The sequence of motor recovery of 121 patients with hemiplegia was investigated at Boston City Hospital. These patients, with the exception of three, were diagnosed as cerebral vascular accidents caused by thrombosis or embolus of one of the cerebral vessels. At the time the report was written, no autopsy results were available, hence the exact location of the lesion could not be determined. Each patient was examined at regular intervals, and electromyographic records were obtained from some of the patients. The motor recovery of both upper and lower limbs were observed, but attention was focused on the upper limb, in particular on the course of restoration of the grasping function of the hand. Slight sensory defects were found present in 87 of the 121 patients.

An analysis of the data collected in this study indicated that the recovery process followed a general pattern. There was a remarkable uniformity in the sequences of recovery of all patients. This was true regardless of whether sensory disturbances were present and whether the dominant or nondominant hemisphere was involved. The patients progressed from one recovery phase to the next in an orderly fashion without any of the phases being omitted. The same sequence was followed by patients who recovered completely as well as by those patients whose recovery was arrested at any one of the stages.

Immediately following the cerebral vascular accident, the condition was essentially flaccid, with loss or hypoactivity of the tendon reflexes. Thereafter, the following recovery phases were observed:

1. The finger jerks could be elicited.
2. Spasticity—that is, resistance to passive movements—appeared.
3. Proprioceptive facilitation was obtained; reflexes and willed movements were mutually facilitatory.
4. Proximal traction response was present (traction on one group of flexor muscles of the upper limb evoked responses of all flexors of that limb).
5. Some voluntary hand movements were performed without proprioceptive facilitation.
6. Tactile stimuli of the palm of the hand facilitated and/or reinforced the grasp.
7. A true grasp reflex could be elicited (see Seyffarth and Denny-Brown, 1948).
8. Recovery became complete. *

| *Recovery was considered complete when movements could be performed as skillfully and as speedily on the affected side as on the normal side. At this time, blindfolding did not adversely affect movements. |

Twenty-five patients were followed until a comparatively stable condition had been reached. In this group were five patients who recovered fully, even though the upper limb of these patients had been completely paralyzed at the onset. These patients passed through each one of the stages outlined above in a comparatively short period of time. Stage 7 (the grasp reflex) was reached in 23 to 40 days. Thereafter, 20 to 40 additional days were required for full recovery.

From a prognostic standpoint, the time required for arriving at Stages 4 and 5 was considered significant. Those patients who recovered completely reached these stages in 10 days or less; a longer period of time (15 to 46 days) was required for patients whose recovery was incomplete and for those who never progressed beyond the flexor synergy of the upper limb. The patients who failed to respond to proprioceptive facilitation did not recover any willed movements whatsoever. In general, the longer the duration of the flaccid period, the poorer the prognosis.

Spasticity when first observed was mild in all patients. Patients who were on their way to good or full recovery, spasticity reached its peak in 10 to 18 days and never became severe.
The less fortunate patients displayed an increase in intensity of spasticity for a longer period of time, and many developed severe spasticity. Such prolonged spasticity indicated that the prognosis for restoration of motor function was unfavorable.

The flexor synergy of the upper limb was the first movement pattern to recover and was generally followed by an extensor synergy. However, in some patients the recovery of the upper limb was limited to the flexor synergy, and the extensor synergy did not appear. In the lower limb return of flexion also preceded extension, but later, extension predominated.

Flexion of the fingers was first obtained as a part of the total flexor synergy, The fingers could not be flexed in an isolated manner until much later (if recovery continued). During the spastic period of recovery proprioceptive stimuli were most effective as facilitatory agents. As spasticity declined and some voluntary hand movements appeared, tactile stimuli were found instrumental in developing a more complete hand function. The true grasp reflex, evoked by a distally moving tactile stimulus in the palm of the hand, always preceded full recovery. Coordinated hand movements evolved gradually by modification of elementary proprioceptive and contactual responses. When the grasp reflex failed to appear, recovery remained incomplete.

In both upper and lower limbs the synergies of flexion and extension developed before isolated movements of the various joints could be mastered. The author points out that, in general, primitive responses constitute the bases from which more elaborate responses and movements evolve.

The tonic neck reflex (evoked by forceful active head rotation) was found to decrease spasticity in one of the upper limbs, in accordance with the rules formulated by Magnus and de Kleijn. The influence of the body-righting reflexes was also well observable. When the patient was lying on his side with the affected limbs on the upper side, flexion of elbow, wrist, and fingers increased; lying on the other side—with the affected limbs on the lower side—had the opposite effect. In the former position, a proximal traction response was obtained in those patients who exhibited hyperactive proprioceptive reactions. In the latter position flexor tension diminished and some extension appeared.

As long as spasticity prevailed, a certain latency of voluntary motor responses was observed, and relaxation following a contraction was slow. For example, a latent period of 2 to 5 seconds was present between the time a command for a movement was given and the time the movement began; relaxation of contraction required 1 to 3 seconds. When a patient attempted to reverse a movement from flexion to extension this could only be done by allowing a brief pause at the turning point. All willed movements were performed slowly and fatigued easily. At this time, the elimination of vision increased the motor defect.

