THE RESTORATION OF MOTOR FUNCTION FOLLOWING HEMIPLEGIA IN MAN

by

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INTRODUCTION

THE disorder of movement so commonly seen in hemiplegia in man has seldom been studied intensively. After the earlier essays of Todd (1856), Bastian (1886), Bergmark (1909), and Walshe (1919, 1923, 1929), it has generally been assumed that the static condition described in every textbook was adequately understood. If variations were recognized, they were in terms of the extreme of spasticity or flaccidity, and their explanation was left to indecisive physiological investigations. Anatomical studies of such cases have been inconclusive (Aring, 1940).

Hemiplegia manifests a large number of variants, both in the different conditions in which it occurs and at different times in the same patient. The present study is chiefly concerned with the course of recovery of movement following cerebral hemiplegia in an attempt to delineate some of the factors concerned. 121 patients have been observed, With the exception of 3, all suffered from a hemiplegia caused by thrombosis or embolism of one of the cerebral blood vessels. In the exceptions the hemiplegia was presumably due to a hæmorrhage infarct in one, in another an angioma (shown by cerebral angiography), and in the third a glioma of the mid-brain and thalamus. All patients who exhibited coma over a long period of time, evidence of intracerebral hæmorrhage with rupture into the ventricles, or a severe aphasia which persisted for over a week, in addition to hemiplegia have been excluded from this study because of the complicating factors involved.

It was possible to follow 25 patients from the time of admission to the hospital to a point where a comparatively stable condition was reached. At the time of admission, 13 of these could not move either limb, 4 could move only the lower limb, and 8 could make weak movements in both extremities. Periodical clinical examination of each patient and electromyographic studies were employed in this series. From clinical evidence

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the lesions were all situated in the cerebral hemisphere, and most were in the internal capsule or subcortical white matter. To date there have been no autopsies on the patients reported here, and therefore no exact anatomical localization of the lesions can be offered. However, this report is chiefly concerned with a remarkable uniformity in the steps of recovery

in different cases. Since in many respects the condition of cerebral hemiplegia is subject to great variation, it has been necessary to analyse the frequency of such variations in some detail, in order to emphasize the operation of the more constant associations. The anatomical correlation of certain variants awaits a more detailed clinical and pathological study. Some slight degree of sensory defect was demonstrable in 87 of the 121 patients studied, but the presence or absence of such mild sensory disturbance was not associated with any constant difference in the course of

recovery. The changes which we shall describe were the same whether the lesion was in the so-called "dominant" or "non-dominant" hemisphere.

GENERAL COURSE OF RECOVERY

Our first object was to establish the mode of recovery of ability to grasp a designated object with the affected hand, and for this reason we were chi concerned with the events in the course of recovery in the upper limb. Early in the study we observed 5 patients in whom there was finally a complete restoration of motor function in an upper extremity which previously had been completely paralysed. All movements in these extremities could be voluntarily executed as rapidly and as dexterously as those in the uninvolved limbs. The restoration of motor function in these cases followed a general pattern in which certain phenomena predominated during distinct phases or stages of the recovery process. It soon became clear that the evolution of recovery in those patients in whom restoration of motor function was not complete also followed the same general pattern, exhibiting all of the reactions of the recovery process up to a point where recovery halted. Each of the patients whom we could only observe for shorter periods of time could be fitted into one or more stages of this general pattern of motor recovery. Data obtained from some of these "short-term" patients will be used to illustrate special features of motor recovery.

Since the description of a series of special aspects of the process of recovery necessarily obscures the general course of recovery, we may here summarize the main course of events. Immediately following the onset of hemiplegia there was total loss of voluntary movement in the involved extremities, and a loss or diminution of the tendon reflexes. Resistance to passive movement was decreased in the involved extremities, but was seldom so complete as to be called flaccidity. Within forty-eight hours the tendon reflexes (excluding the finger-jerks) became more active in the paralysed extremities than in the uninvolved side, but were not clonic. In most of the patients the finger-jerks also became hyperactive within forty-eight hours, but in about a third of the patients this did not occur until three to twenty-nine days after the onset of hemiplegia. Within a short time a minimal degree of increased resistance to passive movement of the extremities began to appear first in the palmar flexors of the wrist and fingers and in the plantar flexors of the ankle. [1]

The resistance gradually increased in intensity and involved other muscle groups, so that the adductors and flexors were chiefly concerned in the upper extremity, and the adductors and extensors in the lower. Concurrently the tendon reflexes became brisker and clonus appeared one to thirty-eight days after the onset of hemiplegia, and one to thirty days after the first evidence of spasticity. Clonus was first seen in the plantar flexors of the ankle and involved these muscles frequently, although not infrequently the knee extensors and the palmar flexors of the wrist and fingers also exhibited clonus. The clasp-knife phenomenon appeared within three to thirty-one days after the onset of hemiplegia, and one to twenty-five days after the onset of spasticity, and involved the knee extensors and elbow flexors.

The first willed movements to return were a slight flexion of the shoulder and flexion of the

hip, which appeared six to thirty-three days after the onset of hemiplegia. The ability to perform willed movement continued to improve so that eventually flexion of the elbow could also be accomplished, but not as a single movement. It accompanied flexion of the shoulder. Later palmar flexion of the wrist and fingers was added to this total flexion pattern in the upper extremity so that at first flexion of the fingers occurred as part of flexion of the upper extremity as a whole, that is, the shoulder, elbow, wrist and fingers simultaneously.

Even before this total voluntary flexion response developed, the patient could enforce a finger flexion obtained by certain proprioceptive stimuli. Thus if the observer delivered a series of finger-jerks or passive stretches of the tendons of the finger flexors, while the patient was simultaneously trying to flex his fingers voluntarily, the fingers flexed slowly but powerfully. Also, at this time the total flexor synergy could be facilitated by passive stretch of the corresponding muscles, At this time spasticity and other signs of exaggerated proprioceptive reactions were at their height, and passive traction upon the flexor muscles of the arm could by itself cause active contraction of all the flexor muscles in the limb, This response, which we propose to call "proximal traction response," strongly facilitated the slow and feeble flexor synergy in t the same muscles when the latter was the sole response to the effort of the patient. Both the total flexor synergy, in the upper extremity in response to willed effort, and the proximal traction responses were facilitated or depressed by the tonic neck and body righting reflexes.

[1] In this paper thc word flexion, with reference to any particular joint, refers to a movement which would withdraw the limb, and extension to a movement which would straighten the limb. Thus, flexion of the shoulder would be a movement which retracts the elbow. Movements of the digits of thc foot and hand, and movements of the wrists and ankle are more difficult to classify in this way and for them we have retained the words plantar and palmar flexion and dorsiflexion,

An extensor synergy of the arm, consisting of extension of the shoulder, elbow and wrist, and dorsiflexion of the fingers appeared soon after the earliest development of the flexor synergy. As in the case of the flexor synergy, the extensor synergy appeared first as a movement of the shoulder, with movements of the elbow, wrist, and fingers added later.

As the power of willed. flexion of shoulders and elbow further increased, a stage was reached where spasticity in these muscle groups abruptly lessened. At the same time power of willed flexion of the fingers increased. Finger flexion could now be facilitated by a contactual stimulus moving distally across the palm of the hand. Movement of single digits became possible. Later a stationary contactual stimulus to the palm of the hand could also facilitate willed flexion of the fingers. As finger movements became more powerful and dexterous, spasticity abruptly lessened in the finger and wrist flexors and a well-defined grasp reflex could be obtained.

As mentioned previously, several patients recovered completely so that eventually they could execute any movement with the previously paralysed limb with the same degree of rapidity and dexterity as in the uninvolved extremity. Spasticity completely disappeared when complete recovery of the power of willed effort occurred. The tendon reflexes, however, remained slightly more active in the previously paretic limbs, but not clonic. An increased liability to fatigue and

perhaps a very slight weakness in the previously paralysed limbs then remained as the only impairment of function.

The restoration of voluntary movement in the lower limb also began with the appearance of flexor and extensor synergies. As in the upper limb the flexor synergy appeared first, beginning as a flexion of the hip one to thirty-one days after the onset of hemiplegia. Flexion of the knee and dorsiflexion of the ankle were soon added to the hip flexion to make up the total flexor synergy. The appearance of an extensor synergy followed closely the development of the flexor synergy; it began with extension of the hip with extension of the knee and plantar flexion of the ankle occurring sometime later.

We have arbitrarily chosen to designate the recovery as complete when all movements of the limb could be performed with the rapidity and dexterity of similar movements of a limb which had not been paralysed. It must be emphasized that though the recovery process could be divided into "phases" or "stages" each of which could be maintained for a variable period, the recovery process was continuous and the gradual appearance of new factors resulted in overlapping of the different stages. Thus, the period of flaccidity immediately following the onset of hemiplegia was not abruptly followed by a period of exaggerated proprioceptive responses, but the tendon reflexes became more active while resistance to passive movement was not increased. Likewise, while proprioceptive reactions were still exaggerated, certain contactual effects appeared, and spasticity in various degrees could complicate the first voluntary movements.

RESTING POSTURE

The resting posture of the paralysed limbs depended on the intensity of spasticity, and the influence of the tonic neck and body righting reflexes.

Of the 25 patients observed over a long period of time, all but 2 exhibited an abnormal resting posture at some time. Immediately following the onset of hemiplegia when the limbs were "flaccid" the posture depended upon passive placement of. the limbs or upon gravity, but with the onset of spasticity, more constant postural abnormalities appeared. However, resistance to passive movement had to reach a certain intensity before abnormalities of posture resulted. In the 2 patients in whom no postural abnormalities were present, a very slight increase in resistance to passive movement could be demonstrated in thc wrist and finger flexors, though this was never intense enough to produce an increased flexion of the fingers at rest. When postural abnormalities occurred, the first evidence of them was seen in the hand or foot, but without regular precedence in upper or lower limb. They were increased flexion of the fingers, and plantar flexion with slight inversion of the ankle.

9 patients showed increased flexion of the fingers as the only abnormality of resting posture during a long period until a stable condition had been reached or recovery had occurred. This abnormality occurred within forty-eight hours to ten days after the onset of hemiplegia. 4 patients showed increased flexion of the fingers together with plantar flexion and inversion of the ankle as the only postural abnormality during the entire period of observation. 2 patients developed a spastic posture of one limb. In 1 patient flexion of the fingers and plantar flexion of the ankle

occurred within forty-eight hours of the onset of hemiplegia. Three days later a slight flexion of the wrist was detected. Without further change in the arm, spasticity in the hip adductors and extensors and knee extensors increased so that by the twenty-first day after the onset of hemiplegia the leg assumed a rigid, extensor posture. In the other patient no abnormality of resting posture was observed until twenty days after the onset of hemiplegia (this was also the first time any evidence of spasticity was detected) when the fingers were flexed and the ankle plantar flexed. On the thirty-first day after the onset of hemiplegia the wrist was flexed and by the fifty-second day the arm was slightly flexed at the elbow. No other postural abnormality was observed in this patient during a nine-month period when all neurological signs had stabilized.

