Postoperative C5 Palsy: Conjectured Causes and Effective Countermeasures

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Abstract:

Postoperative C5 palsy (C5 palsy) is defined as *de novo* or aggravating muscle weakness mainly at the C5 region with slight or no sensory disturbance after cervical spine surgery. The features of C5 palsy are as follows: 1) one-half of patients are accompanied by sensory disturbance or intolerable pain at the C5 region; 2) 92% of patients have hemilateral palsy; 3) almost all palsy occurs within a week after surgery; 4) the incidence is almost the same between the anterior and posterior approaches to the cervical spine; 5) the prognosis is relatively good even in patients with severe muscle weakness. Even now, the precise causes of C5 palsy have not yet been revealed. From the viewpoint of the kinds of nerve tissue involved, the uncertain causes of C5 palsy are divided into two theories: 1) the segmental spinal cord disorder theory and 2) the nerve root injury theory. In the former, the segmental spinal cord, particularly the anterior horn cells, is thought to be chemically damaged because of preoperative ischemia and/or the aggression of reactive oxygen during postoperative reperfusion. By contrast, in the latter, the anterior rootlet and/or nerve root are believed to be mechanically damaged because of compression force and/or distraction force. In this theory, the features of C5 palsy can be well explained from anatomical viewpoints. Additionally, various countermeasures have been proposed, such as the intermittent relaxation of the tension of the hooks to the multifidus muscles during surgery; prophylactic foraminotomy to decompress C5 nerve root; prevention of excessive posterior shift of the spinal cord, which may cause the tethering effect of the nerve root; and prevention of excessive postoperative lordotic alignment of the cervical spine. These countermeasures have been proved effective, and may support the nerve root injury theory as the main conjectured theory on the causes of C5 palsy.

Keywords:

postoperative C5 palsy, cause, segmental spinal cord disorder, nerve root injury, countermeasure

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Summary of Postoperative C5 Palsy (C5 Palsy)

C5 palsy is defined as *de novo* or aggravating muscle weakness mainly at the C5 region with slight or no sensory disturbance after cervical spine surgery. The paresis of other cervical nerve regions (C6, C7, C8) has been actually reported in isolation or combination; however, these incidences are lower than those of the C5 region. Therefore, muscle weakness appearing after cervical spine surgery is generally called "postoperative C5 palsy (C5 palsy)."

According to Sakaura et al.[1] who reviewed 343 cases in the literature, the features of C5 palsy are as follows: 1) one-half of patients are accompanied by sensory disturbance or intolerable pain at the C5 region; 2) 92% of patients have hemilateral palsy; 3) almost all palsy appears within a week after surgery; 4) the incidence is almost the same between the anterior and posterior approaches to the cervical spine; 5) patients with severe muscle weakness have relatively good prognosis, but have longer recovery times.

Scoville[2] and Stoops[3] reported neurological complications after cervical laminectomy and foraminotomy in 1961 and 1962, respectively. Scoville[2] referred to the tethering effect of the spinal root sleeves after decompression and reported one case in which the operative contusion of the left C5 nerve root temporarily increased the weakness of the left arm.

In Japan, in 1973, the idea of cervical laminoplasty (CL) was first proposed by Oyama et al.[4] under the name of "expansive lamina-Z-plasty." Thereafter, various methods of CL were developed and performed in many hospitals, thus re-

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placing previous laminectomy. Given the increasing number of patients who had undergone CL, C5 palsy and the tethering effect of the nerve root related to it have been gradually noticed by many surgeons and have been reported[5-9]. C5 palsy occurred in not only CL as a posterior approach but also in anterior decompression and fusion as an anterior approach[10].

In the beginning, the causes were thought to be directly related to surgical maneuvers, such as obvious contusion to the spinal cord or the nerve root during surgery[2,11], and the compression of the nerve roots by a dislocated transplanted strut bone after surgery[10]. However, it has been gradually revealed that there are many manifestations of C5 palsy with uncertain causes unrelated to surgical maneuvers. At present, the issues concerning C5 palsy are as follows: 1) Why does muscle weakness dominantly occur at the C5 region? 2) What are effective countermeasures to prevent C5 palsy even if there are many cases with uncertain causes? If some trial countermeasures appear to be effective in decreasing the incidence of C5 palsy, such countermeasures may be helpful in revealing the actual causes of C5 palsy.

