds of the central and peripheral nervous systems have varied but little in the previous armed conflicts in which these disturbances have been studied. The causalgia that Weir Mitchell described so graphically in the American Civil War differs in no respect from that observed today in Army General Hospitals. In this war the character of the inflicting forces has altered both the type and particularly the frequency of injuries to neural tissue as exemplified in the first instance by blast injuries and in the second by aerial bomb and antipersonnel land mine injuries.

I—VESSEL-NERVE INJURIES OF THE NECK

Among these previously well recognized neurologic syndromes are those extracranial lesions of the last four cranial nerves caused by gunshot wounds. As Pollock and Davis[1] have stated, the number of such syndromes is limited only by the possible combinations of complete or incomplete paralyses of these several cranial nerves and the descriptive ability of the various observers. Among the classical syndromes involving all or several of the last four cranial nerves are those of Avellis,[2] consisting of a unilateral paralysis of the soft palate and the larynx, from a lesion of the vagus and the internal branch of the spinal accessory nerve; of Schmidt,[3] characterized by the inclusion of the external branch of the spinal accessory nerve to the foregoing symptoms, and of Jackson,[4] which includes, in addition to all these symptoms, involvement of the hypoglossal nerve.

During the course of and following World War I, Vernet,[5] Collet,[6] Sicard,[7] Villaret,[8] Barbé,[9] Körner,[10] and Pollock[11] added significant contributions to the literature of War injuries involving these neural structures.

These authors gave to the combination of cranial nerves involved descriptive terms denoting either the neurologic distribution of or the anatomic location of the causative wound, such as, "syndrome of the posterior lacerated foramen" (Vernet), "glosso-laryngo-scapulo-pharyngeal hemiplegia" (Collet), "complete syndrome of the last four cranial nerves" (Sicard), and "syndrome of the posterior retroparotid space" (Villaret).

Whatever the syndromes may be termed, the resulting neurologic dysfunction depends upon the close anatomic continuity of the last four cranial nerves in the early part of their extracranial courses. In the jugular foramen, the glossopharyngeal, vagus and spinal accessory nerves, in order from before backward, lie between the inferior petrosal sinus anteriorly and the internal jugular vein posteriorly. At its exit from the jugular foramen, the internal jugular vein lies to the outside of the nerves and the carotid artery, with its accompanying sympathetic plexus, is placed closely in front and medially to these nerves. The hypoglossal nerve is in close proximity since it leaves the skull through the adjacent anterior condyloid foramen. In their more peripheral courses, the four nerves remain in close approximation to a point slightly below the tip of the mastoid, in the so-called retroparotid space. More laterally placed, in the coronal plane of the head, lie the seventh and fifth nerves.

Although reports of injuries to the jugular vein and carotid artery in association with injuries to the last four cranial nerves are rare in the World War I literature, such a combination of nerve and vessel injury must have occurred with some frequency. Among these infrequent observations of similar cases is that noted by Heyrovsky[12] who described involvement of the last four cranial nerves with a spurious aneurysm of the vertebral artery. Aneurysmorrhaphy was successful, but as in Case 1 (*infra vide*) the neural defect showed no evidence of improvement. That they were not reported would indicate that these cranial nerves may be injured without concomitant

vascular injury, as noted by Vernet, or that such vascular injuries may occur and heal spontaneously, or that the vascular injury was so formidable that, lacking modern methods of shock control, these cases did not survive. It is obvious that grave vascular injuries of a chronic, progressive nature do occur with the various classical syndromes indicating involvement of the last four cranial nerves, and that, furthermore, the control of such vascular injuries is of paramount importance in the rehabilitation of the wounded soldier.

Case 1.—*C. J. H., Hosp. No. 991: Gunshot wound of face. Removal of .44 caliber bullet in left occipitomastoid region. Involvement of V, VII, X, XI and XII cranial nerves and production of A-V aneurysm. No improvement in neurologic status after resection of vascular lesion.*

On November 10, 1942, this soldier was struck in the left malar region at close range with a French .44 caliber revolver bullet. The missile traversed the face, the lateral aspect of the neck, and became imbedded in the soft tissues of the left occipitomastoid region. He staggered back under the impact of the bullet but did not lose consciousness. He noticed at once loss of feeling in the mouth and lower left side of the face. His voice was hoarse, and he was unable to fully lift the left arm from his side. There was no extensive external hemorrhage, but the tissues of the side of the neck swelled considerably. Within a few days he became cognizant of a loud buzzing in his left ear. Upon occasion, his pulse rate would become very rapid. On December 22, 1942, the bullet was removed from the left occipital region, and on February 22, 1943, the left common carotid artery was ligated, without change in his symptoms.

On admission to the Ashford General Hospital, two months later, there was a small scar over the left malar region, denoting the wound of entry. There was a short operative scar 4 cm. mesial to the tip of the left mastoid. The tissues in this region appeared full in comparison to those on the right. Palpation revealed a deeply situated pulsating mass with a distinct systolic thrill. Auscultation noted a continuous bruit accentuated in systole with maximal intensity over the occiput which could be obliterated by deep pressure over the sternomastoid muscle. Arterial pulsation was absent over the left common carotid artery, the site of the earlier ligation. It was thought that this was an arteriovenous fistula of the cirsoid type, probably involving the occipital artery and vein deep to the muscles and posterior to the mastoid process. The question of involvement of the vertebral artery with accompanying veins in this region was considered.

Neurologic examination disclosed the following findings of significance. The pupils were of equal size, and there was no enophthalmus. There was analgesia and anesthesia over the third branch of the left fifth nerve; there was loss of taste over the anterior two thirds of the left side of the tongue; the soft palate was pulled to the right upon phonation, and the gag reflex was absent on the left side; the voice was hoarse and laryngoscopy disclosed a left abduction palsy; the left arm could not be abducted above 90 degrees, and there was atrophy of the left sternomastoid and of the lower two-thirds of the trapezius muscles; the superior third of this structure was incompletely paralyzed. The left side of the tongue was atrophic and furrowed and protruded to the left (Fig. 1).

The neurologic diagnosis was involvement of V, special visceral afferent (taste) of VII, IX, X, XI, and XII cranial nerves, similar to those syndromes reported by Collet, Vernet and Sicard, with the addition of the V and VII nerve lesions. To this was added the diagnosis of an arteriovenous aneurysm, probably of the occipital vessels.

An excision of these vessels was carried out on June 15, 1943. A semicircular

incision beginning over the left mastoid muscle was carried upwards over the base of the skull to the midline. The deep muscles were cut and retracted caudally. The external carotid artery was ligated, but this had but little if any effect upon the cirsoid aneurysm, which could be seen and felt at the base of the skull. This aneurysm was excised by ligating and cutting numerous arteries and veins which were seen to communicate with vessels entering the skull. Considerable bleeding was encountered,

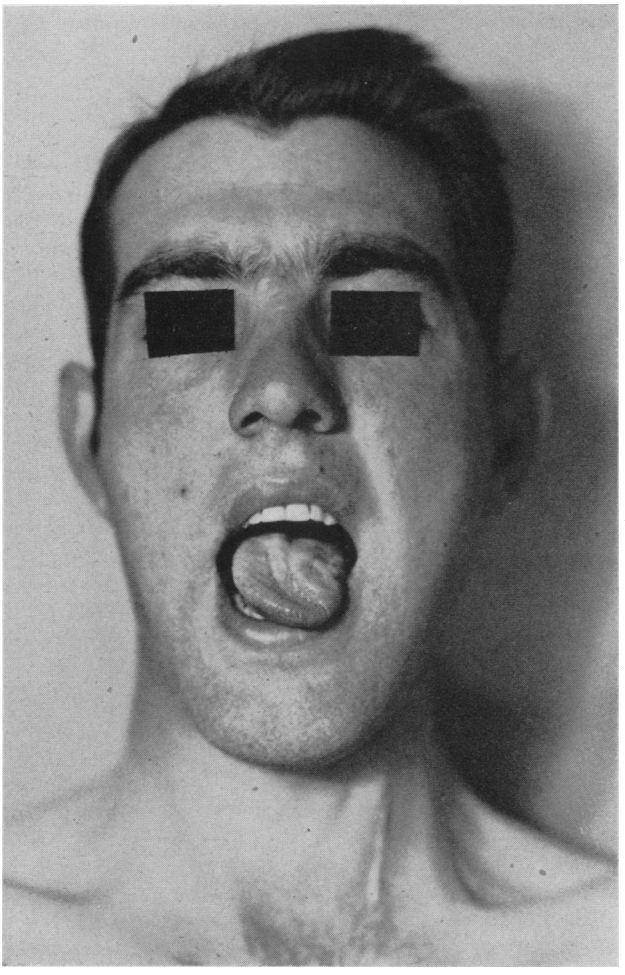

FIG. 1.—Case 1: Paralysis of left hypoglossal nerve.

which was controlled by coagulation and by the use of fine silk ligatures. At the end of the operation the bruit and thrill had disappeared.

