The Process of Recovery After Hemiplegia

BY MICHAEL NEWMAN, M.D.

Introduction

Some knowledge of the natural history of any disease condition is necessary, not only in order to plan management and to judge the effect of treatment, but also to test the credibility of theories of the underlying mechanism. One must try to understand what disease processes and what mechanisms of natural recovery are taking place and in what way treatment can be expected to modify them. It is well known that hemiplegia following cerebral thrombosis or embolism has a tendency to recover. What processes in the brain and circulation contribute to this recovery? Although much of the work of rehabilitation after a stroke is directed toward prevention of complications and of other factors which obstruct recovery, it may well be that some of the natural processes could be aided or speeded up if one could understand exactly what is going on. Furthermore, any regime of management must be judged against a standard of what is likely to occur naturally, without specific treatment. A number of physiological and psychological processes can be considered as contributing toward recovery. It is interesting to compare the time course of such factors with the actual sequence of recovery as seen in the hemiplegic.

Subjects

Thirty-nine patients admitted to the Manitoba Rehabilitation Hospital were selected. These patients were all transferred from acute hospitals within four weeks of a sudden stroke producing hemiplegia and diagnosed as either thrombosis or embolism. Patients transferred later than one month from onset, and those suffering from serious general disease or complications, were excluded from the series. There were 15 women, ranging in age from 28 to 77 years, and 24 men, ranging in age from 22 to 72 years. The average age was 55. Twenty-five of the patients had a right
RECOVERY AFTER HEMIPLEGIA

AGE, SEX AND SIDE OF HEMIPLEGIA

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<tr>
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<th>MALE</th>
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<td>RIGHT</td>
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<td>LEFT</td>
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AGES

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Distribution of patients studied.

hemiplegia, with speech disorder in 19 of these, and 14 had a left hemiplegia. The cause of the hemiplegia was considered to be embolism from known cardiac abnormality in three of the cases and was diagnosed as thrombosis in the others.

Measurement of the Hemiplegia

The normal type of diagnostic neurological examination is insufficient to give any useful data about the process of recovery of hemiplegia. Some form of quantitative, or at least semiquantitative, estimate of the neurological disorder is needed. The ordinary MRC grading of muscle strength is not satisfactory because, in hemiplegia, the muscles do not recover individually or independent of posture and associated movements. On the other hand, a pure measure of motility, such as that described by Stern et al., may be beyond the ability of most of the patients and so fails to register much useful recovery which does occur. For this reason, a simplified numerical assessment of motor and sensory function and higher mental functions was undertaken with a parallel assessment of functional recovery. Those items were selected which have been shown by experience as likely to show improvements. Recovery of hemianopia, for instance, is exceedingly uncommon and therefore was not included in the numerical assessment. Table 1 indicates the points system used. In the upper limb, shoulder movement was graded 3—1 point for ability to contract the trapezius, 2 for active abduction of the shoulder in the sitting position, 3 for full power