8 patients developed the classical hemiplegic posture in both limbs. Flexion of the fingers and plantar flexion of the ankle were followed by increased flexion of the wrist and elbow, and extension of the hip and knee. Later the shoulder was found to be adducted and internally rotated, the arm flexed at the elbow, the wrist pronated (in 5 patients) or supinated (3 patients) and slightly flexed, and the fingers flexed. The leg was extended and adducted at the hip, extended at the knee, and plantar flexed at the ankle with slight inversion of the foot. No patient was observed with a flexion posture of the lower extremity or extensor posture of the upper extremity.

The time required to develop full hemiplegic posture varied consider ably in the 8 cases. 4 patients showed the first postural abnormality within forty-eight hours following the onset of hemiplegia, while the others showed no abnormalities until twelve to twenty-four. days following the onset of hemiplegia. 3 of them developed a total hemiplegic posture in five days; 2 patients did so in about four weeks, while the remaining 3 required from two to six months. In general, those patients who showed the first postural changes rather late after the onset of hemiplegia took the longest time to develop a complete hemiplegic posture.

In 97 patients whom we observed over shorter periods of time, postural abnormalities were not seen in all. Six exhibited a typical hemiplegic posture with flexed arm and extended leg as described above, 7 exhibited a flexed arm without any postural abnormality in the leg, 2 exhibited extension of the leg without any abnormality in the arm, and 22 presented minor degrees of hemiplegic posture. The other 60 patients showed no abnormality of posture, but at the time of their last examination, most of these were just beginning to develop mild spasticity, and it is probable that some postural abnormalities would have been detected were it possible to follow these patients for a longer time.

The classical hemiplegic posture of flexion in the upper limb and extension in the lower was therefore uncommon in fully developed form. Flexion of the fingers and wrist and plantar flexion of the ankle are the most commonly found spastic postures. As the response to effort became more precise, spasticity lessened, and the associated postural abnormalities eventually disappeared. The most persistent postural abnormality in the upper extremity was then an increased flexion of the wrist and fingers. This abnormality disappeared in forty-three to sixty days following the onset of hemiplegia. Abnormalities of resting posture persisted in the patients whose recovery was not complete.

EXAGGERATION OF PROPRIOCEPTIVE RESPONSES

Tendon reflexes.—ln 25 patients observed over a long period of time, the tendon reflexes (excepting the finger-jerks) became more active in the paretic limbs within forty-eight hours following the onset of hemiplegia. In 5 of these the biceps and the knee jerks were less depressed than the other tendon reflexes, and became hyperactive sooner. In the other 20, however, all of the tendon reflexes were equally depressed and became hyperactive at the same time. In 18 of these patients the tendon reflexes became, hyperactive concurrently with the earliest appearance of an increased resistance to passive stretch of the muscles, while in the other 7 patients hyperactivity of the tendon reflexes preceded the earliest appearance of an increased resistance by three to twenty days. The tendon reflexes were not markedly exaggerated at this time, for any increased activity was minimal at first, and only by carefully comparing each tendon reflex with its counterpart in the uninvolved limb could any difference be detected. Within four or five days the tendon reflexes (excluding the finger-jerks) were quite obviously more active in the hemiplegic limbs, each reflex being a single contraction of both greater speed and amplitude than normal. A double beat or true clonic jerk was not present at this time.

The finger-jerks, on the other hand, did not always become hyperactive .as soon following the onset of hemiplegia compared with the finger-jerk on the non-paretic side (which were absent in 19 of 25 patients). Hyper-active finger-jerks could be elicited within forty-eight hours following the onset of hemiplegia in 14 of the 25 patients. In the remaining patients a period of three to twenty-nine days elapsed before, hyperactivity of the finger-jerks appeared, being preceded by some evidence of increased resistance to passive movement in 8. In the remaining cases, hyper-activity of finger-jerks appeared concurrently with the first evidence of increased resistance to passive movement. At this stage the finger-jerks were a quick, single jerk, and were never clonic.

A typical Hoffmann response, elicited in the usual manner by "flicking" the nail of the middle finger, was obtained in 16 of the 25 long-term patients. In 10 of these patients it could elicited on the same day that the finger-jerks were found to be hyperactive. In the others a period of one to twenty-four days intervened between the earliest appearance of hyperactive finger-jerks and the Hoffmann response. The finger-jerks were not necessarily clonic when the Hoffmann response appeared.

Resistance to passive movement.—The resistance of the muscles to passive movement varied both qualitatively and quantitatively from individual to individual. It also varied during different phases of the recovery process and under the influence of the tonic neck reflex, body righting reflex, and the proximal traction response.

Following the period of "flaccidity" some increased resistance to passive movement was usually detected within forty-eight hours after the onset of the hemiplegia. However, in 6 of the 25 patients observed over a long period the first evidence of increased resistance presented on the third, fifth, ninth, seventeenth, eighteenth, and twentieth days respectively, and all eventually showed some increased resistance to passive movement.

Increased resistance to passive stretch did not appear abruptly. Its onset could not be

demonstrated by shaking the limb passively, but only by inspection and palpation while the muscle was being stretched, and by comparing its resistance with that of its fellow of the opposite, normal, limb. In this way the initial appearance of resistance to stretch could be detected before the usually accepted signs of spasticity had appeared.

At its earliest onset, any increased resistance could be demonstrated only by stretch of the muscle through its full range. Thus, for example, it was necessary to extend the wrist beyond 180 degrees in order to demonstrate any increased resistance in the wrist flexors, and the resistance when encountered was very slight, having a soft and yielding character.

The increased resistance usually developed first in the flexors of the wrist and fingers and the plan r flexors of the ankle (15 of the 25 patients), but in I patient it first appeared in the flexors of the elbow. Gradually, after a further one to seventeen days all the flexors of the upper limb and extensors and adductors of the lower limb offered similar resistance. In the remaining 9 patients the onset was so rapid that when its appearance was first noted, it was present in all these muscle groups. 4 patients followed for a long period eventually exhibited an increased resistance to passive movement in the adductors and retractors of the shoulder. the flexors, and (with less intensity) the extensors of the elbow, the pronators and flexors of the wrist, and the flexors of the fingers. the lower extremity an increased resistance was present in the extensors and adductors of the hips, the extensors of the knee, the plantar flexors of the ankle, and (with less intensity) in the flexors of the knee and hip. 3 patients exhibited an increase in resistance as described above except that the wrist supinators showed an increased resistance to passive move ment rather than the pronators. More commonly the muscle groups in each extremity exhibited the pattern described above, except for absence of resistance in the shoulder and hip musculature. In many of these, .and some others, the extensors of elbow and flexors of knee were not involved.

In spite of these variations in distribution of increased resistance, it was evident that the elbow flexors were involved three times as frequently as the shoulder muscles, and the wrist and finger flexors four times as frequently. In the leg, the knee extensors and ankle plantar flexors were involved twice as frequently as the hip groups.

In the larger group of patients who were observed for shorter periods, this distribution of spasticity was also observed. In addition 7 patients exhibited some increased resistance in the shoulder extensors, and 7 showed an increase in resistance in the shoulder abductors. The elbow extensors, as well as the flexors, showed increased resistance in 23. Increased resistance in the wrist and finger dorsiflexors was seen in 7 and 4 patients respectively and in the supinators in 2. Increased resistance was present in the flexors of the hip in 4, the knee flexors in 20, and in the ankle dorsiflexors in 3.

The intensity of resistance to passive movement was in general greatest in those muscles in which it first appeared. Of the whole group of patients examined, 6 cases showed a variation in this distribution of intensity. In 4 resistance to passive movement was more pronounced in the proximal groups of the upper limb, and in 2 it Was more pronounced in the proximal muscles of the lower limb. In 4 of these 6 patients this distribution of intensity persisted, while in the other two this distribution changed so that within five to ten days the resistance was greater in the distal

parts than in the proximal.

At its very onset the resistance to passive movement was difficult to demonstrate, and at this stage may be called slight. Yet within five to ten days, the resistance to passive movement was very obvious and easy to demonstrate, and sufficient to affect the resting posture of the joint. One might speak of such degree of resistance as being "moderate" in intensity. More usually a slight degree of resistance progressively increased to reach an intermediate "mild" degree within one to ten days. In 8 cases the resistance to passive movement could actually be considered "intense," and the early development of contractures was noted in this group. Occasionally the intensity of resistance could vary from mild to moderate and back again in the same muscle groups in day-to-day examination. This type of variation was rare, however. The changes in the intensity of resistance under the influence of the tonic neck reflex, body-righting reflex, proximal traction response, and during attempted voluntary movement were in contrast relatively constant.

Both the flexors and extensors acting on a joint presented an increased resistance to passive movement in a number of cases. In most of these the intensity of resistance was greater in the flexors of the arm, and extensors of the leg. In 5 cases the resistance in agonists and antagonists was seemingly equal, though the hemiplegic posture persisted. In 2 patients when lying supine an apparently equal degree of resistance was encountered in the flexors and extensors of the knee, and by the operation of the lengthening and shortening reactions the leg might be flexed or extended depending on the position in which it had previously been placed. In no case was the flexor resistance so great that the leg remained continually in an attitude of flexion, or the arm in extension.

The quality of increased resistance to passive stretch also varied. In the great majority of cases it was of a type having the generally recognized qualities of spasticity. While the muscle under examination was passively stretched, after a range of stretch during which no appreciable resistance was encountered, there occurred a progressive though slight increase in resistance. This resistance appeared more suddenly and was more pronounced, if passive stretch was carried out rapidly. When passive stretch was carried out resistance gradually increased in intensity, finally reaching a maximum, and then melted away rapidly before the end of the range of passive stretch. With the passage of time and further heightening of the stretch reflex, the range of passive movement before resistance was encountered became less, and the intensity of t resistance became greater.

10 of the long-term patients developed a degree of abruptness of appearance and of melting of resistance, such as to be classified as a true clasp-knife phenomenon. 5 patients presented a clasp-knife phenomenon only in the knee extensors. The other 5 patients showed a clasp-knife phenomenon in both the knee extensors and the elbow flexors. The clasp-knife phenomenon appeared in three to thirty-one days after the earliest appearance of spasticity. In general, it appeared one to eight days after the onset of spasticity in the muscles concerned. No correlation was found between the intensity or quality of spasticity and the development of the clasp-knife phenomenon or clonus. In about half of the patients showing both of these phenomena, the resistance to passive movement was quite mild, while in the other half the resistance was moderate

to intense.

In 3 patients, instead of the usual type of resistance which rose to a maximum toward the end of passive stretch and then melted quickly, there was a resistance which rose to a maximum somewhat sooner before the end of passive stretch and faded away thew-more gradually. In 1 of these patients this type of resistance was present in the flexors of the elbow, wrist and fingers while the muscles of the leg presented the more usual type of resistance described previously. In the other 2 this variation was encountered only in the elbow flexors. In none of these patients was there any change in this quality of resistance during the period of observation.