Two weeks later, as a result of a friendly wrestling match, the wound was opened and a severe secondary hemorrhage occurred. This was controlled by hemostats which were left in place, and by packing. Following this the vertebral artery was ligated, which apparently controlled the bleeding since no further hemorrhage occurred with the removal of the clamps and packing. The wound healed uneventfully. There was no return of the bruit and thrill. There was no change in the neurologic findings upon discharge three months later and upon review examination three months after discharge.

Case 2.—*H. C. H., Hosp. No. 1397: Gunshot wound left side of neck, April 31, 1943. Involvement of cranial nerves VII, X, XI, XII, and second cervical with incomplete cicatricial occlusion of left common carotid artery. Artery freed from cicatrix. Progressive improvement in neurologic defect.*

On March 31, 1943, this soldier was injured by aerial bomb fragments which struck the left side of his neck, left thigh, and left frontal region of the scalp. He was unconscious for two hours. Severe hemorrhage persisted from the neck wound, and two plasma transfusions were given during the first 24 hours after the injury. Forty-eight hours later the neck wound was explored, and foreign bodies were removed. At this time he was unable to talk, move the left side of his face, or shrug the left shoulder. He noted loss of feeling over the lower third of the left side of the face. He had some difficulty swallowing fluids. Three weeks after the injury, the aphonia cleared, and his voice became progressively less hoarse. Both facial and shoulder movements improved slowly but steadily. From that period in his convalescence until his admission to Ashford General Hospital on May 10, 1943, he noticed an intermittent singing noise in the left ear.

There was a small, stellate, puckered scar below and anterior to the tip of the left mastoid. Palpation over the scar and in the anterosuperior cervical triangle disclosed a small, tubular expansile mass. Auscultation over the mass revealed the presence of a harsh, continuous bruit, accentuated in systole, which could be obliterated by compression of the common carotid artery.

Neurologic findings included an apparent enophthalmus on the left, moderate weakness of the musculature about the left angle of the mouth, normal taste perception and hoarseness with an abduction paralysis of the left vocal cord. The superior third of the left trapezius muscle was atrophic with weakness of shoulder elevation, but with normal abduction of the left arm. The tongue was atrophic and protruded to the left. There was an area of hypalgesia and hypesthesia corresponding to the anterior fibers of the second cervical nerve.

The neurologic diagnosis was involvement of the VII, X, XI and XII cranial and the second cervical nerves. With the exception of the VII nerve disturbance this case was similar to the syndromes previously recorded. The more inferiorly and laterally placed site of injury in this instance explained the lack of involvement of the glossopharyngeal nerve. Surgical exploration of the expansile mass in the cervical region disclosed a cicatricial, incomplete occlusion of the common carotid artery with proximal dilatation of the vessel. The neurologic disturbance, with the exception of the abduction paralysis of the left vocal cord, improved steadily until discharge three months later.

The release of scar tissue about the carotid artery led to complete disappearance of the bruit and thrill, a finding worthy of note, since apparently scar tissue contracting about a vessel may produce the cardinal physical findings of an arteriovenous fistula.

Case 3.—*R. Z., Hosp. No. 3007: Gunshot wound, 20 mm. shrapnel, left mastoid region on December 3, 1942. Involvement of cervical sympathetic, VII, IX, X, XI and XII cranial nerves, associated with A-V aneurysm of internal carotid artery and internal jugular vein and evidence of increased intracranial pressure. Resection of A-V aneurysm. Moderate improvement in neurologic status.*

This soldier was injured on December 3, 1942, by pieces of shrapnel from a 20 mm. shell which struck the left mastoid region and left shoulder. The former wound bled profusely for a few seconds, and then hemorrhage ceased spontaneously. Three days later the mastoid-cervical wound was explored and a foreign body removed. On the following day, he noted a buzzing sound in the left ear, the appearance of a swelling below the wound and for the first time, developed moderately severe generalized headache. There was weakness in the muscles of the lower half of the left side of the face, and his voice was hoarse. He noticed that choking would follow the ingestion of fluids, but that

solid food was tolerated without difficulty. When he became ambulatory, he realized that he could not abduct the left arm above 90 degrees. On April 3, 1943, the left common carotid artery and left internal jugular vein were ligated. On June 11, 1943, the A-V aneurysm involving these vessels was resected, both procedures being performed by Lt. Colonel William V. Cox, M.C. The patient's convalescence from these operations was without untoward incident. Neurologic examination was carried out in the Ashford General Hospital on August 30, 1943.

The site of the operative procedure showed nothing abnormal, and there was no evidence of recurrence of the arteriovenous aneurysm. Vision in the O.D. was 20/20, in the O.S. vision was reduced to hand movements, with fingers recognized in the

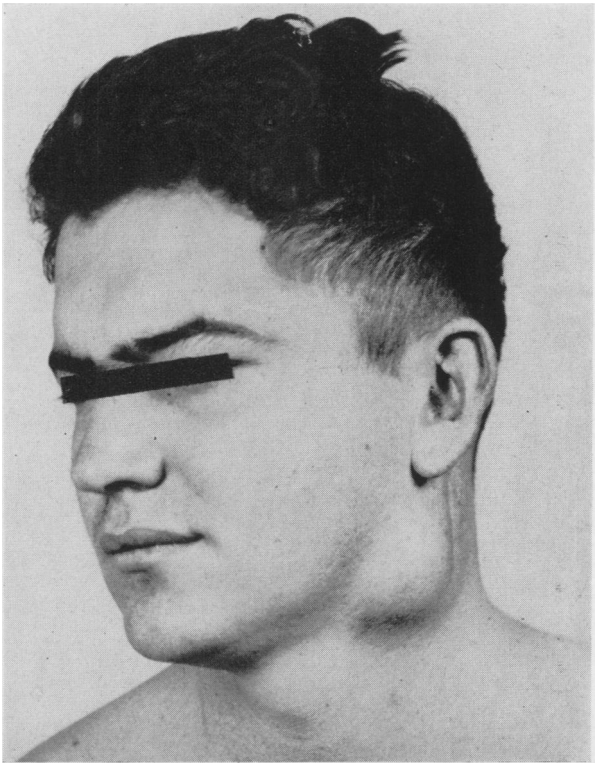

FIG. 2.—Case 3: Arteriovenous aneurysm of internal carotid-jugular vein. The vascular lesion and the narrowed palpebral fissure are apparent. (Case of Lt. Colonel William V. Cox.)

temporal quadrants. The optic nerve head of the right eye was normal. The left neuroretinal outline was clear, but the temporal half of the disk was pale. Adjacent to the temporal margin were two or three hyaline deposits and similar deposits were noted nearer the macula. An area just above the disk was pale. The changes in this fundus suggested an earlier papilledema with subsequent atrophy. Previous studies, prior to operation, had disclosed a bilateral papilledema of three diopters.

The pupils were equal and active to light stimulation and upon accommodation. There was intermittent conjunctival injection on the left. On July 9, 1943, Captain C. N. Eastman, M.C., had reported a narrowing of the left palpebral fissure, enophthalmus, some decrease in intra-ocular tension as compared to the right and normal side, loss of the ciliospinal reflex and loss of sweating over the left side of the face. There was

no change in sensation over the domain of nerve V. There was a contracture of some muscle fibers at the angle of the mouth, causing inversion of this structure. There was weakness of the risorius, triangularis and lower fibers of the orbcularis. There was slight loss of the normal palatal arch on the left and the soft palate pulled to the right upon phonation. The voice was hoarse and laryngoscopy revealed an abduction paralysis of the left vocal cord. The left sternomastoid muscle was functionless, and the left arm could not be abducted above 90 degrees.

