SERIAL sensory examinations in a large number of cases of nerve injury and suture, which were observed during a two and a half year period in an Army Neurosurgical Center, have shown that faulty localization of tactile stimuli after nerve suture is a valuable prognostic aid in determining the effectiveness of regeneration. By faulty localization is meant marked disturbance in the ability to localize a tactile stimulus within the cutaneous distribution of the involved nerve. Within the limits of the cutaneous distribution of a regenerated nerve, faulty reference occurs for both touch and pain. In the ensuing case reports both pain and touch modalities were tested systematically.

The phenomenon of faulty localization has often been attributed to poor regeneration of nerve fibers. As a matter of fact, it may be the earliest demonstrable evidence of nerve regeneration, and thus assumes considerable practical significance. On the other hand, when accurate localization accompanying a return of sensation occurs soon after nerve suture, it has been often misinterpreted as evidence of nerve regeneration. Despite the fact that faulty localization was described as early as 1895 and has been noted frequently since then by various workers, its true significance has not been common knowledge. John Mitchell,\textsuperscript{12} in reviewing certain Civil War cases of his father, Weir Mitchell, described in 1895 a brachial plexus injury showing this phenomenon, and noted a communication from W. H. Howell ascribing it to misdirection of regenerated nerve fibers. Trotter and Davies\textsuperscript{24} reported the phenomenon after experimental section and suture of cutaneous nerves in man. They say that faulty localization of cutaneous stimuli "... is by far the most valuable and least equivocal of all the evidences of regeneration. ... Peripheral reference is the earliest phenomenon of recovery and the last sign of abnormality." Boring\textsuperscript{3} in 1916 also described faulty reference as occurring 135 days after experimental section and suture of a human cutaneous nerve.

CASE REPORTS

Four illustrative cases will be reported. The first case demonstrates the fallacy of accepting ordinary sensory return in the cutaneous distribution of the involved nerve as a prognostic sign of regeneration after suture. The second case demonstrates that faultily localized sensations are carried by the

\* Presented before the Halsted Surgical Club meeting in St. Louis, May 1, 1947.
pathway of the sutured nerve. The third case illustrates the character of faulty localization, proving that the phenomenon is restricted to the limits of maximum distribution of the nerve involved. The fourth case shows the persistence of faulty localization after regeneration within the area of distribution of the involved nerve.

Case 1. C.B., a 20-year-old male, incurred a wound at the junction of the middle and lower thirds of the right forearm on Nov. 12, 1944, following which he had total anesthesia of the distal 2 phalanges of the 2nd and 3rd digits of the right hand. On Mar. 1, 1945, an extensive neuroma in continuity was resected and a 3 cm. gap was overcome in a suture of the median nerve. Five months following suture there was return of sensation in the entire median nerve distribution. Although the sensation was still definitely hypesthetic, ability to perceive touch was present throughout the median nerve distribution. Localization of tactile stimuli was not as accurate as on the opposite normal hand, but the limit of error was consistently less than 2 cm. Wide misses of 3 or more cm., or misses from one finger to another, as occur with typical faulty localization, were never observed. Additional doubts of the success of the neurorrhaphy were aroused by the absence of a rapidly advancing Tinel's sign. Furthermore, apparent sensory recovery had occurred gradually over a considerable period of time, rather than appearing suddenly with a rapid burst.

Because of the foregoing factors, the median nerve was re-explored in August 1945. Operation was performed under local anesthesia. Tantalum foil was removed and the enclosed suture line was found to be intact and appeared adequate. The nerve was infiltrated with procaine above the suture line. Electrical stimulation below the point of procaine infiltration gave no sensory response, indicating an effective nerve block. Sensory examination of the undraped hand was then carried out. There was no change in sensation in the median cutaneous area from that which was present prior to nerve block. The patient himself could detect no subjective sensory change.

Comment. This case demonstrates conclusively that the recovery of "accurate" localization of sensation—"accurate" in contrast to the marked faulty localization seen in regeneration after nerve sutures—must have been carried by pathways reaching the point stimulated through nerves other than the one sutured.