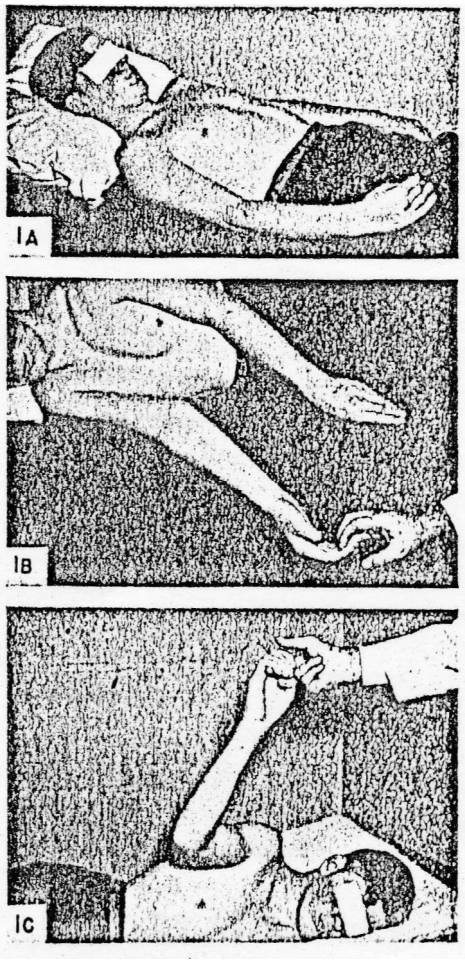


Fig. 1--Photograph of patient R. B. with right hemiplegia, A, position of right arm with patient in supine position, B, attempt at elicitation of proximal traction response with patient lying on his right side. Flexion of fingers is no more than that occurring with the patient in the supine position, C, elicitation of the proximal traction response with thc patient lying on his left side.

In 2 patients another variation was observed. Here as stretch was continued after the onset of resistance, the resistance remained of the same intensity throughout the range of passive stretch and then melted rapidly at the end of passive movement. This was observed only in the elbow flexors while the resistance in other muscles was the more usual type.

The resistance of the muscle did not always melt completely if stretch continued, In three

cases a "plastic" type of resistance resembling the rigidity of extrapyramidal disease was encountered. Here increased resistance appeared after the beginning of passive stretch, allowing freedom for only about 10 degrees of the range of passive movement. The resistance then continued with about the same intensity throughout the further range of passive movement and without fading away at the end. This form of resistance was most apparent at the elbow where both the flexors and the extensors presented this same quality of resistance.

14 of the long-term patients eventually developed clonic tendon reflexes. 5 of these patients showed only clonic ankle-jerks, 4 had clonic knee and ankle-jerks, 3 had clonic finger and ankle-jerks, 2 had clonic finger, wrist, knee and ankle-jerks, and I had only a clonic knee-jerk. Clonic reflexes were not obtained elsewhere in these patients or in the other patients, although occasionally a double beat might be elicited in the biceps or triceps jerk.

In 5 patients clonic ankle-jerks were obtained forty-eight hours follow, ing the onset of hemiplegia. It is to be noted that clonic ankle-jerks were not obtained on the non-paretic side. In other patients clonic reflexes were obtained one to thirty-eight days after the onset of hemiplegia, and one to thirty days after the earliest appearance of spasticity. In general, clonic tendon reflexes reached their maximum seven to twenty days after the earliest appearance of spasticity in the muscles concerned in those tendon reflexes. In those patients who developed sustained clonus, it could be elicited within a day or two after the appearance of clonic tendon reflexes. A short time before a sustained clonus could be elicited, a clonic tremor could be felt superimposed upon the steadily increasing resistance to passive movement thus resembling the "cog-wheeling" of Parkinsonism. This phenomenon could not always be elicited, and rapid passive stretching of the muscle was most effective in eliciting it,

In 5 patients in whom voluntary movement was ultimately restored completely in the upper extremity, the intensity of spasticity increased parallel with the developing exaggeration of thc proprioceptive reactions and decreased with the restoration of the power of willed movement. Spasticity was first noticed in these patients one to nine days after the onset of hemiplegia. In 1 of these patients, spasticity was found only in the wrist and finger reflexors, while in the other 4 it occurred in the elbow, wrist and finger flexors, and wrist pronators. In these patients, spasticity was never intense, being only moderate at its most pronounced stage. Spasticity reached its maximum intensity in from eight to twenty-one days. One of these patients developed clonic finger-jerks. The other did not develop clonic tendon reflexes, although all of the reflexes were markedly hyperactive. 2 patients developed a clasp-knife phenomenon in the elbow flexors. As power of voluntary movement increased, spasticity abruptly lessened, first in the shoulder and elbow flexors and later in the wrist and finger flexors. With complete recovery of motor ability no evidence of spasticity could be detected.

RESTITUTION OF MOVEMENT IN RESPONSE TO WILLED MOVEMENT

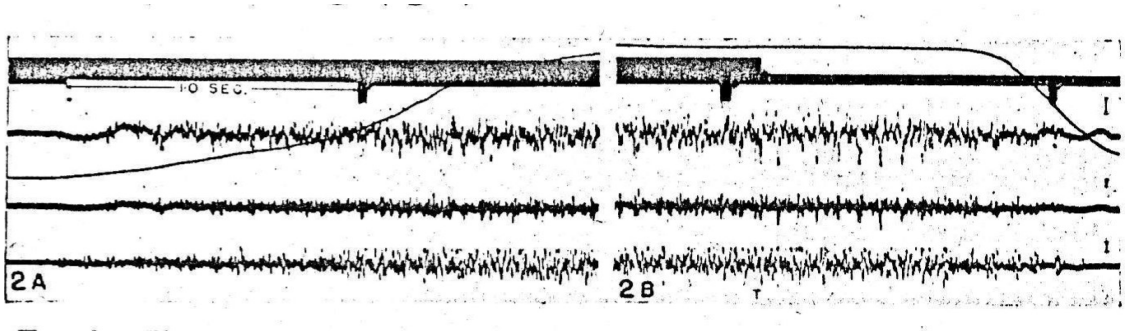
Recovery ,from hemiplegia has been stated by many to occur first and be most complete in the proximal muscles of each limb, while willed movements of the hand and foot return last, are weaker, and are much more severely affected as far as dexterity of movement is concerned.

In our series of 25 patients followed throughout their course 13 could move neither arm nor leg, and 4 were completely paralysed only in the arm, at the time of admission to hospital. Of the 13 patients, 8 recovered some movement in both the arm and leg, 1 recovered movement in the arm only, I recovered movement-in the leg only, and 3 did not recover any willed movement whatsoever. Of the 4 patients with only a paralysis of the arm, 3 recovered some movement of the arm, and I did not. The other 8 patients retained some power of willed movement from the onset, and their course could be fitted into the recovery process at a later stage. 2 of these patients could not perform any willed movement of the hand, however. All of our patients followed the general pattern of recovery, except that in 3 patients recovery followed the reverse order in the upper limb, for the ability to perform willed movement returned first in the hand, and later in the shoulder. The recovery of movement in the leg, however, followed the usual pattern in all our patients.

The first evidence of ability to perform willed movement in the upper limb was observed within six to thirty-three days following the onset of hemiplegia. This movement was a flexion of the upper arm at the shoulder, with exceedingly slow onset, limited range and power. One to six days later an associated flexion of the elbow occurred. This flexion of the elbow could not be performed separately. At this time the range of movement was extremely limited, and the power was only sufficient to overcome the force of gravity.

The ability to flex the fingers and wrist in response to willed effort occurred one to thirteen days after the development of the shoulder-elbow synergy, and six to forty-six days following the onset of hemiplegia. The ability to flex the fingers and wrist in response to willed effort, could only be performed as an addition to the shoulder-elbow flexion synergy. At the shoulder a slight adduction and internal rotation of the upper arm were now associated with the flexion. Therefore, the first stage in the evolution of ability to flex the fingers by willed effort resulted only in this flexor synergy of the arm, consisting of a flexion of shoulder (associated with a slight adduction and internal rotation) and flexion of the elbow, wrist, and fingers. At this stage the patient was totally incapable of performing any individual part of this movement separately, and any attempt to do so merely resulted in the execution of the total flexor synergy.

While these first movement patterns were appearing, a latent period of two to five seconds existed between the time the command to execute a movement was given and the time the actual movement took place. As the movement began it was very slow, but the speed increased toward the end of its limited range (fig. 2). Likewise, after the movement had been executed and the patient was told to relax, relaxation did not take place immediately, but required one to three seconds to do so. It was also noted that immediately after the patient was told to cease voluntary contraction, the resistance in the contracting muscles was more intense than that present before voluntary contraction, and the tendon reflexes were more active than before voluntary contraction. This increase in the resistance and activity of the tendon reflexes also lasted for one to three seconds.



FIG, 2.—Electromyographic tracing from patient M. C, to show characteristics of the earl" return of the response to willed effort alone. Notches in the upper line of the tracing indicate time intervals of one second as shown. The line between the upper and middle galvanometer tracing is a balloon myograph record of muscular contraction of the flexors of the fingers. A command to flex the fingers was given one and a half seconds before fig. 2A and a time of one second separates fig. 2A and fig. 2B. At T in 2B the command to cease flexion was given. The two upper electromyograms are from m. flexor profundus digitorum. The lower electromyogram is from m. flexor sublimis digitorum, Calibrations to the right of 2B are for 100 microvolts.

4 patients still exhibited the ability to produce this flexor synergy as the only recovery of willed movement in the upper extremity after periods of observation up to one and a half years. In these patients isolated movements at any joint could not be effected, nor did an extensor synergy of the arm ever develop. In the other patients further recovery occurred, As the flexor synergy of the arm appeared, an extensor synergy also developed. The first movement to return at any joint was always a flexor movement, yet extensor movements could occur in the upper extremity before the ability to perform the total flexor synergy. Extensor movements could be made in the shoulder and elbow by the time the flexor synergy included the wrist and fingers. In 2 patients after willed flexion of the shoulder could be effected and before flexion of the elbow had returned, a weak extension of the upper arm at the shoulder could be made. Later extension of the elbow was added to this shoulder extension so that these two movements occurred together, Later, the extensor synergy consisted of extension with slight adduction of the shoulder, extension of the elbow and palmar flexion of the fingers. Dorsiflexion of the fingers began to occur some time later. Development of the total extensor synergy of the arm followed closely (within forty-eight hours) the development of the total flexor synergy.

In the leg, the return of voluntary movement followed the same general pattern seen in the arm. Movement returned first at the hip to be followed later by movement of the more distal parts, and flexor movements returned before movement of extension.

In 9 patients in whom the leg was completely paralysed at the onset of hemiplegia, and who recovered voluntary movement, willed flexion of the hip returned in one to thirty-one days. Willed flexion of the knee occurred within a day or two after the hip movement, except in I patient in whom willed flexion at the knee did not occur until twenty days had elapsed. At this time flexion of the knee could not be executed alone, and attempt to do so merely resulted in a combined

flexion at the hip and knee as a flexor synergy of the lower limb, With the return of voluntary flexion of the knee, voluntary adduction of the hip could also be accomplished.