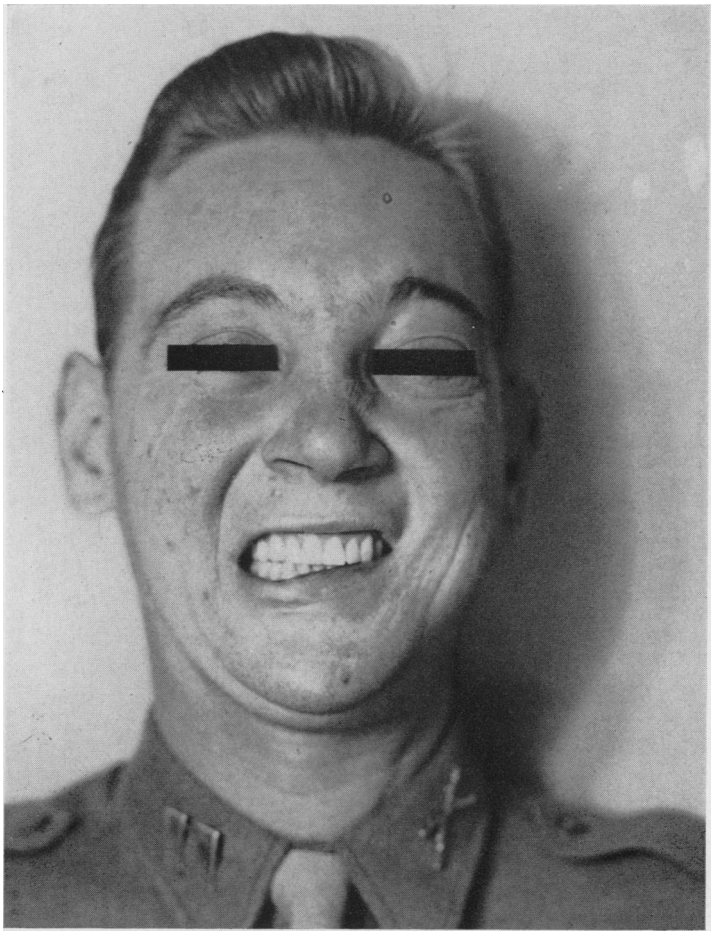

FIG. 3.—Case 4: Exophthalmos. Left seventh nerve paresis. Note wound of entry on lateral aspect of nose.

The neurologic diagnosis was incomplete involvement of the cervical sympathetic chain, and cranial nerves VII, IX, X, XI and XII, the syndrome described by Villaret. All components of the neurologic picture had improved considerably except those involving cranial nerves X and XI. Among six gunshot wounds of the last four cranial nerves described by Vernet with Lanvois and Patel is one quite identical with this case, but concerning which no postoperative course is available.

From the overseas clinical record it is clear that this patient had bilateral papilledema of three diopters before operation. This would suggest an increase in pressure in the venous circulation of the brain. Such a course is not incompatible with what is known

of the physiology of an arteriovenous fistula, where arterial blood at an increased pressure enters the venous system (Fig. 2).

Case 4.—*J. D. M., Hosp. No. 1345: Gunshot wound of face and neck. Involvement of V, VII, and XIth cranial nerves with development of arteriovenous aneurysm of cavernous sinus and internal carotid artery. Ligation of common carotid artery.*

On December 12, 1942, this officer was struck by a .30 caliber bullet. The missile entered 1 cm. mesial to the inner canthus of the left eye, and made its exit beneath the left mastoid region. He became unconscious at once and remained so for 24 hours. He was confused and drowsy for the following four days. Débridement was carried out on the evening of the injury, and sequestrectomy of a portion of the condyloid process of the mandible was done ten days later. He early appreciated numbness along

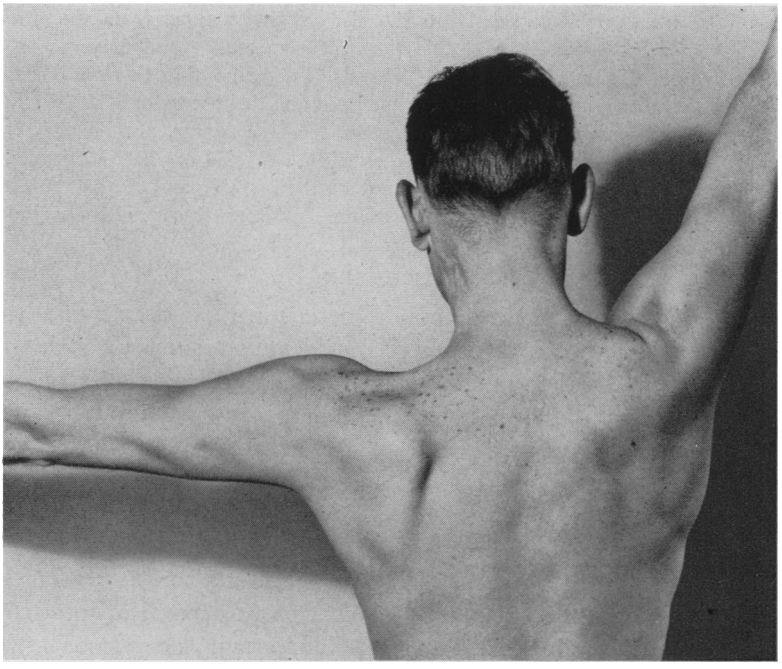

FIG. 4.—Case 4: Paralysis of spinal accessory nerve, left. Note wound of exit beneath mastoid region.

the lower half of the left side of the face, weakness of the left facial musculature and an inability to abduct the left arm above 90 degrees. From the time of adequate orientation, he had noted a protrusion of the left eyeball which had never diminished in size. Occasionally he reported hearing a "roaring sound like the ocean" in his left ear.

The striking feature upon first examining this officer was the moderate proptosis of the left eyeball. There was conjunctival edema and vascular congestion. There was a small, round scar on the lateral aspect of the nose 1 cm. from the inner canthus of the left eye. Below the tip of the left mastoid was a 2 x 4 cm. scar which was adherent to the deep structures of the neck.

Neurologic examination disclosed normal direct and peripheral vision. The left neuroretinal outline showed minimal blurring along its nasal margin. The retinal arterioles pulsated, and the veins were full. There was hypalgesia and anesthesia over the third branch of nerve V. There was almost complete paralysis of the circumoral musculature on the left side. There was pronounced atrophy of the sternomastoid muscle on the left. The trapezius muscle was atrophic with marked winging of the vertebral border of the scapula and loss of abduction of the left arm above 90 degrees (Figs. 3 and 4).

The supra-orbital veins were engorged, and a distinct, continuous thrill could be felt in both the upper and lower left eyelids. This thrill was transmitted into the neck over the carotid vessels. Over the left eye, forehead and left side of the neck there was a harsh, continuous roaring bruit, accentuated in systole. The left eyeball pulsated slightly. The whole picture was one of pulsating exophthalmos and was thought to be due to a communication between the cavernous sinus and the left internal carotid artery. On May 26, 1943, the left common carotid artery was partially compressed with an aluminum band, and it was felt that about 80 per cent of the blood flow through this vessel was stopped. Following this there was definite improvement in the exophthalmos, and, on June 23, 1943, the carotid artery was again exposed, together with its terminal branches. At this time temporary occlusion of the common, internal, and external carotid vessels did not obliterate the bruit previously described, and it was felt, therefore, that a large blood supply to the fistula was coming from the other side and from the vertebral vessels. At this time the common carotid artery was doubly ligated with braided silk and the wound closed. Following this there was additional improvement in the exophthalmos, although the bruit and thrill were only slightly affected.

The patient was seen in consultation by Dr. Rudolph Matas, and he agreed in the opinion that no further operative procedure should be carried out at this time.

