Original article:

The observational study of course of recovery of reflexes in spinal cord injury patients

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Abstract:

Introduction: The observational study of the course of recovery of reflexes in spinal cord injury patients.

Objectives: To study the normal pattern of recovery of reflexes in spinal cord injury patients and to study in how many days the reflexes recover and which reflex recovers first.

Methodology: Thirty five patients admitted in general municipal hospitals after spinal cord injury were examined over a period from the day of admission till recovery of following reflexes as mentioned below. These patients were observed for the following reflexes; Bulbocavernous (BC), Cremasteric (CRM), Ankle jerk (AJ), Knee jerk (KJ), and Plantar reflex for continuous 7 days a week and 4-5 weeks duration.

Results: Thirty five subjects examined of which 18 complete and 17 incomplete transection of spinal cord patients are included. In our study Bulbocavernous reflex appeared first followed by cremaster then ankle jerk and knee jerk along with Babinski’s sign. In incomplete and complete transection there was no difference in the recovery pattern. Chi-square test was applied to study the statistical significance between superficial and deep reflexes and between complete and incomplete injury patients.

Conclusions: Findings of our study conclude that the superficial reflexes recover first within 2-3 days and they follow the same pattern in both complete and incomplete. The deep reflexes then recover within 3-4 weeks and follow the same pattern in both complete and incomplete patients. The Babinski sign was observed within 3-4 weeks and was observed slightly earlier in incomplete injury patients.

Keywords: Spinal cord injury, Monosynaptic deep tendon reflexs, Polysynaptic superficial reflexes.

Introduction:

The motor control systems, because of their anatomy, are susceptible to damage. The nature of patients motor deficit often allows the neurologist to diagnose the site of neural damage. When higher centers of the motor system are damaged by stroke, trauma, or demyelinating disease, the signs and symptoms are distinctly different from those caused by lower damage. Complete transection of spinal cord leads to profound paralysis below the level of lesion. This is called as paraplegia, when only both legs are selectively affected and quadriplegia when the legs, trunk, and arms are affected. For a few days after an acute injury, there is Areflexia and decreased muscle tone, a condition called as Spinal Shock. Spinal shock was first defined by Whytt in 1750 as a loss of sensations accompanied by motor paralysis, with gradual recovery of reflexes following a spinal cord injury, most often a complete transection. When spinal cord is transected, essentially all cord functions, including reflexes, immediately become depressed to the point of total silence, a reaction...
called spinal shock. The reason for this is that normal activity of the cord neurons depends to a great extent on continual tonal excitation by the discharge of the nerve fibers entering the cord from higher centers, transmitted through corticospinal, vestibulospinal, rubrospinal, tectospinal, oligospinal tracts \(^{(2)}\).

According to Holdsworth, as segmental reflexes recover (i.e. as spinal shock resolves) from spinal cord injury, and no accompanying recovery of voluntary movement is observed, then the prognosis for ultimate functional recovery is extremely poor \(^{(4)}\).

According to Harrison prospect of recovery from acute SPI fades after approximately 6 months \(^{(3)}\).

Although Guttman stated that the recovery of reflexes was quite variable in man, he indicated they followed a pattern and the first reflexes to return was the Bulbocavernous reflex (BC) and the anal cutaneous. Guttman suggested, however, there was a need to study the reflex recovery serially and systematically in order to better understand the pattern of recovery from spinal shock and its prognostic significance. \(^{(3)}\).

All the skeletal muscle reflexes integrated in the spinal cord are blocked during the initial stages of shock. In human beings, 2 weeks to several months are required for these reflexes to return to normal. However some reflexes may eventually become hyper excitable, particularly if a few facilitatory pathways remain intact between brain and the cord while the remainder of the spinal cord is transected. The first reflexes to return are the stretch reflexes, followed in order by more complex reflexes like flexor reflexes, postural antigravity reflexes \(^{(2)}\).

However, does recovery of reflexes often follows this pattern; therefore in this review we revisit spinal shock from a fresh perspective in an attempt to explain clinical observations of spinal shock.

**Aim:**
To observe and study the pattern of recovery of reflexes in Spinal cord injury patients.

**Objectives:**
The purpose of the study is to observe and study the pattern of recovery of reflexes following Spinal cord injury in order to determine which reflex recovers first.