A complete flexor synergy of the leg became possible in these patients twenty-five days to three months from the onset of hemiplegia. It consisted of a flexion and adduction of the hip, flexion of the knee, and dorsiflexion of the ankle and toes. As this flexor synergy was appearing in the leg, an extensor synergy was also forming, Thus at a time when flexion of the hip and knee could be effected by willed effort, extension at the hip became possible. As in the arm, formation of the extensor synergy lagged behind the formation of the flexor synergy. The extensor synergy in its complete form consisted of an extension and adduction of the hip, extension of the knee, and plantar flexion of the ankle and toes, And, as in the arm, these synergies in the leg had a moderately long latent period of from two to five seconds from the time the command to move had been given and the time actual willed movement occurred. The individual movements comprising the synergy could not performed separately.

As recovery progressed the latent period before voluntary movement shortened, the range of movement increased, and the power of movement became greater. In the arm pronation and supination were added to the flexor and extensor synergies. In all but 2 patients pronation of the hand accompanied both the flexor and extensor synergy. In these 2 patients. supination accompanied the flexor synergy, and pronation accompanied the extensor synergy.

The ability to flex either the wrist, fingers or elbow alone, without evoking the total movement synergy, returned gradually as power of willed movement increased. When the patient was first able to perform some individual movement (e.g. flexion of all the fingers, or flexion of the elbow) the remainder of the synergy was still difficult to suppress. Thus if the patient was asked to flex the elbow, and could actually perform this movement, some slight contraction of the flexor groups of the shoulder occurred also. Likewise, when the ability to flex all the fingers without flexing proximal joints first returned, this flexion was often associated with some contraction of the wrist and elbow flexors. This same difficulty also persisted in the lower limb. Spasticity could be quite intense even at the time the ability to perform these isolated movements returned, but as power of movement at the shoulder and elbow became comparable to that of the uninvolved shoulder and elbow, spasticity suddenly lessened, although it did not then disappear completely. In patients in whom a considerable degree of recovery took place, with increasing power of movement, spasticity abruptly lessened in the distal segments also, and at this time isolated movements of these segments could be performed without evoking even a fragmentary movement synergy.

3 patients exhibited a restoration of movement in the arm in a manner opposite to that just described. That is, willed movement returned first in the hand while movement of the elbow and shoulder returned later. The first response to willed effort in these patients was a weak flexion of all the fingers. This movement was followed in about five days by an associated flexion of the wrist and elbow which accompanied the finger flexion. Thus a flexor synergy formed in reverse order. At this time the power of contraction of the wrist and elbow flexors was less than that of the finger flexors. And at the time the wrist and elbow flexion occurred, some willed extension of the fingers could also be affected. After three to six more days flexion of the shoulder was added,

completing the formation of the flexor synergy of the arm. The extensor synergy of the arm also developed, but lagged slightly behind the formation of this flexor synergy. The power of muscular contraction also developed in reverse order in these cases, being greater in the hand and wrist than in the shoulder. The ability to perform movements in an isolated fashion without evoking the total movement synergy also occurred first in the fingers in these cases. In I of these patients before any movement of the shoulder could be obtained by willed effort, the spasticity of the finger, wrist and elbow flexors abruptly lessened with an increasing power of movement. In this patient isolated movements such as flexion and extension of the fingers and opposition of the thumb to each finger could be performed even before any willed movement at the shoulder was possible. The ability to perform shoulder movements which finally did return was not accompanied by any movement synergy in this patient. Some peculiarity of the lesion in these 3 patients evidently greatly shortened the process of recovery in the distal parts.

The movement defect in the arm in the presence of mild or moderate spasticity was studied in the 5 patients in whom recovery was complete and in 8 patients who were not completely paralysed at the time of admission to the hospital. We employed several simple tests to determine the dexterity of movement. These were alternating flexion and extension of the elbow, alternating pronation and supination of the wrist, finger-nose test, ,alternating flexion and extension of the wrist, flexion and extension of the fingers, and the ability to oppose the thumb to each finger from the index finger to the little finger and back again. All of these tests were carried out as rapidly as possible. The long latency which existed at the time of the flexor and extensor synergies were appearing thereafter steadily decreased. Some obvious latency might still remain, however, even when isolated movements could be performed. Thus, when attempting alternating flexion and extension of the elbow, the patient might show a latency (certainly not over one second) between each flexion and extension movement. As recovery progressed this latency rapidly lessened. Weakness was a major defect in the presence of spasticity, and was associated with rapid fatigue. Extension lagged behind flexion in the recovery process in all these respects, and this was especially apparent in the fingers.

In spite of a considerable return of power, the deficit in movement in the presence of spasticity was still striking. The more simple movements such as alternating flexion and extension, or pronation and supination, were performed more slowly than normal and appeared stiff and awkward. Occasionally the smooth flow from flexion to extension would cease at one point, the limb remaining flexed or extended for more than one second before executing the reverse component, On finger-nose testing or even simple alternating flexion and extension at the elbow, a clonic tremor at the elbows developed in 2 patients.

The disorder of willed movement in the presence of spasticity was most clearly demonstrated in the fingers. When the ability to flex and extend the fingers without evoking the total movement synergy of the arm occurred, isolated movements of each finger still could not be performed. Thus, if the patient was instructed to move the index finger, or the little finger alone, any attempt to do so caused either flexion or extension of all the fingers.

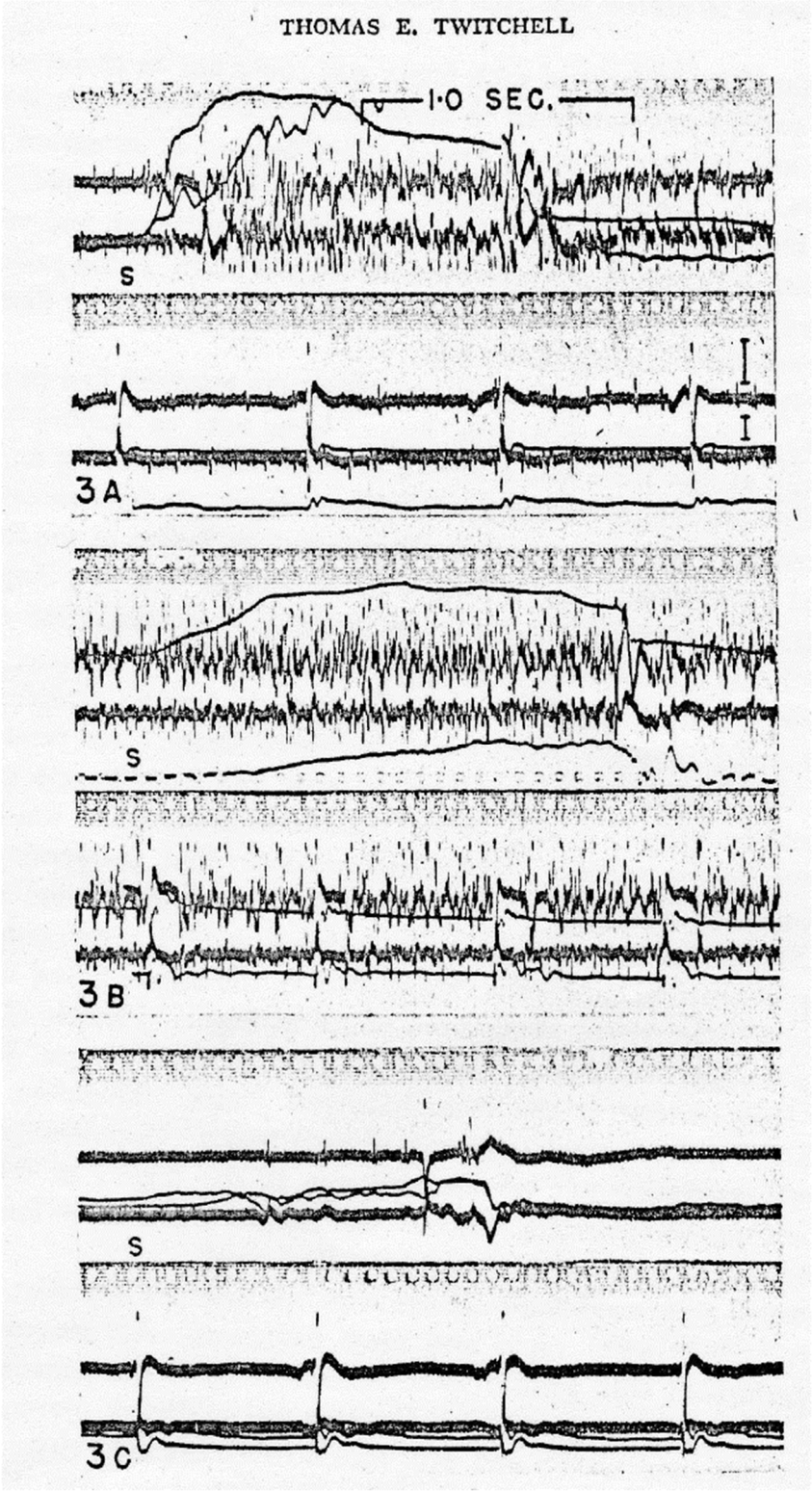


FIG. 3.—Electromyographic tracing from G. A. with right hemiplegia. The galvanometer tracings are accompanied by two black lines, the upper being a recording of the pressure exerted by the stimulus, and the lower being a myographic tracing for the flexors of the fingers. The upper EMG is from m. flexor digitorum profundus, and the lower from m. flexor digitorum sublimis. Upper record for each series is of response to passive stretch, lower record of tendon jerks over a stretch reflex background. Stimulus at S. Calibration at 100 microvolts.

FIG. 3A.—Patient in supine position. FIG.3B- on left side. FIG. 3C.—Patient on right side.

At this stage the fingers could not be closed completely, •most of the movements occurred in the joints of the phalanges, and very little movement occurred at the metacarpophalangeal joint. If the patient was instructed to make a fist, the thumb did not close around the flexed fingers,' but remained slightly extended.

The ability to flex or extend one finger separately returned first in the index finger. The middle and little fingers were next to acquire this ability, though at first there was a flexion or extension of the ring finger, associated with a flexion or extension of the little finger. In later recovery this' associated movement disappeared. In one patient flexion of the little finger caused an associated slight flexion of the middle and ring fingers and a rapid hyperextension of the index finger. This association also vanished with the full recovery of dexterity in this patient.

When opposition of the thumb and digits could first be performed, the thumb could only be exposed to the index finger. As recovery progressed, the thumb could be opposed to more and more fingers until finally it could be opposed to any. When opposition to all fingers was first possible, several movement defects were still apparent. First, in opposing to the fingers, the thumb did not flex in the natural manner, but remained partially extended. In early opposition of the thumb to each finger, most of the activity was carried out by the thumb, that is, the fingers flexed little, and the thumb was moved along from finger to finger. Flexion of the fingers to meet the thumb was mostly at the metacarpophalangeal joint, with very little flexion at the interphalangeal joints. Therefore, the thumb was not truly opposed to the tip of each finger. Occasionally the finger-thumb opposition test revealed tremors of the fingers or thumb while the test was being carried out, and with fatigue "contraction fasciculations" were not uncommon in the muscles concerned.