The neurologic diagnosis was involvement of the V, VII and XIth cranial nerves associated with an arteriovenous aneurysm of the left internal carotid artery and cavernous sinus (Figs. 3 and 4).

DISCUSSION

Any specific syndrome of the cranial nerves in their extracranial courses depends upon the unpredictable vagaries incident to trauma through gunshot wounds and upon the time interval at which the neurologic observations are made. Although it is obvious that their close anatomic grouping sets the stage for multiple involvement of the last four cranial nerves, yet their separation into syndromes is of dubious neurologic import. From a study of these cases, and from other traversing wounds of the face and neck without significant change at the time of neurologic study, several observations of prognostic import seem worthy of comment. In the first place, injuries of nerves V and VII of transient or of rare permanent nature, may be engrafted upon the usual classical syndromes of the last four cranial nerves. This superimposed neurologic defect has exhibited a strong tendency toward spontaneous regression in the patients under observation. In the second place, a lesion of the last four cranial nerves, whether acute or of a chronic and perhaps regressive character, may incapacitate the patient in only two significant respects. In Case 3, there was pronounced difficulty in the early days after the injury, in swallowing fluids. Six months after the injury, the persistent abduction paralysis of the left vocal cord precipitated respiratory collapse at the beginning of induction of anesthesia, preliminary to the introduction of an intratracheal tube. A similar episode occurred in this hospital under identical circumstances in the treatment of Case 2. The involvement of the external branch of the spinal accessory nerve has shown little tendency toward improvement and the loss of abduction of the upper extremity above 90 degrees remains as a defect of considerable magnitude. Direct exploration of the region of the jugular foramen with the view of decreasing this neurologic disturbance has not been carried out or contemplated in these cases.

Finally, the experience that vascular injuries are associated with many injuries of the last four cranial nerves may not represent the true frequency of the combined injury, since this hospital has been designated as a Vascular Center. The combined injury, whether immediately fatal from hemorrhage or amenable to treatment, must be fairly common because of the anatomic contiguity of the vascular and neural structures.

The vascular lesion may include, in individual cases of a chronic nature, cicatricial occlusion, A-V aneurysm, false aneurysm or even similar lesions from indirect violence similar to Case 4. The fact that the vascular lesion in Case 2 was unrecognized previously is worthy of comment. The vascular injury may bear no relationship to the extent or duration of the neural damage except that of a concomitant injury. In these patients at least, the neural disturbances developed immediately following injury and were not favorably influenced by repair of the vascular lesion. That the aneurysmal mass may influence adversely the extent of neural injury in some instances is not disputed.

II—VESSEL-NERVE INJURIES OF THE UPPER EXTREMITY

It is the consensus of opinion of former writers on this subject that neural complications are much more common in aneurysms of the upper extremity than in those of the lower extremity. Thus Fromme,[13] in a long, carefully documented article, recorded 13 aneurysms of the upper extremity with neural changes out of a total number of 16. Makins[14] stated that 25 per cent of the subclavian aneurysms showed involvement of the brachial plexus and out of 54 cases involving the axillary and brachial arteries, 43 per cent showed involvement of neural structures. In Maurer's[15] series of 71 aneurysms of the upper extremity, approximately 25 per cent showed neural complications. On the other hand, in Makins' extensive series of 170 femoral artery injuries and 85 popliteal artery injuries, concomitant nerve injury was reported as rare. Illustrative of the combined vessel-nerve injuries to the first part of the axillary artery is that described in the following case:

Case 5.—*D. F. H., Hosp. No. 2869: Traversing gunshot wound of left infraclavicular space on March 28, 1943, followed by paralysis of left arm for two months. Subsequent resection of false aneurysm of first part of left axillary artery with slow improvement in neurologic status.*

On March 28, 1943, this soldier was struck by a rifle bullet in the left infraclavicular space which traversed the apex of the axilla and made its exit lateral to the tip of the left scapula. External bleeding was minimal at the time of injury and a massive subcutaneous hematoma did not develop. The left arm became paralyzed at the moment of injury and did not exhibit evidence of improvement for two months. Return of function was marked first by flexion of the forearm, and then by extension of the wrist. Sensory loss was noted over the entire arm after injury, but regressed slowly until it involved the outer aspect of the forearm, the thumb, and first three fingers. After hospitalization in the Zone of the Interior, an aneurysm of the first part of the axillary artery was disclosed, and he was referred to the Ashford General Hospital for treatment.

Examination noted a small, well healed wound of entry 4 cm. below the outer third of the left clavicle and a larger, irregular scar denoting the wound of exit lateral

to the tip of the left scapula. In the left infraclavicular space an oval, expansile mass, 3 x 2 cm. was palpable. Upon auscultation, a faint, systolic bruit was audible, which radiated into the distal third of the extremity. Blood pressure in the right arm was 122/78, in the left 110/76. The radial pulse on the left was weaker than on the right; the ulnar pulse was absent.

Neurologic study noted complete involvement of the musculocutaneous nerve, an incomplete lesion of the median nerve and minimal disturbance of the radial nerve, with corresponding muscle dysfunction. There was an area of hypalgesia and anesthesia corresponding to the sensory distributions of the lateral antebrachial cutaneous and median nerves. On August 16, 1943, a false aneurysm of the first part of the left axillary artery was resected. At operation, an extensive gap in the musculocutaneous

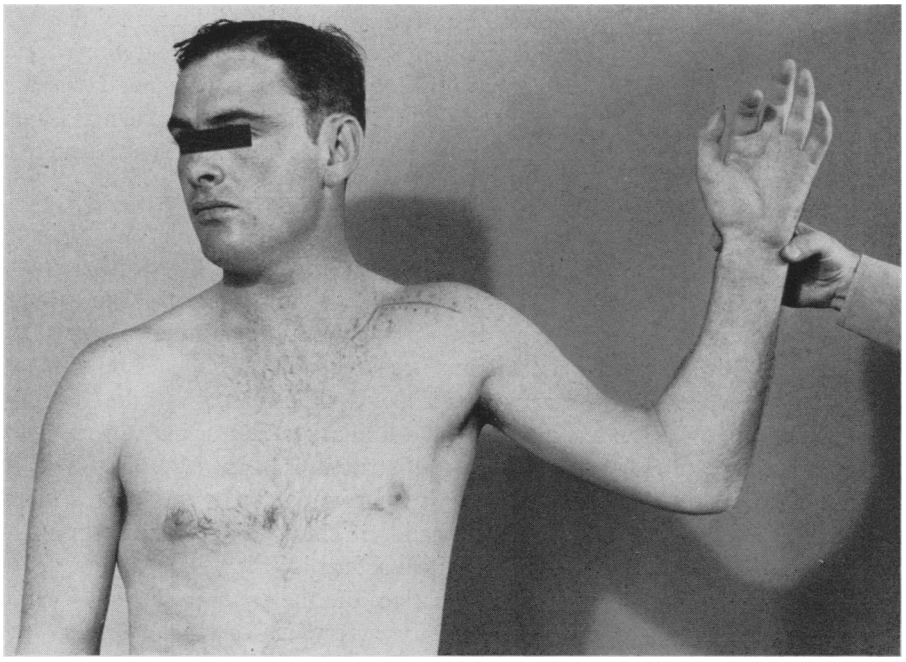

FIG. 5.—Case 5: Note operative approach to aneurysm of axillary artery and neural defects.

nerve was observed. The mass of the aneurysm compressed the median nerve in particular. There was no demonstrable change in the vascular supply to the extremity following operation. Beginning improvement in the neural defect is detectable at this time, three months after operation (Fig. 5).

Case 6.—*J. K. C., Hosp. No. 4246: Rifle wound of right chest with subsequent shrapnel wound of right shoulder on April 23, 1943. Early paralysis of right arm with rapid functional recovery. Residual serratus anterior and triceps muscles paresis. Arterial aneurysm of lateral thoracic artery resected.*

This soldier sustained a rifle bullet wound of the upper right chest on April 23, 1943. He remained on the field under fire until the following day when he was again injured by a shrapnel fragment in the right shoulder. At this time, the right arm was paralyzed with the hand and fingers in flexion spasm. The hand and arm recovered normal function, as far as the patient could tell, in the following two months. Chest pain persisted, and he was evacuated to the Zone of Interior.