**Materials and methods:**

**Research site:**
The study was conducted in the inpatient department of neurology and orthopedics of KEM Hospital and NAIR Hospital, Mumbai, over a period of 18 months.

**Ethical consideration:**
Approval to conduct the study was obtained from the committee for academic research and ethics (CARE) of the hospital where the data was collected. Patients were informed about the study, the examination involved and the usefulness of its outcome; assured their confidentiality and anonymity would be maintained, informed they could withdraw any time, without repercussions, and, given an opportunity to ask questions by them or by their relatives. Those who consented to participate in the study were asked to sign or give their thumb impression on the consent form.

Thirty five patients admitted in general municipal hospitals after spinal cord injury were examined over a period from the day of admission till recovery of following reflexes as mentioned below. I have taken history and done the examination once the neurologist, orthopedic surgeon and resident doctors have examined and diagnosed the patient. I have checked for the following reflexes as mentioned below.

1) Bulbocavernous reflex
2) Cremasteric reflex
3) Ankle jerk
4) Knee jerk
5) Normal plantar reflex
The pattern of recovery of mentioned reflexes in these 35 patients was then studied.

**Study procedure:**

1) Proper consent of the volunteer patients was obtained before the procedure.
2) In case of severely ill patients, consent from their relatives was obtained.
3) History taking, general examination and systemic examination were done before the procedure.
4) Reflexes examined were
   i) Bulbocavernous reflex
   ii) Cremasteric reflex
   iii) Ankle jerk reflex, also known as the Achilles reflex
   iv) Knee jerk, also known as patellar reflex
   v) Normal plantar reflex

**Results and observations:**

The study procedure was carried out in Spinal Cord injury patients admitted to the general municipal hospitals. The sample size of the study was 35 which was calculated by census method i.e. we included each and every patient who fulfills our inclusion and exclusion criteria.

All the patients and their relatives were explained about the study procedure and then the patients were examined.

The data obtained was entered using the Microsoft Excel 2007. Statistical analysis was done using the SPSS v16.0 software.

The statistical test used were (as per the requirement of the data)

- Chi-square test was used to do the comparison between superficial reflexes in complete and incomplete spinal cord injury patients.
- Chi-square test was used to do the comparison between deep reflexes in complete and incomplete spinal cord injury patients.
- Comparison was done between monosynaptic and polysynaptic reflexes in complete and incomplete spinal cord injury patients.
- Comparison was done for recovery of Babinski sign between complete and incomplete spinal cord injury patients.

The P value < 0.05 was considered statistically significant.

According to this study the no of days taken for the recovery of various reflexes studied by is as follows.

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Average Day of Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulbocavernosus</td>
<td>2</td>
</tr>
<tr>
<td>Cremasteric</td>
<td>3</td>
</tr>
<tr>
<td>Ankle jerk</td>
<td>18</td>
</tr>
<tr>
<td>Babinski sign</td>
<td>21</td>
</tr>
<tr>
<td>Knee jerk</td>
<td>26</td>
</tr>
</tbody>
</table>
Comparison of Superficial versus Deep Reflexes (excluding Babinski sign)

![Graph showing comparison of superficial and deep reflexes excluding Babinski sign.]

Chi square Test, P value <0.05 (Significant)

Comparison of Superficial versus Deep Reflexes (including Babinski sign)

![Graph showing comparison of superficial and deep reflexes including Babinski sign.]

Chi square Test, P value <0.05 (Significant)

**Discussion:**

Thirty-five subjects were examined in the study from their day of admission to the general municipal hospitals. They had been diagnosed with Spinal Cord injury, which had a clinical evidence of upper motor neuron lesion. Of these 35 patients 18 were complete Spinal cord injury patients and 17 were incomplete Spinal cord injury patients.

1. In these 35 patients the earliest reflex to return was the bulbocavernosus which appeared within 2 days followed by Cremasteric which appeared in 2-3 days, then ankle reflex which appeared in 21-22 days, followed by plantar reflex (Babinski sign) and finally...
the knee jerk which recovered within 25-28 days. This pattern of recovery of reflexes was same for both complete and incomplete spinal cord injury patients. However the recovery of deep tendon reflexes was hyperexcitable (hyper-reflexia). To understand this pattern of reflex recovery, we must understand first how the neurons regenerate after spinal cord injury, and what is the neuronal process that mediates spinal hyperreflexia after spinal cord is injured.