As recovery progressed these defects were less and less apparent, and movements became more rapid and more dexterous. All movements could be performed with greater speed and dexterity if the patient was allowed to watch his 'hand or limb. If vision was excluded, movements were carried out more slowly and with considerably less dexterity. Any wavering ataxia or tremor was increased by excluding vision even though no defect in sensation could be found by clinical testing. Such defects in movement still occurred in the presence of only slight or mild spasticity. Spasticity finally disappeared with complete recovery of speed or dexterity of movement. Not only were the tests mentioned above performed as quickly and as dexterously as with the normal limbs, but there was also an absence of difficulty in performing such tasks as writing, sewing, buttoning or unbottoning a shirt, or tying shoe-laces, with the eyes open or closed. The only defects then remaining were a very slightly diminished power and an increased liability to fatigue.

PROPRIOCEPTIVE FACILITATION

During the period when proprioceptive responses were increasingly exaggerated, and before any muscular contraction could be made in response to willed effort, certain proprioceptive reactions and the willed effort to produce a movement could mutually facilitate each other. These effects could be most easily and effectively demonstrated for flexion of the fingers.

The mutual facilitation of proprioceptive reactions and willed effort could be demonstrated in

the earliest phase of recovery of stretch and tendon reflexes. Indeed a willed effort could sometimes enable one to elicit a latent tendon reflex or other pathological sign. For example, in one patient the biceps, triceps and supinator reflexes had all become hyperactive within forty-eight hours following the onset of hemiplegia. Finger-jerks could not be elicited until the tenth day following the onset of hemiplegia. The arm was completely paralysed. However, on the eighth day, if the patient was instructed to attempt flexion. of his fingers while the examiner simultaneously attempted to elicit finger-jerks, small, brisk finger-jerks were obtained. (Finger-jerks were elicited by resting the patient's fingers, palmar Surface down, on the examiner's middle finger and tapping the examiner's finger with the reflex hammer.) If an attempt was made to elicit finger-jerks without the patient's willed effort to flex the fingers, no response occurred. Likewise, if the patient attempted to flex his fingers while the examiner was attempting to elicit a Hoffman sign (in the customary manner by flicking the finger nail of the patient's middle finger), a Hoffman response with flexion of all the fingers and thumb was obtained. Without the simultaneous willed effort of the patient, no responses could be obtained.

Within a day or two after these latent pathological signs were elicited by such willed facilitation, the same responses could be obtained without requiring the willed effort of the patient. However, at this time the feeble, small finger-jerks could be facilitated in amplitude and speed by the simultaneous willed effort to flex the fingers.

At a time when voluntary flexion of the fingers still failed, an attempted flexion of the fingers while the examiner delivered a series of taps to the lightly stretched fingers at the rate of two taps per second resulted in flexion of the fingers so as to close them almost completely. When this phenomenon first appeared the resulting contraction of the finger flexors was a jerky, irregular movement. As each finger-jerk was elicited (the willed effort to flex the fingers being applied continuously) the relaxation following each finger-jerk was greatly delayed. Thus the fingers closed by accumulation of the increased after-discharge of the repeated finger-jerks (figs. 4 and 5). The flexion which occurred with this facilitation began in

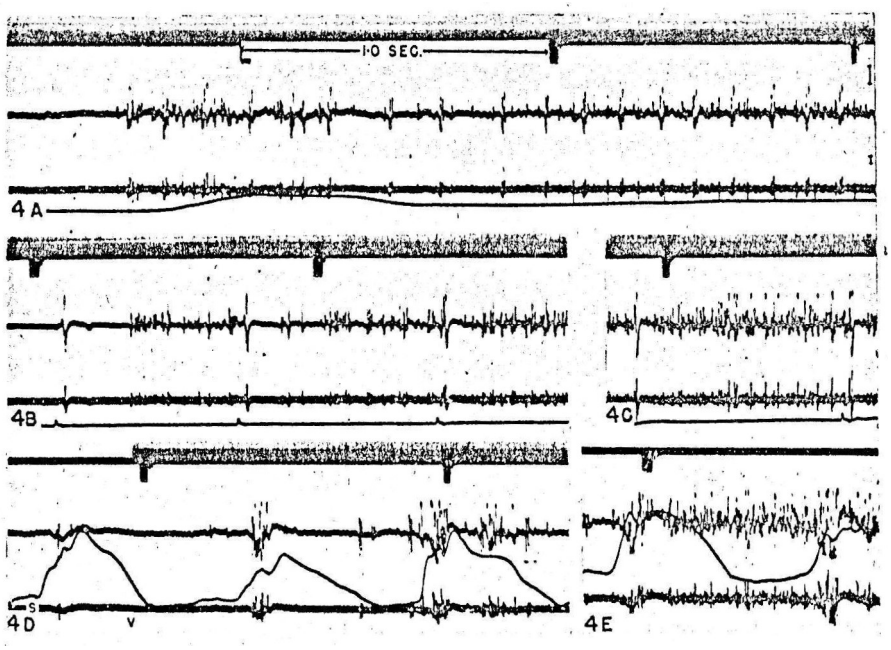


FIG. 4.—Electromyographic tracing of patient G. H. with right hemiplegia, Upper line for time intervals of one second. Both EMGs are from m, flexor digitorum profundus. Lower black line is tracing of balloon myograph over finger flexors. Calibration at 100 microvolts.

FIG. 4A.—Attempt at willed flexion of fingers alone.

FIG. 4B.—Willed flexion of fingers accompanied by repeated finger-jerks.

FIG. 4C.—Continuation of 4B. after about four seconds.

FIG. 4D.—Willed flexion of lingers accompanied by repeated passive stretch applied to the finger flexors. S. signifies passive stretch of finger flexors. V. signifies command to begin willed flexion.

FIG. 4F.—Continuation of 4D after approximately five seconds.

the two distal phalanges; flexion at the metacarpophalangeal joints began after the fingers were half-closed. The facilitated flexion continued for several seconds after cessation of willed effort, and the resistance in the finger flexors 'was then greatly intensified over that previously present, This increased resistance persisted for one to five seconds. During this period the finger-jerks were found to be more brisk and of greater amplitude than before the facilitated flexion.

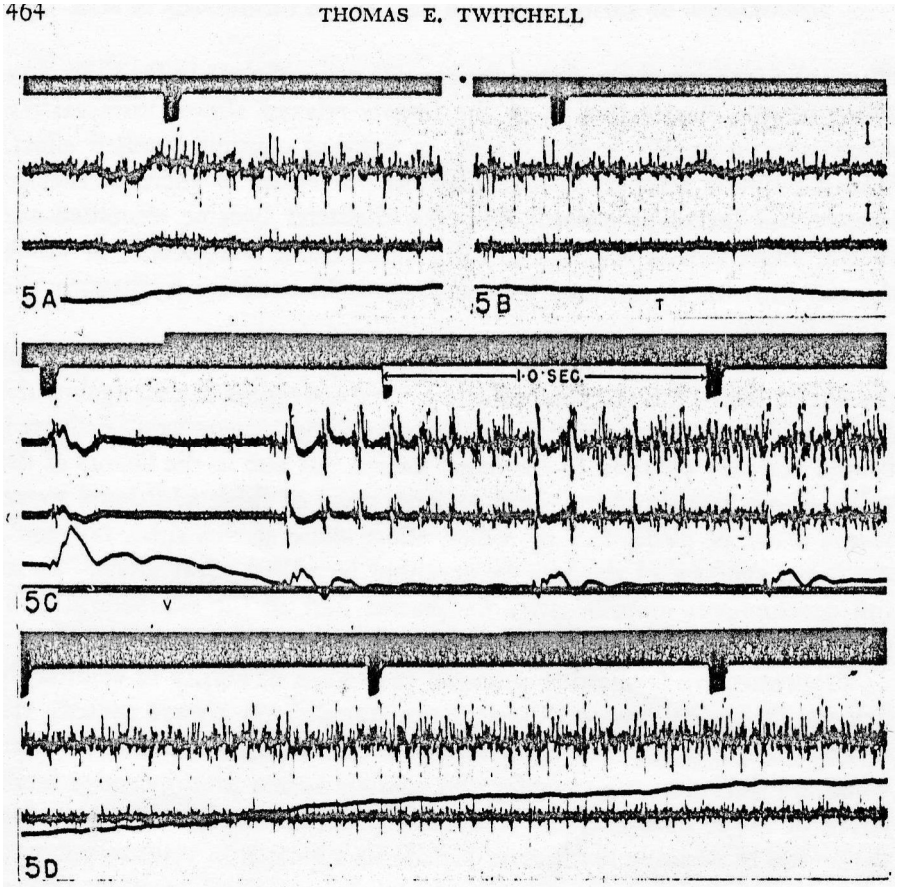


FIG. 5.—Electromyographic tracing from B. W. with left hemiplegia. Upper line time intervals of one second. Both EMGs from m. flexor digitorum sublimis. Lower black line for balloon myograph over flexor surface of index finger. Calibration at 500 microvolts.

FIG, 5A.—Attempt at voluntary flexion of fingers alone. The beginning of the attempt was quarter second before beginning of figure.

FIG. 5B.—Continuation of 5A, but one second later.
FIG. 5c.—Voluntary flexion with repeated finger-jerks. V marks beginning of voluntary effort.
FIC. 5D.—Voluntary flexion of fingers alone around three of the examiner's fingers. Note that the balloon myograph line rises higher in 5D than in 5A and 5B indicating an increased tension.

In 3 patients a latent period of four to five seconds existed between the time the command to flex the fingers was given and the facilitated flexion began, Gradual increase in the finger-jerks occurred towards the end of this period. These effects were obtained more rapidly within three to five days after the phenomenon was first noted. In the other patients thc finger-jerks and increased flexion of the fingers took place immediately f0110M'ing the command to flex, during a series of finger taps. A single tap on the tendons of the finger flexors was not sufficient to facilitate any willed effort. Active flexion of the fingers relaxed almost immediately when tapping on the fingers ceased, in spite of continued willed effort. With continued recovery this facilitation was easier to elicit, of shorter latency and increased power. Its jerky character became smoothed out so that finger flexion occurred as a sustained clonic contraction, of which the rhythm was determined by the finger-jerks used for facilitating the willed flexion.

With increasing facilitation other muscle groups beside the flexors of the fingers joined the response. If the patient attempted to pull against the fingers of the examiner while intermittent finger-jerks were being delivered, flexion not only occurred in the finger flexors but also in the flexors of the wrist, elbow, and shoulder. In 3 patients none of these additional movements could be performed by willed effort alone at this time, nor could the flexor synergy of the arm be executed by willed effort alone. In its full development proprioceptive facilitation had all the characteristics of the proximal traction response.

Proprioceptive facilitation presaged the return of ability to voluntarily flex the fingers by one to thirteen days, but did not always precede the earliest return of any voluntary movement in an arm. In 4 cases some voluntary flexion at the shoulder was possible before proprioceptive facilitation of finger flexion could be obtained, After the first ability to flex the fingers in response to command made its appearance, voluntary flexion accompanied by periodic tendon-jerks or by sustained traction against the fingers was more powerful and of greater range.