Upon admission, October 25, 1943, a rounded, pulsating mass was palpated high in the right axilla in the course of a routine examination. It appeared to be separate

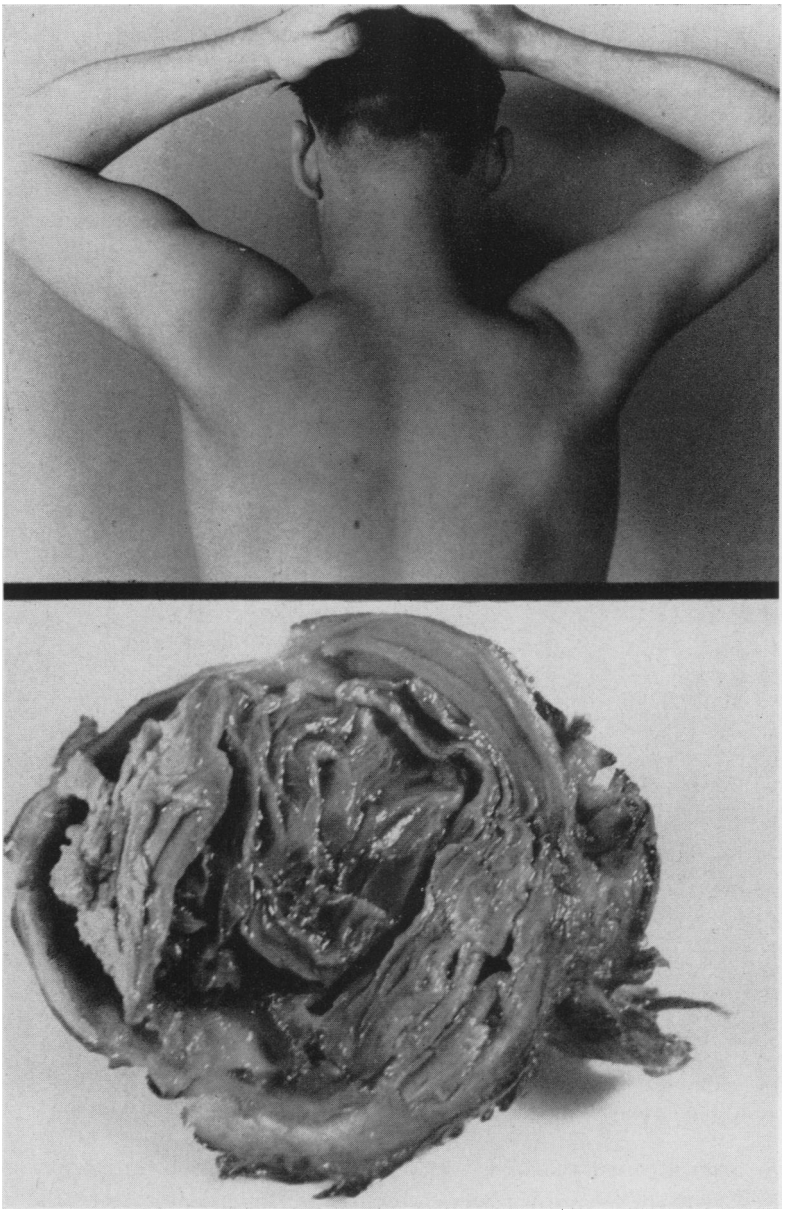

FIG. 6.—Case 6: Note triceps atrophy. Below, bisected aneurysm of lateral thoracic artery, showing lamination of clot.

from the pulsation and course of the axillary artery. A systolic nontransmitted bruit was present over the mass and both pulsation and mass could be obliterated by subclavian artery compression. A small scar was present on the anterior aspect of the right shoulder. Neurologic examination disclosed atrophy of the triceps muscle with associated weakness of extension of the upper arm. The muscle mass of the right deltoid was diminished on the right and early abduction movement of the right arm was weak. There was moderate flaring of the vertebral tip of the right scapula.

On November 2, 1943, exploration of the right axilla disclosed an arterial aneurysm of the long thoracic artery measuring 4 cm. in diameter. The long thoracic nerve curved around and was imbedded in the wall of the aneurysm. After resection of the aneurysm inspection of the axillary and radial nerves revealed no gross abnormality (Fig. 6).

In Makins' series, nerve injuries occurred in ten of 43 instances of injury to the brachial artery. Of striking interest is the fact that only one arteriovenous aneurysm was found in Makins' patients as compared with 12 of the arterial variety. One A-V aneurysm of the brachial artery with associated nerve involvement was encountered in this series. Due to associated infection, it has not as yet been treated. A brief description of this patient is noted under Case 12.

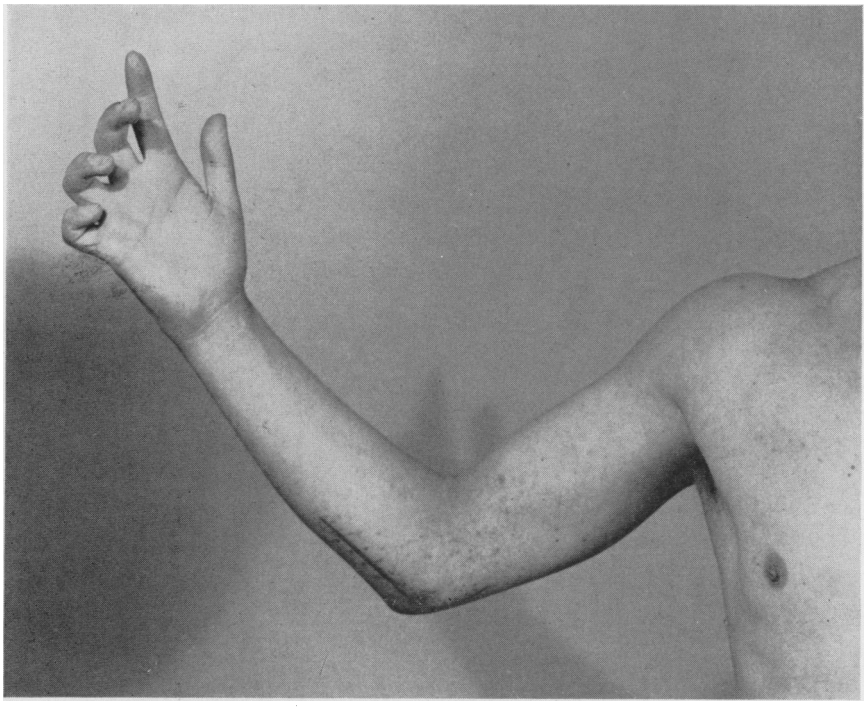

FIG. 7.—Case 7: Postoperative appearance following endo-aneurysmorrhaphy of brachial artery. Note obvious ulnar nerve deformity.

Two arterial aneurysms of the brachial artery in the antecubital space associated with nerve injury were observed. Both demonstrate the value of resection of the aneurysm as compared to endo-aneurysmorrhaphy when concomitant nerve injury is present.

Case 7.—*E. W. B., Hosp. No. 1403: Gunshot wound of right upper forearm. Immediate numbness of ulnar aspect of hand with weakness of intrinsic hand musculature. Obliterative endo-aneurysmorrhaphy of brachial artery with second-stage extraneural neurolysis of ulnar nerve.*

This soldier was shot by a rifle bullet on March 23, 1943. The missile entered the lateral aspect of the middle third of the right forearm, traversed the arm diagonally and made its exit slightly above the medial condyle of the humerus. The wound of exit

bled profusely and a tourniquet was applied. He noticed immediately weakness of the hand grip and loss of feeling along the inner aspect of the forearm, the ulnar aspect of the hand, and the fourth and fifth fingers. There was a transient infection in the wound of exit.