The Wallerian degeneration occurs after axonal injury in both the peripheral nervous system (PNS) and central nervous system (CNS). It occurs in the axon stump distal to a site of injury and usually begins within 24–36 hours of a lesion. Prior to degeneration, distal axon stumps tend to remain electrically excitable. After injury, the axonal skeleton disintegrates, and the axonal membrane breaks apart. The axonal degeneration is followed by degradation of the myelin sheath and infiltration by macrophages. The macrophages, accompanied by Schwann cells, serve to clear the debris from the degeneration.

The nerve fiber’s neurolemma does not degenerate and remains as a hollow tube. Within 4 days of the injury, the distal end of the portion of the nerve fiber proximal to the lesion sends out sprouts towards those tubes and these sprouts are attracted by growth factors produced by Schwann cells in the tubes. If a sprout reaches the tube, it grows into it and advances about 1 mm per day, eventually reaching and reinnervating the target tissue. If the sprouts cannot reach the tube, for instance because the gap is too wide or scar tissue has formed, surgery can help to guide the sprouts into the tubes. This regeneration is much slower in the spinal cord than in PNS. The crucial difference is that in the CNS, including in the spinal cord, myelin sheaths are produced by oligodendrocytes and not by Schwann cells.

In the cell body of the neuron, changes start within 48 hours and continue for 15-20 days. Nissls granule disintegrates and dissolve (chromatolysis). Golgi apparatus, mitochondria and neurofibrils are fragmented and eventually disappear. Cell body draws in more fluid enlarges and becomes spherical. Nucleus is displaced to the periphery. Sometimes it is extruded out of the cell, in which case the neuron atrophies and finally disappears completely.

In the axon proximal to injury changes occur, only upto the first or second node near the injury and are similar to changes in distal part of axon. (Retrograde degeneration)

Regeneration starts within four days of injury but becomes more active after 30 days and may take several months to one year for complete recovery.

**Proposed physiological mechanism for spinal hyperreflexia during the stage of recovery:**

1. Denervation supersensitivity

Denervation supersensitivity likely plays a role in initial reflex re-emergence. Such supersensitivity to neurotransmitters is well known in denervated neurons of both the peripheral and central nervous system. Proposed mechanism for Denervation supersensitivity includes: (1) reduced excitatory neurotransmitter uptake, (2) increased synthesis and insertion of receptors into postsynaptic membrane, (3) decreased removal and degradation of receptors.

2. New synapse growth and sprouting of preserved spinal tracts.

Below a spinal cord lesion, synaptic endings from injured neurons degenerate over days and are then replaced by terminal sprouting neurons below the lesion. This neuronal sprouting may originate from both spinal interneurons from primary segmental
afferents and from preserved spinal tracts. The postulated signals for new synapse growth is the increase in Neurotrophins caudal to spinal cord injury. These Neurotrophins released, bind to the receptors present at the nerve endings. They are internalized by endocytosis and then transported to the cell body by retrograde axoplasmic flow. After reaching the cell bodies, the neurotrophins foster the production of proteins associated with neuronal development, growth and survival.

**Conclusion:**
The pattern of recovery of reflexes showed that superficial reflexes recover first then the deep reflexes; however the Babinski sign recovered along with the deep reflexes.

Comparison of superficial reflexes in both complete and incomplete injury patients was not statistically significant, i.e. they recovered in the same time period in both the cases.

Comparison of deep reflexes in both complete and incomplete injury patients was not statistically significant, i.e. they recovered in the same time period in both the cases.

The recovery of pathological reflex (Babinski sign) in both complete and incomplete was statistically significant.

**References:**
2) Hall M. Synopsis of the diastaltic nervous system: or the system of the spinal marrow, and its reflex arcs; as the nervous agent in all the functions of ingestion and of egestion in the animal economy. London: Mallett J., 1850;
3) Harrison’s principle of internal medicine 18th edition Volume 2 pages no 2596.