<table>
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<tr>
<th>Neurological Score</th>
<th>1 point</th>
<th>2 points</th>
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<tr>
<td>Upper limbs  9 points</td>
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<tr>
<td>Shoulder Trapezius contracts</td>
<td>Adduction less than 90°</td>
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<td>Elbow Flexion synergy</td>
<td>Hand to mouth</td>
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<tr>
<td>Hand Flexion synergy</td>
<td>Active extension</td>
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<tr>
<td>Lower limbs  9 points</td>
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<tr>
<td>Hip Flexion in standing position</td>
<td>Flexion in supine position</td>
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<tr>
<td>Knee Extension in sitting position</td>
<td>Can bear weight on leg</td>
<td></td>
<td></td>
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<tr>
<td>Foot Withdrawal synergy</td>
<td>Independent dorsiflexion</td>
<td></td>
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<tr>
<td>Sensory  2 points</td>
<td></td>
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<tr>
<td>Hand 2-point discrimination less than 5 mm</td>
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<tr>
<td>Foot Position sense in hallux</td>
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<tr>
<td>Mental function  2 points (6 points)</td>
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<tr>
<td>Hand &quot;Draw-a-man&quot;</td>
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<tr>
<td>Foot Jigsaw man</td>
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<tr>
<td>Speech  3 points</td>
<td>Comprehension of simple commands</td>
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<tr>
<td>Adequate communication of needs</td>
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<tr>
<td>Normal speech</td>
<td>Stroke, Vol. 3, November-December 1972</td>
<td>703</td>
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of abduction and external rotation. At the elbow 3 points—1 for the first trace of elbow flexion in the synergic flexion movement of the arm, 2 for ability to touch the mouth with the hand, 3 for good power of extension and flexion of the elbow. For hand and finger movement 3 points—1 for active grip along with the flexion synergy, 2 for active extension as well as flexion of the fingers and wrist, 3 for return of independent finger movements. In the leg, 3 points for hip movement—1 for ability to flex the hip in the sitting position, 2 for ability to raise the leg from the bed in the lying position, 3 for sufficient abduction at the hip to prevent Trendelenburg sign on lifting the unaffected leg in the standing position. At the knee, 3 points—1 for active extension of the knee in the sitting position, 2 for ability to bear weight on the leg without hyperextending the knee, 3 for normal knee flexion and extension. At the foot—1 point for contraction of tibialis anterior, along with general flexion synergy of the leg, 2 for ability to actively dorsiflex the foot with the heel on the ground, 3 for contraction of the peronei sufficient to prevent the foot going into inversion when dorsiflexed. Two points were given for sensory function—1 for 2-point discrimination on the index finger at a threshold of 5 mm or less, and 1 for passive movement perception in the great toe on the affected side. Two points were given for higher mental function—1 for body image testing with the “draw a man” test, or jigsaw puzzle man, and 1 for constructional ability with the Koh block test. Three points were given for speech—1 for comprehension of simple commands, 2 for fluency and 3 for normal speech. Spasticity was graded 2 in arms and legs. Separate functional classification was graded up to a total of 15, as follows (table 2).

Transferring: 1 point for ability to transfer from bed to chair with assistance, 2 for same transfer without assistance, and 3 for complete safety in transferring.

Wheelchair management: 1 point.

Walking: 1 point for ability to walk between parallel bars with assistance, 2 for ability to walk with tripod cane with assistance of one person, 3 for ability to walk with cane independently, and 4 for gait with even stride, with or without short leg brace.

Climbing stairs: 1 point for ability to climb four stairs with assistance of one person, and 2 points for ability to climb with aid of rail only.

Dressing: 1 point for ability to put on upper garment, 2 for upper and lower garment, and 3 for normal dressing, including fastenings.

Toilet: 1 point for continence of urine, 2 points for ability to manage washing and toilet activities independently.

These were assessed as soon as the patient was first seen, usually within a day or two of the stroke. Some of the tests, such as the mental function, could not be done at this early stage. All the tests were then repeated weekly, from the first week of transfer to the Manitoba Rehabilitation Hospital until the time of discharge, and then follow-up visits up to a year after the stroke. The relative weighting of the various sections could certainly have been done differently. For instance, we have recently put more stress on mental function and its recovery. It has proved simple to score the Koh block test out of four, with four sets of patterns of increasing difficulty. A much more sensitive and satisfactory grading of speech has been undertaken with the aid of a Porch Index of Communicative Ability.2

Results

Only five patients in this series failed to improve as much as 5 points on the above scale and made no significant neurological recovery. The other 34 all made some recovery, the most striking being from a score
RECOVERY AFTER HEMIPLEGIA

![Mean time course of recovery in patients studied.](image)

Mean time course of recovery in patients studied.

Of 2 on the day after the stroke to a score of 24 by the seventh week. Neurological recovery may occur at various rates (see fig. 2). Owing to the manner of selection of these patients, those with very rapid recovery (fig. 3) (transient ischemic attacks) are not included in this series, and in those who improved at all, recovery was still proceeding at the fourth week (fig. 4). Even with a fairly simple numerical scale of this type, there is always some variability in assessment, and 80% of the final score seems a suitable measure of the end point of neurological recovery. Such neurological recovery (80%) took from three to seven weeks with a mean of six weeks. There was no significant difference in rates between right and left hemiplegics or between those with aphasia and those without. The longest time to reach 80% of recovery took 14 weeks (fig. 5), and little neurological improvement occurred after 12 weeks from the onset of an ischemic brain lesion. Of 30 patients followed for more than 20 weeks, only one showed improvement of more than two points between the fifteenth and twentieth weeks.