Dissociated Motor Loss in the Upper Extremities

The primary features of C5 palsy are dominant muscle weakness with slight or no sensory disturbance. This neurologic condition is similar to a case with dissociated motor loss in the upper extremities in chronic compressive cervical myelopathy, which was reported by Keegan[12] in 1965.

The important point is that he demonstrated the recognition of the symptomatic separation of motor and sensory functions and estimated the causes from an anatomical viewpoint. He pointed out a bony prominence at the posterolateral corner of the vertebral body and called it a "high spot," and estimated that the anterior motor roots within the dural sac were compressed by this bony prominence, thus resulting in dissociated motor loss. Ota and Ono[13] reported that the Luschka joints significantly contributed to the compression of the anterior roots in autopsy series. Matsunaga et al.[14] performed posterior decompression and clinically proved that anterolateral osteophyte compression was predominantly responsible for dissociated motor loss. In contrast to this mechanical viewpoint, Yanagi et al.[15] and Ito et al.[16] proposed another idea that a circulatory disturbance to the anterior horn cell at the paramedian spinal cord may be the contributing factor. In any event, both the anatomical and pathological changes of the cervical spine, particularly at the C5 region, must be attended when considering the causes of postoperative C5 palsy.

Conjectured Causes of C5 Palsy

Even now, the precise causes of C5 palsy have not yet been revealed probably because multiple factors are related to its occurrence. The obvious causes of C5 palsy that are directly related to surgical maneuvers include nerve tissue contusion by an air-drill during surgery (can be observed by a surgeon), and the compression of the nerve roots by a dislocated transplanted strut bone (can be revealed by images). At present, the uncertain causes of C5 palsy are divided into the viewpoint of the time of onset and the kinds of nerve tissue involved.

During surgery, the spinal cord and/or the nerve root may be damaged by the unintentional compression of a retractor and/or the high friction heat of the tip of an air-drill[17]. However, the reason for the dominant impairment of the C5 region cannot be well explained.

According to Sakaura et al.[1], almost all C5 palsy appears immediately after and/or within a week after surgery. This means that there may be many causes of C5 palsy from the viewpoint of disease onset. One of the reasons of the late appearance of the palsy is the following: under a new circumstance in which cervical spine alignment is more or less changed after decompression surgery, the spinal cord and/or the nerve root may be distracted and/or compressed by adjacent anatomical structures such as the facet joint and the vertebral body, particularly when patients start rehabilitation upon regaining the ability to stand within a week after surgery.

Concerning the kinds of nerve tissue involved, there are two theories: 1) the segmental spinal cord disorder theory[18,19] and 2) the nerve root injury theory[9,20-31].

In the segmental spinal cord disorder theory, it is thought that nerve tissues, particularly the anterior horn cells, may be damaged because of ischemia before surgery and/or reperfusion after decompression. At the time of acute reperfusion, the nerve cells may be chemically damaged by reactive oxygen[18,19]. In this theory, the reason for the dominant impairment of a specific region, such as that of the hemilateral C5 motor function, cannot be explained. Capillary networks rising from terminal arterioles within the spinal cord are very rich, particularly in the gray matter[32,33]. The impairment of a limited region is thought to seldom occur after ischemia and reperfusion. Fundamentally, it is unclear whether reperfusion injury can actually occur after ischemia in the spinal cord. According to Tsuzuki et al.[34], the levels of division from the spinal cord are different between the anterior and posterior rootlets, and the difference is more apparent in the middle and upper cervical levels, such as C4 and C5. In C5 palsy, it is shown that one-half of patients are accompanied by sensory disturbance or intolerable pain at the C5 region. If the motor function and sensory function of the C5 region are simultaneously impaired within the spinal cord, a vertically-wide but restricted area must be damaged; however, this conflicting situation hardly occurs.

By contrast, in nerve root injury theory, C5 palsy is thought to be caused by mechanical compression and/or the distraction of the anterior rootlet and/or anterior nerve root[9,20-31]. From this theory, the features of C5 palsy can be well explained. These features include the following: sensory disturbance or intolerable pain at the C5 region, hemilateral palsy, late onset within a week after surgery, and

good prognosis.