Examination of the right upper extremity, May 7, 1943, showed a tubular swelling of the proximal half of the forearm with a fluctuant area 4 cm. below the medial condyle of the humerus. There was a systolic pulsation together with a systolic bruit over the fluctuant area. Elsewhere the skin and subcutaneous tissues were firm and indurated without local heat or change in the overlying skin. Pressure in the region of the ulnar nerve adjacent to the wound of exit caused a tingling paresthesia. There was loss of function in the flexor carpi ulnaris and the two medial heads of the flexor digitorum profundus and the intrinsic musculature of the hand innervated by the ulnar nerve. The little finger and the ulnar aspect of the hand were analgesic and anesthetic. There was a narrow band of hypalgesia along the lateral margin of the lower half of the forearm. Obliterative endo-aneurysmorrhaphy of the brachial artery was done on May 17, 1943, with no alteration in the peripheral vascular supply to the hand. On July 14, 1943, the ulnar nerve was found divided in the thick scar tissue at the site of the aneurysmal sac. An end-to-end anastomosis was carried out with tantalum wire. No return of function is evident at the end of four months (Fig. 7).

Case 8.—*H. A. B., Hosp. No. 3009: Gunshot wound of left forearm. Immediate numbness and weakness of hand. Arterial aneurysm of brachial artery in antecubital space. One-stage resection of aneurysm and neurolysis of ulnar and medial nerves. Slow improvement in neurologic status.*

This patient was shot by a .30 caliber rifle bullet on July 3, 1943. The missile entered the left forearm on its anterior and lateral aspect and emerged just above and posterior to the medial epicondyle of the humerus. He noticed immediate numbness and weakness of the hand. There was marked swelling of the elbow, forearm, and hand within two hours of the injury but there was no external hemorrhage. On the 17th day following injury, an incision was made below the antecubital space and much blood clot was evacuated. On admission, August 16, 1943, slight motion of the wrist and of the third finger had appeared. There had been no improvement in the original sensory loss.

Healed wounds of entry and exit were noted corresponding to the path of the missile. The left upper forearm was swollen and indurated. An oval mass, 6 cm. in length, was palpable on the medial aspect of the forearm below the antecubital fold. There was an expansile pulsation in the mass, synchronous with cardiac systole. A loud systolic bruit was audible upon auscultation. Compression of the brachial artery in the arm caused the mass to become smaller and the bruit to disappear. The ulnar pulse was absent. The radial pulse was weaker on the left side than on the right.

Motor examination of this extremity showed disuse atrophy of the arm musculature. Minimal radial extension of the wrist was possible. Slight extension of the third finger at the metacarpophalangeal articulation was elicited. No other movements of the hand musculature were possible. There was marked contracture of the flexor tendons to all fingers. The entire hand showed cutaneous atrophy and loss of sweating. Analgesia and anesthesia were present over the sensory distributions of the ulnar and median nerves in the hand.

On September 7, 1943, the arterial aneurysm was resected. The median nerve was compressed by the mass of the aneurysmal sac and the ulnar nerve was buried in a mass of scarred, indurated muscle tissue medial to the aneurysm. It was observed that widespread muscle damage was present in the bellies of the flexor musculature of forearm. Slight improvement in the neurologic status has been evident since operation (Fig. 8).

The peripheral nerve injuries visualized at operation in the preceding four patients differed grossly in but two respects from peripheral nerve injuries observed in the absence of vascular injuries. In the first place, to

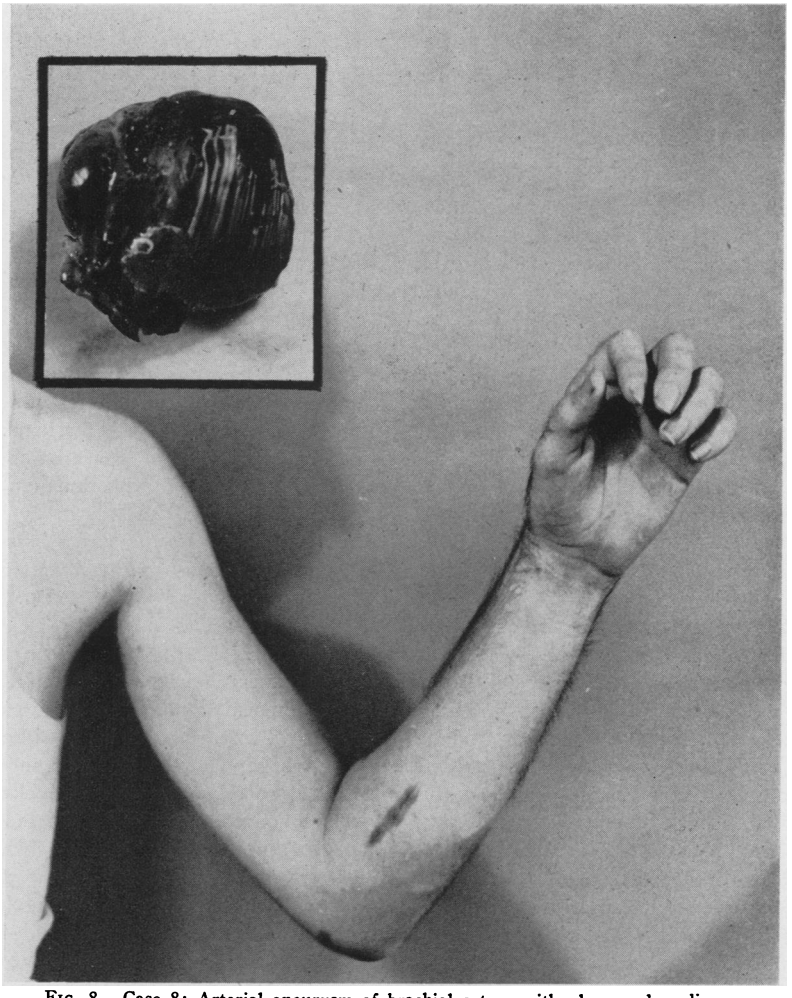

FIG. 8.—Case 8: Arterial aneurysm of brachial artery with ulnar and median nerve paralysis. Preoperative photograph. Note drainage scar.
(Insert) Operative specimen.

the usual pathologic findings of complete or partial nerve section, contusion or compression by scar tissue, is added the factor of compression by the mass of the aneurysmal sac. It is noteworthy in these cases, and in the few recorded in detail in the earlier literature, that the neurologic defect usually appears full blown at the moment of injury. Spontaneous return of neurologic function rarely appears and operative repair of the neural pathology is mandatory. In the second place, the presence of a vascular lesion, be it arterial transection or aneurysm with concomitant nerve injury, tends to exaggerate and make permanent the existing neural dysfunction through local and general alteration in the nutrition of muscle fibers. The frozen, contracted hand, resulting from acute arterial occlusion is well recognized, and if this deleterious factor be added to a neural defect, the resulting deformity may be irremediable.

It is of interest that in Case 7 the question of abscess formation rather

than arterial aneurysm was considered, and in Case 8 actual incision was done. In Fromme's Cases 1 and 25, similar supposed abscesses were incised with resultant severe hemorrhages, and in Case 46, identical with the last two of this series, the diagnosis of abscess was entertained briefly.

Finally, it may be stated, unequivocally, that a single, inclusive surgical approach to the vascular and the neural lesions is preferable to surgical repair of either lesion at separate operations.

III—VESSEL AND NERVE INJURIES OF THE LOWER EXTREMITY

Peripheral nerve injuries occur with arterial or arteriovenous aneurysms of the lower extremity but with less frequency than with those of the upper extremity and neck. They may involve the sciatic nerve in traversing wounds of the thigh, and the peroneal and tibial components in large, open wounds of the popliteal space. In only five of 85 instances of popliteal artery injury were neural structures involved in Makins' series, and the incidence in other major reports is correspondingly low.

Case 9.—*J. C. B., Hosp. No. 2109: Machine gun wound of left femoral region. Immediate paralysis and loss of sensation in extremity with rapid return of function. Resection of A-V aneurysm of common and deep femoral artery and vein.*

This officer was struck in the left femoral region with a machine gun bullet on April 29, 1943. A perforating wound of the thigh was sustained with the wound of exit in the left buttock. This extremity was paralyzed and made anesthetic at the time of impact, but motor and sensory defects cleared within a few hours. A profuse external hemorrhage occurred, which was controlled by pressure. There was no evidence of circulatory deficiency, but pain in the calf and popliteal space developed early. This distress increased with the appearance of a harsh bruit over the femoral triangle, of dilated leg veins and some peripheral cyanosis. On June 11, 1943, the left femoral artery and vein were ligated proximal to the fistula without alteration in the peripheral circulation and without effect upon the aneurysm. At this time, cardiac studies were normal.

