good and bad, obtained by diathermy, nerve sections, electricity, etc., as well as the spontaneous cures. III. The dissociation areas observed prove clearly that in the peripheral system separate and distinct sets of fibers conduct impulses for (a) light touch with possibly a separate set for compasses; (b) prick and (c) for each of the various forms of heat and cold, although Dr. Byrne has only seen one or two instances in which epicritic sensibility for cold was apparently preserved where sensibility for ice was lost. IV. The irregular mode of regeneration with the appearance of island areas of returned sensibility with consequent indentations in the boundary of lost sensibility makes us ask the question: how much of this is due to the procedure employed at operation and how much to the peculiarities, overlapping, of the nerve supply of the region.

Head, after experimental section of the radial nerve at the wrist and both branches of the external cutaneous, in his own arm, found an area of dissociated sensibility similar to areas *A*, *B*, *C* and *B* in our chart, April 15. His area was in the region of the tabatier, and on the dorsal aspect of the wrist. The question arises, was the external cutaneous injured in their case at the time of plating the bone, and if so, were the areas of disassociation existent from the time of operation, and not the result of regeneration. Experiments would seem to indicate that these islands were result of regeneration possibly in areas supplied by a nerve (ext. cutan.) that had been injured but not severed. But with this they had the unusual return of epicritic sensibility for light touch before the return of that for prick and for heat and cold. Head's area would be relevant here, but for the fact that there were found other areas in their case—those of loss for prick and for gross heat and cold. The conclusion is that the irregular form of regeneration was due in part to the form of neuroplasty, and partly to injury without severance of the external cutaneous nerve, and partly to the peculiarity, overlapping, of the nerve supply of the areas affected. Even this guess leaves much to be desired and a fruitful field invites further research into the normal mode of regeneration in nerves.

EXCISION OF BRACHIAL PORTION OF ULNAR NERVE FOR
MULTIPLE NEURO-FIBROMATA, WITH RECIPROCAL
GRAFTING OF THE ULNAR NERVE INTO THE
MEDIAN NERVE, AND OF A PORTION OF THE
MEDIAN INTO THE ULNAR; HYPERALGESIA
OF MEDIAN AREA; MECHANISM; PAR-
OXYSMAL NEURAL PAINS

By R. H. M. Dawbarn, M.D., and Joseph Byrne, M.D.

The patient, a young German, æt. 26, cook, single, had no venereal history, nor trauma. Apparently there spontaneously developed, beginning six years ago, a long swelling over the region of the ulnar nerve, and extending from high in the axilla to a point well below the elbow; involving in fact the entire brachial portion of this nerve. This was accompanied by considerable and steadily increasing tenderness of the diseased area, for which condition relief was asked. The tumor mass was in places as large as the fist, and was translucent. The muscular power of the hand, where supplied by the ulnar nerve, while not wholly lost, was largely so, with obvious wasting of the interossei muscles and of the thenar and hypothenar eminences. Electrical reaction (faradic) was absent or greatly impaired compared with the normal side. The muscles involved left no doubt as to

which nerve was involved in the neuroma. One curious anomaly was observed; namely, that even immediately after the excision was performed the man was able to extend his terminal phalanges fully. One would of course have expected extension of the first and second, and flexion of the last phalanges, in this condition, but Dr. Byrne thought it not unlikely that here the gradual loss of control of the finger ends by the ulnar led to a gradual resumption of more complete control by the common extensors (posterior interosseus nerve). The condition must, Dr. Dawbarn thought, be very rare. In operating on this case, in order to do bloodless work and yet get abundant room in the axilla, Wyeth's pins, back and front, were used with rubber cording above them. The tumor was followed to its ending in normal ulnar nerve tissue. This was in the highest part of the axilla, above, and one inch distal to the internal condyle below. A second and separate incision, the scar of which could now be seen, exposed the median nerve high in the forearm. Next the healthy lower end of the divided nerve was tucked through a slit made beneath the pronator and flexor group of muscles and so brought into easy apposition with a strand split off from the exposed median nerve. Sutures of the finest linen thread were used to unite the sheaths. The proximal end of the ulnar nerve, high in the armpit, was inserted into an opening in the sheath of the median and sutured there. The long incision healed by primary union. It was united by the clip and strip method, using Michel's clips for 24 hours only; the adhesive strips were removed after ten days. The clips did not irritate as when left in for five days. Dr. Dawbarn said that the results of this method were so uniformly ideal that he had ceased practically to suture wound edges.