Anatomical Investigation to Reveal the Causes of C5 Palsy

In the investigation of the causes of C5 palsy, the dominant impairment of the C5 region is the most important issue. The anatomical and pathophysiological features of the C5 region are probably related to this issue. Therefore, it is necessary to analyze the anatomical structures of the cervical spine, particularly the C5 region, by using cadavers.

From our anatomical analysis, we have concluded that this palsy is most likely caused by C5 nerve root compression and stretching near the exit of the foramen[24,25,35]. The essences of our analysis are as follows: 1) Among the cervical nerve roots composed of the brachial plexus, the distance between the division from the dura mater and the exit of the foramen is shortest at the C5 nerve root; therefore, the capacity for moving freely is smallest at the C5 nerve root. 2) The anterior rootlets run adjacent to the narrowest part of the foramen, that is, the tip of the superior facet joint, and they run caudal to the posterior rootles; therefore, the anterior rootlets of the cervical nerve tend to be stretched and compressed mechanically from the caudal side near the tip of the superior facet joint in the foramen. 3) The medial branches of the posterior rami come into contact with the lateral side of the facet joint column in the shortest distance, and the muscle branch of the medial branches are sent to the multifidus muscles.

According to Zhang et al.[36], the length of the posterior ramus proper is shortest in the C4 and C5 nerves. If the multifidus muscles are severely retracted laterally by hooks during posterior approach surgery, not only the medial branch of the posterior ramus but also the posterior ramus proper and the anterior rootlet are simultaneously stretched and compressed against adjacent prominent bone structures because the free movement is estimated to be significantly restricted in the C5 nerve root.

By using a trigonometric function, it can be proved that the traction ratio is the same between a small elevation in a shorter segment and a large elevation in a longer segment, that is, a shorter segment tends to be largely influenced even by small changes of the outer circumstances. Tsuzuki et al.[6] reported that a greater degree of anterior prominence of the superior facet joint leads to a narrower intervertebral foramen, and these conditions are mostly found at the C4 and C 5 levels. Therefore, the anterior rootlets are mostly stretched and compressed at the narrowed intervertebral foramen at the C4 and C5 levels. In patients with hypertrophied facet joints due to degenerative changes, these influences are thought to become larger. After decompression, the influence of the stretching and compression of the nerve root near the exit of the foramen is thought to become higher because of the gradual posterior shift of the spinal cord within the spinal canal, that is, the tethering effect[27,28,31]. In addition to this tethering effect within the spinal canal, the lateral displacement of the multifidus muscles due to postoperative swelling and compression of extradural hematoma outside the spinal canal may cause the stretching of the medial branches of the posterior rami.

Findings of Magnetic Resonance Imaging

There are some reports[18,22,37] that discuss the relationship between the postoperative paralysis in the upper extremity and high intensity area (HIA) in T2-weighted magnetic resonance imaging (MRI). HIA can be often observed in patients with chronic compressive myelopathy and may probably indicate cavity formation or necrosis in the gray matter. However, it has been revealed that the presence of HIA is not correlated with the severity of myelopathy or surgical results[38].

Chiba et al.[18] presented 15 patients with postoperative segmental motor paralysis that occurred mainly at the C5 region. All patients had HIA in the spinal cord, and 10 of them revealed correspondence between the level of the abnormal signal areas and the level of paralysis. As a result, it was concluded that a certain impairment in the gray matter of the spinal cord may play an important role in the development of postoperative segmental motor paralysis.

Sakaura et al.[37] reported a prospective study on segmental motor paralysis in 81 patients who underwent CL. The incidence of paralysis was 12.3 % (6.2% in proximal type, 2.5% in distal type, and 3.7% in diffuse type), and the linear HIA was significantly higher in paralytic segments than non-paralytic segments. However, motor paralysis did not occur in 75% of patients with linear HIA, and there were some cases in which paralysis occurred only hemilaterally even though bilateral HIA was observed at the same level.

Seichi et al.[22] reported that the incidence of HIA was 6.1% in 114 patients who underwent CL, and HIA had a strong relationship with distal and diffuse types of motor paresis. By contrast, no patient with postoperative C5 and C6 palsies showed abnormal HIA spreading; this finding supports the root impairment theory as the cause for the proximal type of palsy.