Recovery of movement in the lower limb is one of the most functionally important processes in rehabilitation of the hemiplegic. In 28 of the patients some movement was beginning to return by four weeks, but two patients still had total lower limb paralysis at the sixth week and yet regained useful movement before the twelfth. On this type of numerical assessment, motor recovery in the leg accounts for just under half the total number of points on the recovery score. As is well known, recovery in the upper limb is much less complete in most cases of cerebral thrombosis.

In 11 patients there was no recovery at all of upper limb movement, and a further 10 had not more than 2 points recovery, which amounts to a weak flexion and abduction synergy of the shoulder and elbow alone. In the remaining 18 patients, recovery of a more useful degree took place, but only nine reached a figure of 6 points or more, and therefore had function sufficient for a useful assisting limb. One of the latter did not start to show any movement at all until the seventh week after the stroke. As in the lower limb, recovery was more or less complete by the twelfth week, and little neurological improvement on this scale took place after that time.

Recovery of sensation may also take place during the second and third month. Five
patients recovered a 2-point discrimination in the fingers of the affected hand, and six patients recovered position sense in the affected hallux. This recovery took place between the third and ninth weeks. In patients who are confused or have depressed consciousness in the postictal period, recovery of alertness is very striking during the second and third weeks. At this stage, however, proper testing of mental function is usually impossible. However, later recovery of mental function also may be seen during the second month. Five patients showed striking improvement in ability to complete the Kob block test when this was administered serially. Such improvement was noted as early as the fifth week and as late as the thirteenth week in one patient. Of
RECOVERY AFTER HEMIPLEGIA

the 13 patients tested with the 4-point Koh block test, eight were producing normal scores in the first test (four right and four left hemiplegics); two right hemiplegics were unable to complete the fourth and most difficult pattern and at later testing could complete them all. One right hemiplegic was completely unable to do any of the patterns at the fifth week, and at the seventh week had improved so that he could complete a perfect score. Two other patients made a partial recovery. Five patients also improved on the body image test (fig. 6) between the fifth and thirteenth weeks. In some patients, a complete constructional apraxia will change into a unilateral body image impairment and in others (fig. 7), neglect of the affected side of space will become rectified, even though hemianopia does not recover. Only two patients showed any change in their visual fields. They improved from a tendency to suppress the affected half fields to an apparently normal field of vision. This occurred on one occasion at the third week and in the other patient at the fourth.

Using the simple 3-point speech assessment, 14 of 19 aphasics showed some recovery of speech, expression and comprehension between the first and twelfth weeks. Six patients had serial tests with the Porch Index of Communicative Ability and all showed improvements ranging from an 11% to a 75% gain over their initial score. This improvement was continuing to take place as late as the twentieth week in one case.

Spasticity

Only one of these patients presented spasticity during the first week. By the eighth week (fig. 8), all but three showed some degree of spasticity and only two of the 39 patients never developed clinical spasticity (fig. 8). The degree of spasticity increased rapidly, particularly between the fourth and the seventh week, and then continued to mount up to the twentieth week from onset. Some patients showed fairly early onset of spasticity which then declined as functional recovery occurred.

All patients had a very poor functional score on admission to the Manitoba Rehabilitation Hospital. The average rate of recovery is shown in figure 2. Functional recovery generally occurred in the same time sequence as neurological recovery, but some patients were able to make reasonable functional rehabilitation with very little active movement of the lower limb and none of the upper. Three patients showed a decline probably related to ment after discharge from hospital, perhaps due to improved motivation. Two other patients showed a decline probably related to poor home environment. Patients with a functional score of less than 8 generally required heavy assistance, and most had to be transferred to nursing homes. On this scale, a functional score below 12 usually means that considerable assistance in the home is necessary.

The Mechanism of Recovery After Cerebral Infarction

Return of functional ability after hemiplegia depends to a considerable extent on neurological recovery in the previously paralyzed limbs. It also depends on the patient's motivation, on
the skill of those involved in retraining the hemiplegic, and on the home circumstances. Neurological recovery, on the other hand, appears to depend mainly on the intrinsic cerebral and circulatory processes which cannot as yet be much influenced by medical or physical treatment.