Upon admission, July 11, 1943, examination showed induration in the left femoral triangle. About 8 cm. below Poupart's ligament was a small wound of entry beneath and lateral to a well-healed surgical incision. A second surgical incision was situated lateral to the first. The scar representing the wound of exit was noted in the left buttock. Beneath the larger incision a loud, harsh bruit was audible throughout systole and diastole, accentuated during the former phase. Occlusion of the femoral artery above the fistula diminished but did not cause the bruit to disappear. Such occlusion caused a transitory slowing of the pulse rate. The peripheral pulses were absent in this extremity and there was slight, dependent cyanosis of the foot. There was definite enlargement of the heart to the left, confirmed by fluoroscopy. Neurologic examination demonstrated only a diminution of the left knee kick and weakness in dorsiflexion of the left great toe. There were no sensory changes. The left sciatic nerve was somewhat tender and pressure over it increased the peripheral pain.

On August 4, 1943, quadruple ligation and resection of the arteriovenous fistula was performed. His convalescence was uneventful and the radiating pain slowly diminished.

Case 10.—*M. J. R., Hosp. No. 2957: Multiple injuries of trunk and extremities from land mine, including extensive laceration of right popliteal space. Development of popliteal A-V aneurysm with involvement of lateral cutaneous sural nerve. Quadruple ligation and excision of aneurysm, neurolysis.*

This officer in the Medical Corps received multiple injuries of the trunk, abdomen, upper and lower extremities from the explosion of a land mine in April, 1943. He re-

mained dazed and disoriented for several days as the result of blast concussion. At this time he recognized the presence of a right ulnar nerve paralysis and appreciated an area of numbness over the lateral aspect of the right lower leg. He stated that there was a profuse hemorrhage from a broad laceration of the right popliteal space at the time of injury, necessitating several plasma and whole blood transfusions. One month following the injury, the ulnar nerve paralysis began to recede. At this time, he became conscious of a continuous thrill in the popliteal space and was the first to recognize the presence of arteriovenous aneurysm of the popliteal vessels. He noticed no symptoms referable to cardiac strain except shortness of breath, and he did not feel that the peripheral blood supply to the involved extremity was impaired.

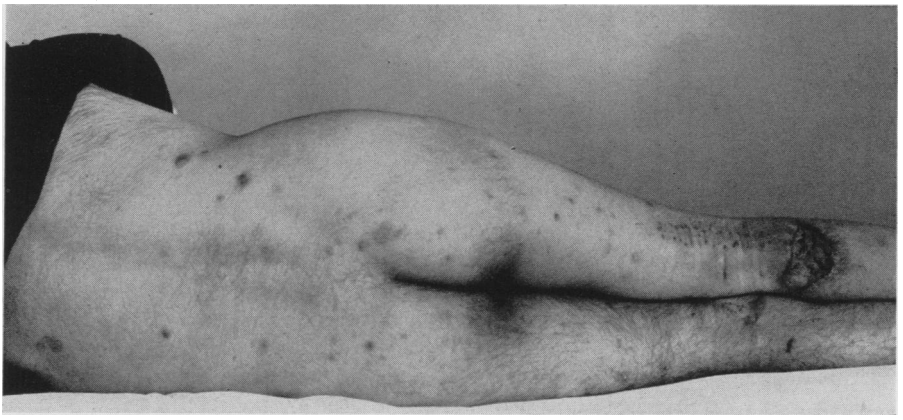

FIG. 9.—Case 10: Multiple injuries from explosion of land mine. Postoperative photograph, following excision of popliteal A-V. fistula.

Examination was noteworthy for the presence of 28 separate scars over the posterior surface of the trunk and lower extremities, characteristic of multiple injuries from the explosion of an antipersonnel land mine (Fig. 9). Over the inferior aspect of the right popliteal space was a broad, irregular scar, extending laterally beneath the tibial tubercle. Above this scar and rather high in the popliteal space the tissues appeared full. Upon palpation at this point, a strong continuous thrill could be felt, and upon auscultation, the bruit was continuous but accentuated with systole. There was no evidence of cardiac damage and the peripheral blood supply of the foot was normal. Hypalgesia and hypesthesia were present over the sensory distribution of the lateral cutaneous sural nerve. There was no evidence of motor dysfunction in this extremity. Neurologic study elsewhere disclosed only hypalgesia over the cutaneous area innervated by the ulnar branch of the medial antibrachial cutaneous nerve. On August 24, 1943, the arteriovenous aneurysm was excised after quadruple ligation. At operation, the peroneal nerve was compressed by scar tissue and an external neurolysis was done. The neurologic defect has improved steadily and he has returned to duty.

Case 11.—*P. W. P., Hosp. No. 3008: Shrapnel wounds of right popliteal space May 5, 1943. Profuse hemorrhage, leg pain and diminished peripheral circulation. Lumbar sympathectomy with improvement in circulation. Resection of popliteal A-V aneurysm. Normal neurologic examination.*

This soldier received wounds of the right knee, leg and left shoulder from fragments of an 88 mm. shell on May 5, 1943. Profuse hemorrhage occurred from a wound in the right popliteal space which was débrided and the hemorrhage controlled. No injury to major vascular or neural structures was reported. The right lower leg and foot remained cold and cyanotic, with considerable aching pain. On July 17, 1943, a right lumbar sympathetic ganglionectomy was carried out with partial relief of pain, and considerable improvement in the peripheral circulation.

Upon admission, August 29, 1943, there was a flat, healed scar in the right popliteal space and a second operative scar in the posterior upper third of the calf. A shrill bruit was audible over the popliteal space, extending throughout systole and diastole and accentuated during the former. Occlusion of the fistula did not change the pulse rate. The right posterior tibial pulse was absent, but the right foot was warmer than the left. There were no abnormal cardiac findings. Neurologic examination was normal (Fig. 10).

On September 6, 1943, quadruple ligation and excision of the arteriovenous aneurysm was done. There was no change in the peripheral circulation and his convalescence was uneventful. The tibial and peroneal nerves, although closely adherent to the fistulous mass, appeared normal at operation.

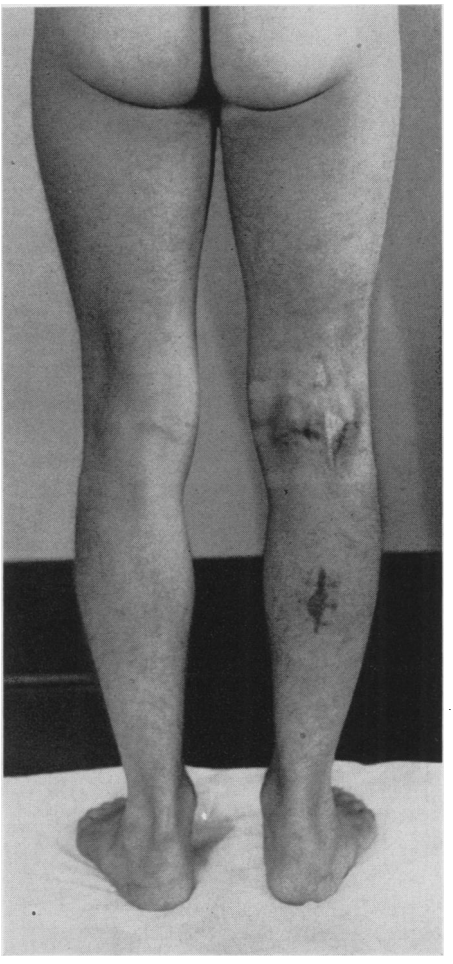

FIG. 10.—Case 11: Preoperative photograph of popliteal arterio-venous aneurysm.