These reports show that there are limits to some extent in investigating the causes of C5 palsy from MRI findings because there are no procedures to directly prove it; therefore, only indirect evidence exists.

Direct Countermeasures to Prevent C5 Palsy

Even though it is unclear which theory is probable (segmental spinal cord disorder theory or nerve root injury theory), real countermeasures to prevent C5 palsy must be clinically proposed.

For segmental spinal cord disorder theory, the administration of neuroprotective agents such as antioxidant drugs and calcium-channel blockers was proposed. However, the effectiveness of these agents has never been reported.

By contrast, for nerve root injury theory, various effective

countermeasures have been proposed probably because this theory is based on mechanical factors; thus, various surgical interventions have been developed and tried.

We have recommended that the excessive lateral stretch of the multifidus muscles for extended periods during surgery must be avoided. During surgery via the posterior approach, the intermittent relaxation of the tension of the hooks to the muscles may be one solution[25,35]. We also recommend that the following procedures be performed as early as possible after surgery from the time of diagnosing C5 palsy: the patient's elbow should be flexed and the shoulder should be slightly abducted to decrease the distraction force to the C5 nerve root. This procedure can be easily performed by simply putting a pillow under the axilla with the patient lying in bed[39].

In CL combined with dekyphosis surgery, foraminotomy is added in advance because the C5 nerve may be compressed at the narrowed foramen by extension during the dekyphosis procedure[24].

There are some reports[20,23,26-29,31] that the tethering effect of the nerve root may be a significant risk factor for C5 palsy, in which the nerve is thought to be stretched and compressed at the intervertebral foramen due to the posterior shift of the spinal cord after decompression. As one of the procedures to prevent this mechanical effect, prophylactic combined foraminotomy[20,23,26,29] has been performed. Sasai et al.[20] reported significant differences in the incidence of C5 palsy in CL: 0% in patients with foraminotomy and 8.1% without foraminotomy. Komagata et al.[23] compared the incidence of C5 palsy in CL: 0.6% with foraminotomy and 4.0% without foraminotomy. Katsumi et al.[29] reported significant differences in the incidence of C5 palsy in CL: 1.4% with foraminotomy and 6.4% without foraminotomy. These reports showed the significant effectiveness of combined foraminotomy in preventing C5 palsy, although all instances of C5 palsy could not be prevented by foraminotomy only.

Another procedure is to avoid the excessive posterior shift of the spinal cord by limiting the angle of the opened lamina and the width of resection of the lamina. Zhang et al.[40] reported the procedure in which the open-door angle in CL is maintained between 15 and 30 degrees; this procedure decreased the incidence of C5 palsy. Uematsu et al.[9] reported that it is important to place the bony lateral gutter on the medial side of the zygapophysial joint and to keep the slope of the opened lamina within 60 degrees to prevent postoperative radiculopathy. Nori et al.[41] proposed cervical laminectomy with a width of not more than 2-3 mm wider than the spinal cord width to prevent the excessive posterior shift of the spinal cord. This reduced the incidence of C5 palsy from 9.2% to 1.2%.

In conclusion, previous reports indicated that mechanical distraction and/or compression force may likely be the main cause of C5 palsy. The tethering effect is the force that acts within the intervertebral foramen and spinal canal during and after surgery. The lateral stretch of the multifidus muscles by hooks is the force that acts outside the spinal canal during surgery. These forces tend to act mainly on the C5 region, which has the following special anatomical structures: 1) Among the cervical nerve roots composed of the brachial plexus, the distance between the division from the dura mater and the exit of the foramen is shortest at the C5 nerve root. 2) The intervertebral foramen tends to be narrower at the C4 and C5 levels in combination with the high incidence of anterior prominence of the superior facet joint. 3) The length of the posterior ramus proper is shortest in the C4 and C5 nerves. It can be thought that not only the medial branch of the posterior ramus but also the posterior ramus proper and the anterior rootlet of C5 are simultaneously stretched and compressed against adjacent prominent and/or narrowed bone structures because the free movement of these C5 nerve tissues is restricted, thus resulting in C5 palsy. Various effective countermeasures that deal with the nerve root may support the nerve root injury theory as the main conjectured theory on the causes of C5 palsy.

Conflicts of Interest: The authors declare that there are no relevant conflicts of interest.

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