Dr. Byrne said that the interesting feature of this case was the hyperalgesia which followed the surgical trauma of the median nerve. True hyperalgesia of the peripheral nerves was a rare condition. It was formerly called causalgia and mentioned as such by Weir Mitchell in his classic *Injuries of the Nerves*. In this case there were: first week, sensory symptoms; second week, burning sensation at the roots of nails in thumb and index fingers; third week, whole median area on palm and fingers exhibited hyperalgesia, the boundaries of which were in contrast to median and ulnar areas. The hand was pink-lilac, glossy, tense. This lasted two weeks and then abated. This was due to injury of the median nerve. Dr. Byrne thought his theory of pain in tabes and gastric crises served to explain hyperalgesia and all paroxysmal pains of neural origin. This was oversteering of potential in the related cells of the sensory ganglia. This overloading of potential resulted in the spontaneous discharge of afferent impulses brainward which caused paroxysms of pain referred to the areas of distribution of the related peripheral fibers. The anesthesia and manipulation incidental to the operation in this case discharged the stored potential in the sensory ganglion cells and before it had time to reaccumulate, conductivity had been reestablished in the median nerve.

Dr. Dawbarn presented, as his third case, an account of a thigh amputation, low down, in a middle-aged man, the operation having been made necessary by severe trauma. The surgeon made a common blunder. He did not shorten the sciatic nerve at the time of amputation and the patient could not bear the pressure of the artificial limb. Whenever he attempted to walk he had violent spasms in the thigh stump. After five years he came for relief and Dr. Dawbarn suspected a neuroma. He drew out the terminal five inches of the sciatic nerve. It was very large and vascular. The irritation of the neuroma had led to hypertrophic changes. The lesson to be drawn was that in every amputation there should be a

shortening of several inches of the pain-bearing nerves, for example, in a mid-leg amputation, the anterior and posterior tibial and internal and external saphenous, and musculo-cutaneous, should be shortened. Formerly he had doubted whether it was wise to shorten the nerve, because of the possible danger of atrophy of the trophic nerves of the skin, but he had found this did not occur. The blood vessels had a very rich nerve supply, both sensory and trophic, and this was carried by them to the skin. Thus the trophic supply was not cut off from the skin. This should be emphasized by surgical teachers.

Dr. Byrne said that Weir Mitchell stated that the nerves should always be shortened. After injury nerve degeneration passed inward as well as outward. Several facts lent support to the theory of the storing of potential in the sensory ganglion cells, but the actual proof of the spontaneous passage of impulses inward awaited future workers.

HERPES ZOSTER OTICUS, WITH FACIAL PALSY AND ACOUSTIC SYMPTOMS

By Norman Sharpe, M.D.

The previous history of this patient was negative except for excessive beer drinking. The present illness occurred in the early part of February with an onset of severe pain in right ear, headaches, dizziness, tendency to stagger, and diplopia. After a week of these symptoms he noticed small pimples and facial palsy on the right side and with the palsy and eruption came lessening of the headache and pain and diplopia disappeared. Examination at this time at the N. Y. Eye and Ear Hospital showed loss of taste sense on the right half of tongue, small red spots on the right side of mouth and right pillars of the fauces. Three weeks after onset the pain disappeared and headaches were only occasional and very slight. He came to the Neurological Institute one month after because of the facial palsy. Several small recent scars were found in the concha of the right ear and there was right facial palsy, lateral nystagmus, and the right corneal reflex was diminished. There was slight hypalgesia round the concha of the right ear and almost complete loss of hearing on that side. The urine was normal, the Wassermann negative for blood and cerebrospinal fluid, the globulin was negative and there were 62 cells. Dr. Dench found both tympanic membranes thickened and depressed. Two months after onset taste had partially returned, palsy was still evident, but nystagmus had disappeared. Hypesthesia and hypalgesia had disappeared. The superficial and deep reflexes were normal from the first. The patient was one of the class of cases, described by Hunt, of herpes zoster, attacking the sensory ganglia of the cephalic extremity. He emphasized the fact that in zoster, though one ganglion was primarily involved the adjacent ganglia did not entirely escape. This should be borne in mind in order to understand multiple nerve complications. In placing the lesion in this case the site eruption was in the distribution of the seventh, ninth and tenth nerves. The tenth nerve could be eliminated because of absence of nausea and vomiting and by the fact that there was no eruption of the mastoid and postero-mesial surface of the auricle. Other symptoms pointed to the geniculate of the seventh, as loss of taste and facial palsy. Loss of hearing pointed to involvement of the auditory ganglia or of the eighth nerve. This occurred by extension from the inflamed geniculate ganglion. The involvement was not entire. There was also slight involvement of the glosso-pharyngeal nerve; and diminished corneal reflex pointed to involvement of the Gasserian ganglion. The case