Case 2. L.W.S., a 28-year-old male, received a laceration of the right forearm on May 11, 1944. Because of a severe wound infection, surgery was delayed until Sept. 24, 1944, when a suture of the ulnar nerve was performed, overcoming a 4 cm. defect. Eleven months following operation there was sensory return with a good concentration of pain and touch spots in the previously anesthetic area. Marked faulty localization of stimuli was present with misses of as much as 8 cm. frequently being made.

Reoperation was performed on Sept. 7, 1945 under local anesthesia. Electrical stimulation of the nerve distal to the suture line gave a good sensory response. The nerve was then blocked with procaine following which electrical stimulation gave no response. Following the effective nerve block, examination of the hand demonstrated total anesthesia of the distal 2 phalanges of the 5th digit with complete disappearance of the faulty localization, even in the hypesthetic intermediate zone of the ulnar nerve. When the effects of the procaine block wore off, anesthesia of the hand regressed and faulty localization returned to its former status.

Comment. This case demonstrates that sensory recovery characterized by faulty localization had occurred over the pathway of the sutured nerve.

Case 3. E.A.T., a 19-year-old male, was wounded in the left forearm on Jan. 21, 1945.
There was complete motor and sensory paralysis of the median and ulnar nerves distal to the lesion. On May 19, 1943, suture of the median and ulnar nerves was performed in the lower third of the forearm, overcoming defects of 3 cm. and 5 cm. respectively. Postoperatively the sensory examination was unchanged and still showed anesthesia of all digits except the thumb. Seven months following operation there was a strong Tinel’s sign to the finger-tips, representing an advance of 20 cm. By this time there was return of sensations with marked overaction to stimulation and faulty localization. It was significant that stimulation of a point, for example, on the volar surface of the 5th digit, an area normally innervated only by the ulnar nerve, might be localized by the patient to the dorsum of that finger; stimulation of another spot might be referred to the 4th finger or to the hypothenar eminence, but never was the localization of such a stimulus referred to an area not innervated by the ulnar nerve (See Fig. 1). Similarly in respect to the median nerve autonomous distribution, wide misses might be made in localizing a stimulus, but the misses were never referred outside the domain of the median nerve.

Comment. This case illustrates marked faulty localization of stimuli which developed coincident with nerve regeneration. It also shows that faulty localization is confined to the maximum distribution of the nerve concerned.

Case 4. K.M., a 30-year-old physician, received a laceration of his left biceps tendon in civilian life in 1942. In December 1944, the tendon suture ruptured and a secondary repair was necessary while in the Army. Following this he noted sensory changes in the distribution of the lateral antebrachial cutaneous nerve. There was a central zone of total cutaneous sensory loss surrounded by a border zone of partial loss. The exact nature of the nerve trauma at operation is unknown, nor it is known whether the nerve was sutured. There was partial regression of the area of numbness which occurred shortly after the injury, evidently the result of overlap. Two and a half years after injury the area of sensory alteration corresponds to the distribution of the lateral antebrachial cutaneous nerve. The patient paid slight attention to the nerve injury until faulty localization was unwittingly noted by the patient himself in March 1947 in the following manner. He had a constant desire to scratch his left wrist for which he could find no apparent cause until he noted a small area of dermatitis in the left antecubital fossa. Scratching the area of dermatitis in turn referred sensation to the area on the wrist where he had felt an urge to scratch. Both the area of dermatitis and the referred area of itch were within the distribution of the lateral antebrachial cutaneous nerve.

The usual sensory examinations were employed and, furthermore, examinations with a condenser shock stimulator. The advantage of the latter is that it can be used as a source of stimulus that is independent of skin resistance, permitting light contact of the needle. In addition, with this form of stimulus pain has a lower threshold than touch. Repeated examinations were similar in result and have shown the persistence of faulty localization in this case. Sensory studies show that the density of sensory “spots” within the abnormal area is about half that in comparable areas of the normal arm. Using the electrical stimulator, faulty localization from stimulating a single point may be referred to one or two loci. In the case of two loci, both reference points may be remote from the site stimulated. At one reference point he may experience pain; at the other, touch. Increasing the strength of the electrical stimulus at such a point giving two loci of reference does not cause the two reference points to be blended into one. Instead the referred sensation may become more vivid.