In summary, motor recovery following hemiplegia began with a simple proprioceptive reaction, the stretch reflex; thereafter, more complex proprioceptive reactions, such as the proximal traction response, evolved; next, the patient learned to utilize the limb synergies—first the flexor synergy, then the extensor synergy. All proprioceptive responses were influenced by neck- and body-righting reflexes. As spasticity declined, willed movements improved and these could be facilitated and modified by tactile stimuli. Tactile stimuli played a continued role in the development of coordinated hand function.

The author concludes:
"The course of recovery from cerebral paralysis does not favour the division of motor function into separate independent entities such as segmental reflexes, neck reflexes, labyrinthine and body-righting reflexes and optic-righting reflexes. Each of the more complex members of these is composed of elements of the less complex. The ability for willed movement is therefore not a separate and indivisible function. The present study indicates the part played by these factors in the course of recovery from hemiplegia, and provides a rationale for proprioceptive and contactual exercises in the retraining of movement."

REFERENCE
Previous investigators, notably Mott and Sherrington (1895), had found that depriving a monkey of all sensation in one upper limb by posterior rhizotomy resulted in an almost complete paralysis of that limb. The limb was not used for walking, running, climbing, or grasping food. However, very little motor deficiency was observed if a portion of the cutaneous sensation in the hand was intact, even when the afferent muscle nerves were severed.

The above-mentioned experiments by Mott and Sherrington were repeated by Twitchell who wished to make his own observations in an attempt to analyze the neuromuscular mechanisms responsible for the return of certain types of motor activity following total or partial deafferentation of one upper limb in monkeys.

RESULT

COMPLETE DEAFFERENTATION

The posterior roots of C3 through T3 were severed, depriving the animal of all sensation in the upper limb. In general, the findings of the previous investigators were substantiated.

Immediately after surgery and for several days thereafter, the limb was functionally paralyzed, although occasionally some uncontrolled associated movements of flexion and extension were observed, particularly when the animal was excited. A few days after the operation the animal began to ward off painful stimuli (such as pin-pricks) with a gross, poorly coordinated flexion movement of wrist and elbow in an attempt to catch the insulting object and pull it toward the mouth. At a later date, an extension movement was also observed as the animal tried to push an annoying object away. Strong motivation was required to evoke these two defensive, motor acts which had to be guided by vision and which could only be used in a limited area on the ventral side of the body. The arm was not used for running, climbing, grooming, or feeding. The ability to use the hand for grasping was permanently lost. The animal was seen chewing on the deafferented limb and precautions had to be taken to prevent him from chewing it off.

It was observed that the above gross movements were related to, and dependent upon, the position of the animal's head with respect to the trunk. The head was markedly ventroflexed during the flexor movement. The posture during the push-away movement was characterized by arching of the back and some dorsiflexion of the head. Head rotation was also seen accompanying flexion or extension movements. The author suspected that the movements observed were neck reflexes which the animal was able to adapt for purposes of defense. After the tonic neck reflexes had been abolished by section of the uppermost cervical roots bilaterally, no movements of the deafferented limb were observed. The limb hung flaccidly and the hand dragged on the ground when the animal moved around. The author's conclusions were thus substantiated.

PRESERVATION OF ONE CUTANEOUS DERMATOME

When one sensory root supplying a portion of the hand was spared, the animal used the limb in a near normal manner for walking, climbing, feeding, and grooming. The grasping function of the hand was quite good, but the grasp was weaker than normal. Some ataxia and overreaching was observed as the animal reached out to grasp an object, but these defects disappeared in about 2 weeks, having apparently been compensated for by vision.

If one sensory root supplying the skin of the upper arm was spared (C5 or T2), all other roots being sectioned, no sensation in the hand remained and much more motor defect resulted. Immediately following operation, the motor behavior of these animals closely resembled that of animals with total deafferentation. The limb was not used for moving around, for grasping, or for defense from pin-prick.
After a considerable time—6 weeks to 2 months or longer—a certain amount of function returned, more rapidly in the C5 animal than in the T2 animal. The limb was used occasionally for walking and running and eventually for grasping. The animal learned to defend itself when teased with a pin by using a gross flexion or extension movement. In both the C5 and the T2 animal movements had to be directed by vision.

In the C5 animal, grasp was observed only in conjunction with flexion of wrist, elbow, and shoulder, that is, as a component of the total flexor synergy. This reaction, the author observes, closely resembles the proximal traction response characteristic of the spastic stage of human patients with hemiplegia (see Twitchell, Abstract 23).

The T2 animal also learned to grasp, but utilized a different neuromuscular mechanism. The proximal traction response was absent. Because T2 also distributes to the finger flexor muscles, the grasp in the T2 animal was believed to have resulted from interaction between the tonic neck reflex and the local stretch reflex, which mechanism the animal was able to adapt to purposeful activity.

This research report contains a wealth of information useful to rehabilitation personnel and deserves to be read in its entirety. In the discussion at the end of the report the following points are stressed:

1. Interruption of the sensory portion of the sensorimotor mechanism results in far greater motor deficit than a lesion in the Rolanic motor area or of the pyramidal tracts (see Denny-Brown, Abstract 3). Without sensation, the limb is practically useless, even though motor areas and motor pathways for the upper limb are intact.
2. Both exteroceptive and proprioceptive impulses are highly important for motor function.
3. The preservation of cutaneous sensation in the hand is indispensable for motor function of the upper limb.
4. Movements of the upper limb, and in particular the grasping function of the hand, is directed by contactual stimuli.

REFERENCE