Other means of facilitating willed effort by proprioceptive stimuli were investigated. Passive hyperextension of the fingers, to set up a stretch reflex in the finger flexors, was found to be ineffective when proprioceptive facilitation first made its appearance. Within two days following thc appearance of facilitation tendon reflexes, some facilitation occurred with passive stretch of the finger flexors in 2 patients, before the ability to flex the fingers in response to willed effort alone had returned. The latent period 'of voluntary flexion was shortened less by passive hyperextension than by repeated tendon-jerks, and the flexion was not as complete in range or as powerful. Later, when some ability to contract the finger flexors in response to willed effort had returned, passive hyperextension of the flexors enabled a more powerful voluntary flexion. Repeated periodic passive extensions were more effective than one (figs. 4D land 4E).

Facilitation was also attempted by hyperextending only one finger, but this was the least effective method, It enabled only a stronger willed flexion of the finger extended and a very slight flexion of the distal phalanges of the finger on either side of the extended one. However, after weak voluntary flexion of the fingers could be accomplished, this means of facilitation enabled the patient to flex all of his fingers more effectively than by willed effort alone, but never as completely or as powerfully as when stretch of all the fingers or finger-jerks were used for facilitation. It was, of course, necessary to stretch all the, finger flexors slightly in order to elicit finger-jerks.

When these proprioceptive facilitating mechanisms first made their appearance, the reactions could be rapidly fatigued by several trials in succession. There was, however, no change in the amplitude or briskness of the finger-jerks or in the resistance to passive movement, both of which continued to become even more exaggerated. It was apparent that the willed part of the response was the seat of fatigue. Proprioceptive facilitation of finger flexion was obtained in 12 patients who were unable to flex the fingers in response to voluntary effort alone at that time. This facilitation followed the onset of hemiplegia by eight to forty-six days; it followed the first appearance of spasticity by one to forty-three days.

FACILITATION BY RIGHTING REFLEXES

Certain reactions caused a change in the resting posture, and in the intensity and distribution of spasticity in the hemiplegic limbs. Since passive turning of the patient's head by the examiner was usually ineffective in eliciting a tonic neck reflex, the response was usually elicited by having the patient voluntarily and forcefully turn his head to one side or the other. Such intensification of tonic neck reflexes was effective in 6 patients in whom the resistance to passive movement was quite marked in the flexors of the arm and extensors of the leg. In these patients, however, the change in the position of the head affected the arm only. In no case wag any effect seen in the leg,

Only in one patient with moderate resistance present at all joints in the upper extremity, including the shoulder, was an actual change in posture of the hemiplegic arm observed. Thus when the patient forcefully turned his face as far as possible to the side of the hemiplegia there was an extension of the hemiplegic arm. The wrist pronated and dorsiflexed slightly and the fingers flexed in moderate degree. With the face turned away from the hemiplegic side, the arm flexed strongly at the elbow and adducted at the shoulder. The wrist supinated, and the fingers remained flexed. Resistance to passive movement underwent corresponding increase in the active muscles. In the other 5 patients, although no active change in the posture of the hemiplegic limbs occurred with turning of the head, a striking change took place in the intensity and distribution of resistance to passive stretch. Thus when these patients forcefully turned their faces to the hemiplegic side an increase in the resistance to passive movement could be demonstrated in the extensor muscle groups, while the resistance in the flexor groups diminished. The diminution in resistance to passive stretch in the flexors resulted in its complete abolition in only one case. In the other cases the flexors always presented more than normal resistance, but not as much as the

extensors. With the face turned away from the hemiplegic side, resistance to passive stretch increased in the flexors and decreased in the extensors. In 2 cases the resistance in the extensors disappeared completely.

The effect of body position on the posture and resistance to passive movement of the hemiplegic limbs was even more striking, With the patient lying in the supine position the upper limbs were flexed and the lower extended. If the patient turned on his side with the hemiplegic limbs uppermost, the flexion of the elbow, wrist and fingers was greatly increased. Likewise, there was great intensification of resistance to passive movement in the elbow, wrist and finger flexors, and to a lesser extent in the retractors and adductors of the shoulder. In one patient the leg also flexed at the hip and knee. In the other patients, however, any effect on the leg was best demonstrated by passive manipulation of the extremity, whereby an increased resistance could be shown in the flexor muscles and hip adductors. The diminution in extensor resistance was more evident, however. The tendon reflexes in the upper limb all became more brisk, with wider amplitude, and occasionally initiated clonus.

When the patient was on his side with the hemiplegic limbs lowermost, an extension of these limbs took place, including some extension of the fingers. The postural change was more evident in the arm than in the leg, although one patient showed active extension of the leg. There was now a pronounced redistribution of resistance in thc various muscle groups, being greatly increased in the extensors and reduced in the flexors. Indeed in one case, all flexor resistance disappeared, but in the others slightly greater than normal resistance still remained. The tendon reflexes again exhibited a remarkable change. Instead of being clonic and of great amplitude, they were now small, rapid, single contractions. Indeed the finger-jerks were very feeble and difficult to elicit. The electromyographic features of this reaction from a patient studied also by Drs. Seyffarth and Denny-Brown (1948) are illustrated in fig. 3.

In work with Dr. Denny-Brown on pyramid lesions in the monkey, the author learned to recognize the characteristics of a righting reflex which was manifested as a facilitation of flexion of all joints in response to traction on the flexor and adductor muscles of the shoulder. If the traction was made by pulling on the fingers, this response must be distinguished from the grasp reflex, by excluding a moving tactile element from the stimulus, and by the strong associated flexion of wrist and elbow which is characteristic of it. When proprioceptive reactions were most highly exaggerated in human hemiplegia this response, which we propose to call the "proximal traction response," could also be obtained.

The response in the hemiplegic patient was a slowly developing, strong flexion of the shoulder, elbow and wrist which could be reinforced by various other reactions, and then included flexion of fingers. The reaction depended on stretch or traction applied to the flexor muscles of the limb, each of which reinforced the others. Thus although simple traction on the finger flexors themselves did not reveal anything more than an increased resistance to passive stretch, traction on the flexors of the fingers of sufficient force to cause also traction on the wrist and elbow flexors and through these traction on the shoulder muscles, was then followed by increased flexion of the fingers together with an associated increased flexion of the wrist and elbow. If the traction

was made on the wrist or elbow flexors alone, and not on the finger flexors, with enough force to cause traction on the shoulder, flexion was intensified in the wrist and elbow flexors, or elbow flexors alone. However, when the reaction was prominent, the fingers also flexed with traction on the wrist flexors. With this method of elicitation it was clear that the response differed completely from the grasp reflex. This response fatigued easily, and renewed traction on the flexors involved was necessary to reinforce it,

The proximal traction response was reinforced to some extent by the operation of the tonic neck reflexes. Thus with the patient facing away from his hemiplegic limbs, the elicitation of the traction response resulted in a considerably more powerful flexion than that obtained with the patient facing forward. The effect of body posture on the proximal traction was response even more striking. With the patient lying on his side with the hemiplegic limbs lowermost, however, the response was abolished and could not be obtained by any stimulus or combination of stimuli (fig. 1B). When the hemiplegic limbs were uppermost the response was greatly intensified (fig. 1 c).

When fully developed the proximal traction response could also facilitate movements in response to willed effort. Thus flexion of the fingers was greatly facilitated if, while the patient voluntarily flexed his fingers, the examiner exerted traction against the fingers causing secondary traction on the elbow and shoulder. The response then became a more powerful flexion at all these joints, and the flexor synergy resulted. Likewise, the ability to flex the limb by willed effort was facilitated by the tonic neck reflex and body righting reflex. The same process doubtless underlies the spread of facilitation with finger-jerks to affect a flexion not only of the fingers, but also of the wrist, elbow and shoulder.

The development of the traction response was followed in 12 patients, This response was first obtained When other types of proprioceptive facilitation were already present, and usually two or three days after the onset of localized proprioceptive facilitation. In 3 patients the traction response could not be obtained until five or six days after facilitation by tendon-jerks and simple stretch first occurred.

CONTACTUAL FACILITATION AND THE GRASP REFLEX

With the first return of willed flexion of all the fingers, this flexion could be facilitated by means other than the proprioceptive methods described above. Thus when willed flexion of the fingers was only a weak and incomplete movement (the patient being able to close his fingers only half-way, for example) a contactual stimulus to the palm of the patient's hand simultaneously with the patient's willed effort to flex his fingers led to a more powerful and complete flexion of the fingers. An effective contactual stimulation could be delivered in several ways. In the most simple methods the examiner used his fingers as the stimulating agent. Either the examiner stroked the palm of the patient's hand with his finger tips, moving in a distal direction out on to the palmar surface of the patient's fingers, or the examiner could draw his fingers across the patient's palm out between the thumb and index finger. The first method was the more effective. In order to demonstrate this effect it is important that the stimulus be light enough not to stretch

the flexor tendons and so introduce an added factor of proprioceptive facilitation. With the first appearance of such contactual facilitation a stimulating agent of broad surface was necessary, and stimulation with an agent, of small contacting surface, such as a pencil, was ineffective. At that stage it was also necessary to use a moving stimulus, stationary contact with the patient's palm being completely ineffective.

Such contactual facilitation, unlike proprioceptive effects, occurred only when some response to willed effort was already possible. Two to three days after its first appearance contactual facilitation became more effective and stationary contact alone could then facilitate willed flexion of the fingers. The stimulating agent still had to be of broad surface area, however. Thus, if the patient was told to close his fingers round the examiner's hand, which had been placed in the patient's palm, a more powerful and to' flex his fingers without contact (fig. 5D). Like proprioceptive stimuli these contact stimuli could also reduce the latent period preceding thc onset of voluntary flexion.

Contactual facilitation could be demonstrated in 10 patients of the long-term series, and occurred within eight to fifty-four days after the onset of hemiplegia.

The grasp reflex.—As movement of the shoulder and elbow became more powerful, spasticity abruptly lessened in the shoulder and elb0M' muscles. Likewise as movements of the wrist and fingers became more powerful and could be effected without development of the flexor synergy of the arm, spasticity in the wrist and finger flexors also suddenly lessened. At that time a contact stimulus alone could initiate a flexion of the fingers which was identical with the grasp reflex. Although the grasp reflex was obtained only with a decline of spasticity, the activity of the tendon reflexes could, nevertheless, continue to increase for a time. For example, in one patient, in whom a moderate spasticity of the wrist and finger flexors declined abruptly on the twenty-ninth day following the onset of hemiplegia, the grasp reflex was first elicited on that day. Within a few days the finger-jerks, which had previously been hyperactive but not clonic, were first shown to be clonic. The patient recovered completely in another thirty days and the clonic element by then had disappeared.

Seyffarth and Denny-Brown (1948) have defined the grasp reflex as "a stereotyped prehensile reaction of the flexors and the adductors of the fingers, and the flexor muscles of the wrist, which can be elicited in fractional parts by appropriate localization of -the stimulus. The adequate stimulus for the full reaction is dual. The first essential is a distally moving deep pressure over a specific area of the palmar surface Of joints in the hand, which elicits a rapid brief muscular contraction (the 'catching' phase), which develops into a strong 'holding' phase only if traction is made upon the tendons of the flexor or adductor muscles thus thrown into preliminary contraction. The response is then maintained only by traction."