Comment. This case demonstrates the persistence of faulty localization for 2½ years following injury. The local stimulus is still referred to distant sites. The vivid and distinct character of such points of faulty reference is well exemplified in a statement of Trotter and Davies: “Moreover, sensations which are referred are not diffuse in the sense of being vaguely localized,
they are in fact extremely definite and are placed by the subject with great precision on a spot which may be as much as a foot away from that stimulated.” On the basis of experience this patient can sometimes recognize faulty points of reference by means of delicate nuances that accompany the sensation. However, in spite of the long period of time that has elapsed, the sensory pattern has not been re-educated to the point where spots of faulty localization are suppressed or fused into a single “accurate” locus.

DISCUSSION

The gross cutaneous distribution of peripheral nerves is well known and is subject to few major variations. With few exceptions each of the major peripheral nerves has an autonomous cutaneous zone which that nerve alone supplies, and, in addition, an intermediate zone where sensation has a dual representation in which neighboring nerves share.13,14 Weddell25,26,27 has made extensive studies of the distribution of nerve fibers in the skin and finds that a rich network of nerve fibers exists. Physiologically it has been shown by Bishop3 and Tower22 that, although rich networks of nerve fibers exist in the skin, cutaneous sensation is punctate and high spots of acute sensation form a mosaic over the sensory surface. These high spots of acute sensation are not innervated by a single unbranched axon. Rather, they are reached by branches of the same axon, and by branches from neighboring axons.2,22,25
Stimulation of one spot thus results in impulses conducted over several separate axons, and a single axon can be stimulated from several sensory spots.

Tower’s theory of sensory localization based on nerve fiber distribution in the cornea and on action potentials from “few fiber” preparations of the long ciliary nerves is accepted as the mechanism of tactile localization.23 Briefly, it holds that a one-to-one relationship between cutaneous spot and cortex does not exist, but instead the central nervous system assigns a locus to a stimulus by integration of the repetitive firings in a single axon and the spatial summation of the several axons activated by a single tactile stimulus.

It is also generally assumed, in speaking of tactile localization, that tactile localization is “learned” in early childhood by associating repetitive sensations of touch with direct vision of the site touched. On what basis it is assumed is not clear, nor can any evidence be found that persons congenitally blind suffer any impairment of tactile localization.

As originally outlined by Head et al.,6,7 the area of altered sensation after a nerve is severed has certain qualities. The area of loss of all sensibility, called the autonomous zone, is the smallest, and next is a zone where deep sensibility is retained. Beyond this is a zone where cutaneous pain can be felt; next, a zone where light touch in addition is appreciated and only thermal sensibility is absent. These zones occur in roughly concentric circles.

Pollock12,14 has shown that, following severance of a nerve, with factors operating to prevent regeneration of that nerve, a gradual shrinkage of these concentric circles of sensory impairment occurs with a progressive improve-
FAULTY LOCALIZATION IN NERVE REGENERATION

ment of sensation, the so-called return of sensation from overlap of bordering nerves. Weddell, Guttmann, and Gutmann\textsuperscript{28} have shown how this sensory return from bordering nerves may occur; fibers from bordering nerves run-

![Diagram of volar surface of hand to illustrate anatomical limitations of faulty localization showing, as Pollock\textsuperscript{18} states, "The total loss of sensation is limited to a much smaller area than one would expect from the anatomical distribution. The residual sensibility [maximal zone] of a nerve is more extensive than its accepted anatomical distribution." Ulnar Nerve—Usual "autonomous" zone on little finger indicated by solid black. Usual maximal zone indicated by dots. Median Nerve—Usual autonomous zone on index and middle finger in solid black. Usual maximal zone indicated by open circles.