Case 12.—*S. M., Hosp. No. 3528: Multiple wounds of right arm, chest and left leg on July 11, 1943. A-V aneurysms of left femoral and right brachial vessels. Minimal hypalgesia in left popliteal space. Quadruple ligation and an excision of femoral A-V fistula with aneurysmal sac on September 7, 1943.*

This patient, in brief review, developed an arteriovenous aneurysm of the right brachial artery, upper third, and a comminuted compound fracture of the right elbow, with associated nerve injuries and an A-V aneurysm of the left femoral vessels.

Upon admission to this hospital, a pulsating, expansile mass was noted on the superior and medial aspect of the left thigh. There was a palpable thrill and audible bruit lasting throughout systole and diastole, with systolic accentuation. Occlusion of the fistula did not alter the pulse rate. The peripheral circulation was adequate and there was no evidence of cardiac damage except a constant elevation of the pulse rate (110 per min.). Neurologic examination showed, in this extremity, only hypalgesia and hypesthesia in the popliteal space, suggesting involvement of some of the fibers of the posterior femoral cutaneous nerve. Quadruple ligation and resection of the fistula and adjacent aneurysmal sac were carried out on September 7, 1943. His convalescence was uneventful from this localized disturbance. Operation upon the brachial aneurysm has not yet been carried out because of local infection in the associated compound fracture (Fig. 11).

SUMMARY

It appears probable that more vascular and nerve injuries of various types will be seen in this war than in World War I, because of improved methods for the control of shock and infection and because of the increased incidence of multiple injuries.

The diagnosis and treatment of aneurysms and peripheral nerve trauma have not been discussed in this paper. However, it should be pointed out that certain basic principles in the treatment of aneurysms must be observed or else failure is sure to follow. For example, the treatment of aneurysm, whether it be arterial or arteriovenous, is never a matter of emergency unless it is rapidly progressing in size or has ruptured, or unless heart failure is impending. Time should be allowed for collateral circulation to develop, usually a matter of three or four months. The use of artificial means to develop collateral circulation, usually proximal compression of the vessels, should be employed. Moreover, the eradication of an arteriovenous fistula

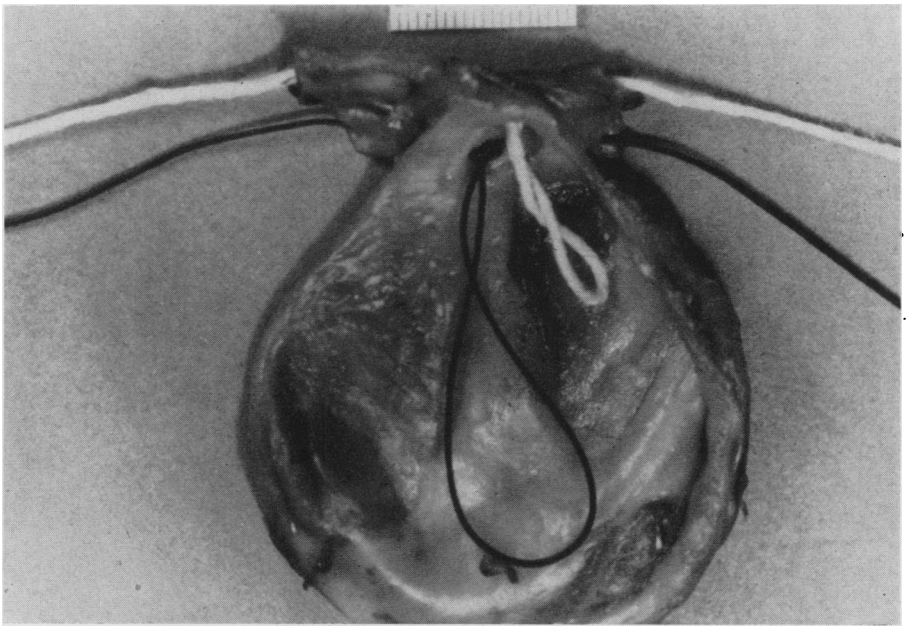

FIG. 11.—Case 12: A-V. fistula with communicating arterial sac. Note white string in artery and black string in vein.

is accomplished only by quadruple ligation and excision. Any other method usually results in recurrence. Partial measures, such as proximal ligation of the artery, may lead to gangrene of an extremity. Complete excision of an arterial aneurysm, together with nerve repair, as a single operative procedure, is the method of choice. When major vessels are involved and where the nutrition to the extremity is questionable, the endo-aneurysmorrhaphy of Matas is preferable. This applies particularly to the common femoral and other major arteries.

It is well recognized in all peripheral nerve surgery that a conservative attitude should be taken regarding technical procedures. This is particularly important in injuries associated with blood vessel trauma because of the added factor of compression by an aneurysm. This factor, producing added nerve damage in the false aneurysm, is not often present in arteriovenous fistulae, which are rarely large enough to produce pressure damage. Nerve injury

must occur at the time of formation of the fistula, through the same inflicting force, and may be due to concussion, contusion, or to actual section of nervous tissue.

In many instances, at the time of the original injury, there is evidence of extensive nerve damage. By the time that vascular repair is indicated, the neural damage may have resolved itself to a large extent, leaving a residual neurologic defect, which may be repaired at the time of operation upon the blood vessel.

Recognizing the slowness with which nerve regeneration takes place and desirous not only of attaining the best functional result, but also of making studies for future reference and treatment, the Surgeon General has adopted a policy of treating all peripheral nerve lesions until maximum hospital benefit has been obtained. Even where soldiers are returned to duty, they are ordered back to the hospital where last treated for periods of observation every three months.

Finally, it is emphasized that the traumatic syndrome of Vernet and Villaret, *viz.*, involvement of the last four cranial nerves, is often associated with blood vessel damage in the neck, due to the close proximity of these tissues.

REFERENCES

[1] Pollock, L. J., and Davis, L.: Peripheral Nerve Injuries. Paul B. Hoeber, Inc. New York, 1933.

[2] Avellis, G.: Klinische Beiträge zur halbseitigen Kahlkopflähmung. Berl. Klin., **40**, 1–26, 1891.

[3] Schmidt, E.: Cit. Pollock, L. J., and Davis, L.[1]

[4] Jackson, J. H.: Paralysis of Tongue, Palate and Vocal Cord. Lancet, Lond., **1**, 689–690, 1886.

[5] Vernet, M.: Syndrome du trou déchiré postérieur (paralysie des nerfs glosso-pharyngien-pneumogastrique-spinal). Rev. neurol., Par., **25**, 117–148, 1918.

[6] Collet: Sur un nouveau syndrome paralytique pharyngo-laryngé par blessure de guerre: Hémiplégia glosso-laryngo-scapulo-pharyngée. Rev. neurol., Par., **22**, 982–984, 1915.

[7] Sicard, J. A.: Syndrome du carrefour condylo-déchiré postérieur (type sur du paralysie des quartre derniers nerfs craniens). Bull. et mém. Soc. méd. de hôp. de Par., 3. s., **41**, 317–327, 1917.

[8] Villaret, M.: Le syndrome de l'espace rétro-parotidien postérieur. Paris méd., **23**, 430–431, 1917.

[9] Barbé, A.: Le syndrome nerveux de l'espace sous-mastoidien. Progrès méd., Par., **34**, 281–282, 1919.

[10] Körner, O.: Über Lähmungen der Nervi vagus, accessorius Willisii, hypoglossus und sympathicus durch Fernschädigung bei Halsschüssen. München. med. Wchnschr., **63**, 1429–1430, 1916.

[11] Pollock, L. J.: Extracranial Injuries of Multiple Cranial Nerves. Arch. Neurol. Psychiat., **4**, 517–528, 1920.

[12] Heyrovsky, J.: Operativ behandeltes Aneurysma spurium de Arteria vertebralis. Ztschr. f. d. ges. Neurol. u. Psychiat., **11**, 390, 1914–15.

[13] Fromme, A.: Über Kriegsaneurysmen. Beitr. z. klin. Chir., **105**, 293–323, 1916–17.

[14] Makins, G. H.: On Gunshot Wounds to the Blood Vessels: Founded on Experience Gained in France during the Great War, 1914–1918. John Wright and Sons, London, 1919.

[15] Maurer, A.: Nôte sur les plaies vasculaires récentés et leur trâitement d'aprés 444 observations. Mém. Acad. Chirurgie, **65**, 1156–64, 1939.