The first mechanism is probably return of circulation of ischemic areas. Studies of regional cerebral circulation by Lassen and others\(^8\) has brought back into prominence the important circulatory effects of localized obstruction of the cerebral circulation. It is clear that vigorous reactive hyperemia occurs all around the territory of the obstructed vessel and will tend to restore circulation to at least some areas with blood supply cut off by the immediate event. It is well known that neurons cannot survive a prolonged period of severe ischemia. Clinical observation and experimental studies suggest that neurons die after about five minutes' loss of total blood supply. There is considerably less work on the effects of subtotal ischemia and on the recovery pattern of neurons damaged in this way, but not to the point of death. Miller and Myers\(^4\) have described how the nervous system of monkeys can withstand up to 20 minutes' total arrest of the circulation if the cardiopulmonary system and the acid-base balance are returned promptly to normal after this time. At the extreme limits compatible with any recovery, the
RECOVERY AFTER HEMIPLEGIA

animals remained comatose for one or two
days and exhibited neurological signs for as
long as a month following the circulatory
arrest. A similar process is seen after cardiac
arrest in man. Fox reports recovery still
occurring at 25 days, and possibly for up to
three months. In cases observed personally,
recovery of mental function appears to contin-
ue at least as long as a month.

In the classical case of transient cerebral
ischemia, recovery occurs within 24 hours, and
it seems clear that actual infarction of brain
substance has not occurred. When infarction
does occur, it seems reasonable to suppose that
the infarct is surrounded by a variable area of
ischemia, which may be brought back to a
functional status by return of the circulation. It
seems unlikely, however, that recovery of the
circulation could be responsible for continuing
improvement in neurological function as late as
the tenth or twelfth week following the initial
episode.

The second obvious mechanism of recov-
ery is transfer of function from damaged
neurons to others. Mooney, for instance,
states that "spontaneous recovery must imply a
recruitment of neurons into new job classifica-
tions." Kinsbourne has shown that recovery of
speech in the hemiplegic may depend to a
considerable extent on use of the nondominant
hemisphere. Injection of Amytal into the right
carotid artery may abolish speech in the right
hemiplegic who is partially recovered from
aphasia. Return of speech has also been
described after dominant-hemispherectomy as
in the cases of Hillier and of Smith; both
these patients had been previously operated on
for tumor and both had postoperative aphasia
with partial recovery. After hemispherectomy,
speech improved up to the third week in one
case, and up to the fifth month in the other.
Hemispherectomy in the adult is usually
followed by return of movement in the lower
limb sufficient to permit walking, and this
generally takes place in the first one to three
months (Krynauw, Austin and Grant).

These findings suggest that at least part of the
recovery of the lower limbs after hemiplegia
may be due to intact mechanisms in the brain
stem, the basal ganglia and the opposite
hemisphere. After removal of one cerebral
hemisphere, the upper limb generally does not
recover, a situation comparable to many cases
of cerebral thrombosis.

A third mechanism of recovery is the
resolution of cerebral edema. Edema is
probably the cause of depressed consciousness
during the first week after a large infarct. How
much it contributes to contralateral paralysis is
uncertain. Serial angiograms and the results of
isotope brain scan suggest that edema is largely
resolved by four weeks.

It is generally assumed that neurons
cannot regenerate in the central nervous
system. Some recent reports suggest that this is
not entirely true, and that small terminal
dendrites can regenerate, sprout and form new
functional connections following central ner-
vous system damage. Rose et al. have shown
this following localized radiation damage of the
cortex and Wall and Egger have demonstrated
this after trauma to the nucleus cuneatus.

Conclusions
It is possible that retraining of the patient may
encourage the process of transfer of function to
undamaged areas of the nervous system but, in
general, physical treatment would seem to have
little effect on any of the above mechanisms.
The patient with a severe hemiplegia who is
going to have a permanent disability does need
retraining, but the most important medical
effects of rehabilitation therapy are in the
prevention of complications. In addition to the
obvious complications produced by recumben-
ty, there are the two major enemies of the
hemiplegic, despair and dependency.

It is hoped that further studies on the
recovery of cerebral function following circula-
tory arrest will clarify the importance of
circulatory recovery in hemiplegia. Careful
follow-up after the removal of brain tissue for
nonprogressive disease will give additional
information on the processes of transfer of
function within the nervous system. It could
then be possible that treatment aimed at these
major processes of recovery after hemiplegia
might have some beneficial results.

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