In 5 of our patients the development and elaboration of the grasp reflex was studied. A grasp reflex of the elementary type described by Seyffarth and Denny-Brown, which requires a heavy pressing, moving contact for its elicitation, was first obtained twenty-three to forty days following the onset of hemiplegia, and preceded complete recovery, according to our criteria, by twenty to

forty days (see Table I).

The ability of the patient to move the fingers was profoundly modified the appearance of these contactual reactions. Prior to the appearance of contactual facilitation of willed movement first mentioned above, voluntary flexion of the fingers was very weak. The range of movement was extremely limited, the fingers being able to flex only 2 or 3 cm. Contactual facilitation itself enabled the fingers to flex completely, with considerable increase in power. Although contactual facilitation appeared

THE RESTORATION

TABLE I

	Finger-jerks	Spasticity	Proprio facil.	Traction response	Vol. movt. hand	Tactile facil.	Grasp reflex
Complete recovery							
G. H.	9 days	9 days	9 days	10 days	10 days	16 days	40 days
N. F.	48 hours	48 hours	8 days	13 days	15 days	15 days	23 days
*C. Y.	48 hours	48 hours	8 days	8 days	11 days	11 days	29 days
†K. B.	48 hours	48 hours	48 hours	48 hours	7 days	8 days	23 days
A. M.	6 days	48 hours	6 days	6 days	12 days	14 days	26 days
‡H. C.	48 hours	9 days	9 days	9 days	15 days	?	?
Recovery incomplete							
J. H.	18 days	18 days	18 days	18 days	23 days	26 days	0
M. C.	48 hours	48 hours	18 days	18 days	19 days	?	0
J. A.	48 hours	48 hours	15 days	15 days	25 days	26 days	0
Flexor synergy							
R. B.	20 days	20 days	31 days	36 days	43 days	46 days	0
W. W.	3 days	3 days	46 days	46 days	54 days	54 days	0
B. W.	48 hours	48 hours	25 days	31 days	38 days	39 days	0
E. D.	9 days	9 days	?	?	30 days	?	0
No recovery							
J. N.	6 days	5 days	0	0	0	0	0
W. M.	28 days	9 days	0	0	0	0	0
M. M.	3 days	48 hours	0	0	0	0	0
G. M.	7 days	48 hours	0	0	0	0	0
§M. M.	48 hours	48 hours	0	0	0	0	0
B. T.	48 hours	48 hours	0	0	0	0	0

*t These patients were able to move their upper limbs at the shoulder and elbow by effort of will alone at the time of admission. They could never execute willed movements of the wrist and fingers, however.

$This patient died of pneumonia before the recovery process had halted. Because of the time-sequence of events in early recovery he is included under the group who recovered completely.

$This patient did recover the ability to flex the upper arm at thc shoulder.

in these patients when willed effort produced only the flexor synergy, the movements at each joint could not be performed separately until after the appearance of contactual facilitation With the appearance of the grasp reflex, willed movement of the fingers was even more profoundly changed. Prior to the appearance of the grasp reflex, the finger movements were hindered by spasticity; they were slow and clumsy, and stiffness appeared to contribute largely to the defect. Isolated movements of the digits and opposition of the thumb and fingers, if present, were carried out only with the greatest difficulty. Attempts to move a finger alone often could not be accomplished, and any attempt to do so resulted in movement of all the fingers. Prior to the appearance of the grasp reflex, all of the defects described were made much worse if the patient was blindfolded so that he could not watch his fingers. This occurred even though no sensory defect could be demonstrated. After the appearance of the grasp reflex, finger movements were much more powerful and dexterous. Then slowness and clumsiness due to spasticity was greatly reduced. Speed of movement was greatly increased. Isolated movements of the fingers and finger-thumb opposition could be performed with relative ease.

The proximal traction response and the grasp reflex might be confused, but they are two entirely different reactions. The traction response was obtained when proprioceptive reactions were at their height. The grasp reflex on the other hand was only obtainable with a decline of spasticity. Stretch of the muscles concerned was not a primary factor in obtaining the grasp reflex. Indeed, the grasp reflex could be obtained with the wrist flexed to 90 degrees so that the flexor muscles were well relaxed and the element of stretch was eliminated. The fully developed grasp reflex involved flexion and adduction of the fingers and only secondarily a flexion of the wrist. The strong flexion at the wrist and elbow which was seen in the traction response did not occur in the grasp reflex. Finally, the grasp reflex was not influenced by the operation of the tonic neck and body righting reflexes.

RELATIONSHIP OF THE VARIOUS PHENOMENA TO ULTIMATE RECOVERY

19 patients were studied in detail with observations over a prolonged period, with special reference to the inter-relation of the various postural mechanisms and the recovery of voluntary movement. 17 of these patients could not move the arm at the time of admission. 2 could perform willed movements of the shoulder and elbow when first observed, but not of the wrist or fingers. At the end of the period of observation 5 of these patients recovered completely; 5 still could not move any part of the arm; one could only flex the shoulder; 4 could only execute the flexor synergy of the arm; and 3 recovered all movements of the aril), though spasticity and weakness remained to a marked degree. 1 patient who had recovered considerable voluntary movement died of pneumonia while recovery was still in progress.

Although return of the tendon reflexes was the first evidence of recovery of motor function, simple hyperactivity of the tendon reflexes with the exception of the finger-jerks could not be related to recovery of willed movement. For, with the exception of the finger-jerks, the tendon reflexes in all our patients were hyperactive within forty-eight hours following the onset of hemiplegia. To some extent, a correlation could be made between the return of the finger-jerks

and the degree of recovery, but it was certainly not absolute. Of 5 patients who recovered completely, 3 showed hyperactive finger-jerks within forty-eight hours after the onset of hemiplegia, and the other 2 showed hyperactivity of the finger-jerks six and nine days after the onset of hemiplegia. In 5 patients who recovered no voluntary movement whatsoever, the finger-jerks became hyperactive within forty-eight hours after the onset of hemiplegia in one; while in the others hyperactivity appeared in three, six, seven, and twenty-eight days after the onset of hemiplegia. Patients who ultimately recovered only the flexor synergy required forty-eight hours, and three, nine, and twenty days to develop hyperactive finger-jerks. Of 3 patients showing further recovery, 2 showed hyperactivity of the finger-jerks within forty-eight hours after the onset of hemiplegia while the other showed no hyperactivity until eighteen days after the onset of hemiplegia. Therefore, early return of finger-jerks more frequently presaged a more complete recovery of motor function than late return of finger-jerks. These findings are summarized in Table I.

The time of onset of spasticity paralleled the onset of hyperactivity of the finger-jerks. Although the intensity of spasticity was mild in all the cases at its onset, spasticity never became very intense in patients who later recovered completely. The maximum intensity in these patients was reached within twenty days at the most, after the onset of hemiplegia. In patients who recovered completely, spasticity was not detected in the shoulder. Clonus could be demonstrated in only I of these patients, and the clasp-knife phenomenon in only 2. In patients who did not recover completely, spasticity became much more intense in its later development. In 2 of these patients the maximum intensity was not reached until six months after the onset of hemiplegia. 12 patients exhibited spasticity in the shoulder groups well as in the flexors of the elbow, wrist and fingers. Spasticity in the elbow extensors was encountered more frequently in this group. Wrist or finger clonus seen in 5 patients and the clasp knife phenomenon in 2.

The relationship of proprioceptive facilitation and the traction response to the return of voluntary movement was striking. In Table I it will be seen that in the patients who recovered completely, proprioceptive facilitation could be elicited within nine days following the onset of hemiplegia and the traction response within thirteen days. In the patients who did not recover completely these reactions were not elicited until at least fifteen days after the onset of hemiplegia. It will also be noticed that in the latter group, these two reactions could be elicited earlier after the onset of hemiplegia in those patients recovering some independent movement at each joint, than in those recovering only the ability to perform the flexor synergy. These reactions could not be obtained at any time in the patients who did not recover movement of the hand. However, this does not mean that proprioceptive facilitation or the traction response could not be obtained in the absence of voluntary movement or that 'the ability to elicit these responses was an absolute sign of later recovery of willed movement. One striking example was that of a patient who had had a stroke six months prior to admission to the hospital and was left with a spastic hemiplegia with total inability to move the left arm. Nevertheless, in this patient proprioceptive facilitation with finger-jerks could produce a flexor synergy of the arm. Apparently the recovery process had halted at this early stage. In this series it is evident that the 6 patients shown in Table I who did

not recover the ability to perform willed movement had not even reached the stage in the recovery process at which proprioceptive facilitation appeared.

We may conclude that the most reliable prognostic sign for recovery from hemiplegia was the time of occurrence of proprioceptive facilitation and the proximal traction response. Although late return of tendon reflexes and late onset of spasticity certainly are poor prognostic signs of voluntary movement, there are exceptions to this rule. Only the occurrence of proprioceptive facilitation and the traction response proved to be infallible prognostic signs of recovery of willed movement.

With the appearance of facilitation by contactual stimuli the previously weak and incomplete movements became greater in amplitude and considerably increased in power of voluntary movement. Ability to perform only one part of the flexor synergy without the others did not begin to take place until willed movements could be facilitated by contact stimuli, but the first signs of contact facilitation could appear in the stage of the flexor synergy.

The grasp reflex could be obtained only in those patients who later recovered completely. It was never obtained in the absence of some voluntary movement. Movement was gravely defective in the presence of spasticity. With the return of the grasp reflex, spasticity of the wrist and finger flexors abruptly diminished. At this time, both power and range of movement increased and dexterity greatly improved. Prior to the appearance of the grasp reflex, flexion and extension of the fingers was slow and clumsy. Opposition of the thumb to all the fingers was extremely difficult; at this time in no case could the thumb be opposed to the tips of the fingers, nor could the thumb be opposed to the little finger. Attempts to move one finger alone usually resulted in an associated flexion of all the fingers. In several cases the index finger could be feebly moved alone prior to the appearance of a distinct grasp reflex, but attempts at movement of the little finger caused an associated flexion of the ring finger. Following the appearance of the grasp reflex, opposition of the thumb to all the fingers could be performed with a great degree of rapidity and dexterity. Movements of one finger alone could then be performed without causing associated movements of other fingers, and fatigue was considerably reduced.

If vision was excluded, movement was performed only with great difficulty prior to the appearance of the grasp reflex. Any willed movement was then diminished in speed and dexterity if the patient was not allowed to watch what he was doing. In attempting to oppose the thumb to each individual finger, the patient failed to oppose the thumb and fingers in several instances and missed one or two fingers entirely. This motor defect occurred even though no disorder of sensory perception could be demonstrated. With the appearance of the grasp reflex the motor defects introduced by the exclusion of vision were found to be markedly reduced. Occasionally there was some hesitancy before beginning a movement, but the marked ataxia and inability to execute certain more difficult movements such as finger-thumb opposition did not occur. With complete recovery and final disappearance of spasticity exclusion of vision did not cause any movement defect whatsoever.