(a) After regeneration of a sutured ulnar nerve, stimulation of the ulnar autonomous zone may refer sensation to anywhere within the maximal distribution of that nerve, but never outside the maximal zone (outside area of stippling), never to index finger, thumb or thenar eminence. (b) After regeneration of sutured median nerve, stimulation within its autonomous zone may result in localization anywhere within the region of the open circles, but never outside the maximal distribution of the median nerve.

ning in the same sheath as the degenerating fibers may bud out and grow to innervate a larger area. This same process undoubtedly occurs in pedicle skin grafts, as studied by Davis and Kitlowski\textsuperscript{4} and Kredel and Evans,\textsuperscript{8} where sensation returns as a slow progression of sensory return from the periphery inwards.
To differentiate between the sensory return from overlap and that resulting from regeneration of a severed nerve, the criteria of Pollock\textsuperscript{14,15,16} are generally accepted. In overlap the recovery occurs centripetally from the periphery as a slow wave, and fits in with the process outlined by Weddell, Guttmann, and Gutmann.\textsuperscript{28} Pollock holds that where sensibility to pain returns without return of touch sensation, or where sensibility is lost by blocking adjacent nerves, that return is due to overlap. In contrast, according to Pollock, in true regeneration the sensibility returns in small islands as the regenerating twigs reach the surface and arborize. Pollock states that sensibility never returns in the autonomous zone from overlap.

The phenomenon of faulty localization has long been recognized by numerous investigators,\textsuperscript{3,5,9,10,12,19,20,21,24} but its significance and usefulness has not been fully appreciated. Its mechanism of production as studied by
Cajal\textsuperscript{18} and Weiss and Taylor\textsuperscript{29} is by the misdirection of regenerating fibers across the suture line. No chemotactic influence draws a regenerating fiber to its previous distal tubule; instead, fibers grow in a haphazard manner down whatever tubule they chance to meet. The result is a gross disorganization of the sensory pattern. Spot “A” in the course of regeneration may now be supplied by fibers “B” and “X,” and, when stimulated, sensation is referred to sites “B” and “X” (Fig. 2).

The evidence presented indicates that the phenomenon of faulty localization of cutaneous stimuli is of great practical use in evaluating the presence of sensory regeneration in sutured nerves. It has been observed in a wide variety of sutured nerves—median, ulnar, radial, axillary, musculo-cutaneous, medial antebrachial cutaneous, sural, saphenous, tibial, and peroneal. It is applicable as a criterion of regeneration not only in nerves with an autonomous zone, but also in the intermediate zone of the cutaneous supply. It is a sign that can always be elicited when sensory regeneration of a sutured nerve has occurred. Sutured nerves that show this phenomenon give evidence of regeneration, those not showing it are failures.

Faulty localization is also the most persistent sign of regeneration in a sutured nerve. Years after regeneration is complete, faulty localization persists. Ford and Woodhall\textsuperscript{19} found persistence of faulty localization many years after regeneration of a sutured nerve. Because faultily localized stimuli cannot be fused or suppressed to give a single correct locus, it would indicate that learning and integration of sensory stimuli are possible only to a limited extent, and that a rather well marked anatomical basis for cutaneous localization exists in the cerebral cortex. This is compatible with the finding of Purdy\textsuperscript{17} that faulty reference of sensation in translocated tissue remained for ten years, and the cutaneous localization, in spite of central conflict of visual appreciation of the site stimulated, was still referred to an amputated non-existent area. Marshall, Woolsey and Bard\textsuperscript{11}, studying cortical potentials after cutaneous stimulation in animals, found well localized cortical potentials indicating a precise anatomical cortical representation of sensation.

**SUMMARY**

Four representative case reports have been presented to show the characteristics of faulty localization of sensory stimuli after nerve suture and regeneration. It has been shown that:

1. Faulty localization occurs by the pathway of the sutured nerve and is evidence of regeneration.
2. Where faulty localization fails to occur, sensory regeneration cannot be claimed for the sutured nerve.
3. Faulty localization is confined to the cutaneous distribution of the severed nerve.
4. Faulty localization persists after regeneration, indicating limitations of central fusion of tactile impression, and that a well marked anatomical cortical representation of cutaneous sensibility exists.
REFERENCES