DISCUSSION

Our investigation revealed that hemiplegia, except in a late stage of its most severe and complete form, is neither static nor stereotyped. Great variation was found in each of the classical criteria of distribution of paralysis and process of recovery. This is manifestly determined in part by differences in the situation and type of causative lesion. Nevertheless, in this series of cases an orderly progression of associated phenomena was found to characterize the course of events in any particular muscle group. The process of recovery in the paralysed hand or foot was a constantly evolving series of reactions and followed a general pattern with some variations. With the onset of hemiplegia the affected limbs were completely paralysed, with diminution of the tendon reflexes and resistance to passive movement. Within forty-eight hours the tendon reflexes became hyper active and resistance to passive movement increased in the wrist and finger flexors and the plantar flexors of the ankle, This resistance became more intense over a period of time and involved other muscle groups, particularly the flexors and adductors in the upper limb, and the extensors and adductors in the lower limb. The tendon reflexes became more brisk and sometimes clonic.

The return of voluntary movement appeared first as flexion at the shoulder and hip. Later flexion of the elbow, wrist and fingers, and knee and ankle were added to these movements. At this time any attempt at willed movement resulted in a flexion of the shoulder, elbow, wrist and fingers, or hip, knee and ankle together. Thus the "flexor synergies" of the arm and leg became manifest as total reactions. In later, but overlapping sequence, the extensor synergies of the arm and leg developed,

When the tendon reflexes had become hyperactive, it was possible to facilitate willed flexion of the upper extremity by a series of tendon reflexes and by stretching. At this time the proximal traction response also appeared. This was an enhancement of the spasticity in the flexors at all joints when traction was made on the shoulder flexors and to a less degree when wrist, finger or elbow flexors were stretched. This response could also facilitate willed flexion of the arm. Both the first willed flexion and the traction response activated the same muscles and could be facilitated or depressed by action of the tonic neck and body-righting reflexes. The body-righting reflex had the most profound effect, so that with the patient lying on his side with the paralysed limbs uppermost, both the traction response and the effect of willed effort were greatly facilitated. With the patient lying on his side with the paralysed limbs lowermost, the traction response was abolished and willed movement was extremely weak and limited in range. Our attention had been directed to the proximal traction response in a study of hemiplegia in the monkey following ablation of area 4 and section of the medullary pyramid, of which a preliminary note has been published elsewhere (Denny-Brown, Twitchell and Saenz-Arroyo, 1949). In such experiments this response was utilized by the animal in the first recovered spontaneous movement. We were convinced that in the monkey it is identical with the reflex grasping of the thalamic animal, and was primarily a body-righting reflex. The relationship found in this study identifies the same responses as the first stage in recovery from hemiplegia in man, and its development as the first willed movement.

It was therefore clear that in its beginning, the return of ability to flex the fingers was a part of the total flexor synergy of the arm, and occurred together with flexion of the wrist, elbow, and shoulder. The next event in recovery of movement was the occurrence of ability to flex either shoulder, elbow, all the fingers, or wrist, each without the others. This separation of the elements of the synergic complex was only gradually achieved. While it was still only incompletely attained, it was found that flexion of all the fingers together could now be greatly facilitated by con tactual stimulation of the palm of the hand. If power and dexterity of voluntary movement continued to improve, a stage was reached where spasticity abruptly lessened, first in the shoulder and elbow muscles, and later in the flexors of the wrist and fingers. In close association with this decline of spasticity in the wrist and finger flexors the grasp reflex returned, and with it the first ability to perform independent movement of the fingers. The effect of a contactual stimulus moving distally in the palm was no longer just facilitation of willed effort, but alone was adequate to initiate the small active flexion which Seyffarth and Denny-Brown (1948) termed the catching phase, and upon which stretch of tendons elicited the proprioceptive phase of the grasp response. Further, this reaction could be fractionated by the limitation of the stimulus to the palmar surface of one finger, then inducing a "catching" flexion only of that finger. Coincident with the appearance of such fractions of the grasp reflex there was found the ability to move each finger independently, Finally, when willed movement of the affected limb became as rapid as dexterous as the unaffected, spasticity disappeared completely.

The course of recovery has been more particularly studied in relation to the use of the hand and upper limb, though the data accumulated regarding the recovery of the lower limb indicated that a similar series of changes was occurring. Whereas the primary events of recovery in the upper limb are associated with the performance of prehension, and therefore flexor reactions, those of the lower limb soon became dominated by extensor activity. We have not attempted to determine the significance of such extension, though it is clearly related to the reflex mechanisms of walking and standing.

Recovery of movement in the upper limb could in general be divided into three distinct stages, the first being dominated by proprioceptive reactions, the second by contactual stimulation of the extremity, and finally a seeming total independence of movement upon such externally applied agents. It was apparent that vision had a facilitating effect in the first two stages, but at a period when the grasp reflex had become well developed, exclusion of vision gradually ceased to cause any resulting defect in the performance of movement. Movement-freely projected in space without visual control is the last accomplishment to be attained.

Our findings indicate that the different phases of the return of ability to make willed movements in hemiplegic limbs are associated with the appearance of a series of responses each of which is derived from stimulation of the limb concerned. In the earliest stages the basic responses are simple proprioceptive and contactual reactions. Their successive modification as recovery proceeds allows the assumption that the ability to move a part by effort of will alone is itself a modification of these elementary contactual and proprioceptive reflexes. The static spasticity of residual states when power of voluntary movement remains minimal or absent is derived from

imperfect integration of these segmental responses. Though this study does not attempt, to provide thc explanation for the relatively greater spasticity of some residual states compared with others, it does indicate that the absence of voluntary movement is associated with a relative absence of motor adaptation as a whole.

These findings provide further evidence for the thesis advanced by Denny-Brown (1950) that the rolandic region of the cerebral cortex is a "stereognostic apparatus for exploring space." based upon elaboration of contactual modifications of proprioceptive reactions. He pointed out that the first event in disintegration of motor function in progressive frontal lobe lesions is commonly the release of certain cortical motor automatisms, such as instinctive grasping, from their natural integration with total behaviour. When these motor reactions become impaired, the relatively coarse triggering of proprioceptive reactions by contact stimuli, such as the grasp reflex, makes its appearance. Finally, with the loss of the contact conditioning, the proprioceptive reactions are fully released in the form of spasticity. In recovery of function the reverse sequence could occur. The present investigation of a large number of cases of cerebral hemiplegia reveals the regularity of operation of the same factors in the sequence of recovery of function. The steps in recovery from loss of motor function in the disorder known as hemiplegia begin with simple and then more complex proprioceptive reactions. These in turn become modifiable by contactual reactions, each of which is soon adapted to the purpose of will.

The great disability which results when recovery is halted in the phase of heightened proprioceptive activity has prompted many earlier investigations. Walshe (1919) clarified the previously confused views as to the nature of spasticity, and showed its identity with the type of exaggeration of postural reflexes seen in decerebrate rigidity. The analysis of Sherrington and his collaborators subsequently identified the stretch reflex as the fundamental reaction of such disorder. It has often been assumed that if spasticity could be abolished, willed movement could be more effectively performed. Though this might be possible in certain spinal disorders, the present study indicates that the first movements to appear following hemiplegia are themselves facilitated stretch reflexes. The problem at that stage is not so much to abolish the spastic reaction, as to harness diffuse hyperactivity.

The return of the tendon reflexes (with the exception of the finger-jerks in several instances) was found uniformly to be the earliest event in the course of motor recovery. This was closely followed by the first evidence of heightened response to sustained stretch, In its first appearance the returning stretch response was a soft, yielding resistance felt at the end of a full range of passive stretch of the flexor and adductor muscles of the arm and the extensor and adductors of the leg. It appeared first in the wrist and finger flexors and ankle plantar flexors, then in the elbow flexors and knee extensors, and still later in the retractors and adductors of the shoulder and extensors and adductors of the hip. At its full development increased resistance to passive stretch showed the generally recognized qualities of spasticity, for the resistance in any muscle group when encountered rose to a maximum as stretch was continued and abruptly declined towards the end of passive movement. In the course of development of spasticity in any muscle group the resistance to passive stretch appeared earlier in the course of passive lengthening of the muscle.

The clasp-knife phenomenon was of late appearance, in general within two to four weeks after the first appearance of increased resistance, and was observed in the flexors of the elbow and the extensors of the knee. In the presence of clonic tendon reflexes the initial peak was well developed, the subsequent decline rapid, and at times rhythmical subsequent peaks of resistance showed the well-known relationship between the occurrence of clonic tendon reflexes and "sustained clonus." We therefore regard the clasp-knife phenomenon and clonus as having some general relationship. It was not possible to correlate the appearance of the clasp-knife phenomenon or clonus with any variation in the quality or intensity of spasticity. They occurred in patients who made a complete recovery and in patients who never recovered the ability to produce movement by effort of will alone. It was apparent that the resistance to passive movement and the tendon reflexes did not always develop in parallel manner. That is, these reactions did not necessarily become exaggerated or subside at the same time. Thus it was seen that the tendon reflexes could remain hyperactive for a long time before spasticity made its appearance. And in one patient the finger-jerks became clonic after the subsidence of spasticity in the finger and wrist flexors, with increasing power of willed movement.

The appearance of spasticity of some degree in the flexor groups of the upper limb, the extensor groups of 'the lower limb, and the adductor groups of both limbs was general in the series of cases studied. "Flaccidity" was a transient phenomenon in these muscles, although there was great variation in its duration. Some spasticity appeared in addition in the opposing muscles, particularly in the extensors of the elbow and the flexors of the knee, in about one-fifth of the patients, but was without significance in relation to course or ultimate recovery. On general principles, anomalies in the duration of flaccidity, and in the distribution of paralysis and spasticity may be expected to be related to the extent and location of the cerebral lesion. As no autopsy material is available from the case material presented, we have not attempted such correlation. Although there was not complete correlation, there was a suggestion of poor prognosis for recovery if the period of "flaccidity" was long. We were not able to determine any difference between the spasticity of the completely paralysed hand, and that which preceded return of voluntary power, except that the latter was, in those cases in which it was observed, more easily modified by neck and body-righting reflexes, and by traction on the proximal segments of the limb. Early return of the proximal traction response, and of facilitation of willed flexion of finger flexion by a series of finger-jerks had good prognostic significance for later recovery of willed movement.

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 re-training of movement.

CONCLUSIONS

If motor function recovers following the' hemiplegia caused by a lesion of the cerebrum, the common course of recovery shows a regular sequence of reflex changes, each of which is associated with a corresponding increase in ability for willed movement. The process of recovery may become arrested at any stage in this sequence. After the initial phase of depression of all motor function, the proprioceptive responses become abnormally active, These proprioceptive responses do not constitute a simple entity, for they are modified and conditioned by other factors, such as stretch on associated muscles, the position of the patient's head in relation to his body, and the position of the body in relation to ,the supporting surface, and later by certain contactual stimulation. As the recovery process proceeds, the more elementary proprioceptive responses become subordinated to special exteroceptive stimulation. Voluntary movement appears as a further facilitation of the available responses at each stage. It is not a separate entity, but from its first appearance it takes the form of conditioned proprioceptive and contactual responses.

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