Patterns of neurological deficits and recovery of postoperative C5 nerve palsy

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OBJECTIVE Paresis of the C5 nerve is a well-recognized complication of cervical spine surgery. Numerous studies have investigated its incidence and possible causes, but the specific pattern and character of neurological deficits, time course, and relationship to preoperative cord signal changes remain incompletely understood.

METHODS Records of patients undergoing cervical decompressive surgery for spondylosis, disc herniation, or ossification of the longitudinal ligament, including the C4–5 level, were reviewed from a 15-year period, identifying C5 palsy cases. Data collected included age, sex, diabetes and smoking statuses, body mass index, surgical levels, approach, presence of increased cord signal intensity, and modified Japanese Orthopaedic Association (mJOA) scores. Narrative descriptions of the patterns and findings on neurological examination were reviewed, and complications were noted. The minimum follow-up requirement for the study was 12 months.

RESULTS Of 642 patients who underwent cervical decompressive surgery, 18 developed C5 palsy (2.8%). The incidence was significantly lower following anterior surgery (6 of 441 [1.4%]) compared with that following cervical laminectomy and fusion (12 of 201 [6.0%]) ($p < 0.001$). There were 10 men and 8 women whose mean age was 66.7 years (range 54–76 years). The mean preoperative mJOA score of 11.4 improved to 15.6 at the latest follow-up examination. There were no differences between those with and without C5 palsy with regard to sex, age, number of levels treated, or pre- or postoperative mJOA score. Fifteen patients with palsy (83%) had signal changes/myelomalacia on preoperative T2-weighted imaging, compared with 436 of 624 (70%) patients without palsy; however, looking specifically at the C4–5 level, signal change/myelomalacia was present in 12 of 18 (67%) patients with C5 palsy, significantly higher than in the 149 of 624 (24%) patients without palsy ($p < 0.00003$). Paresis was unilateral in 16 (89%) and bilateral in 2 (11%) patients. All had deltoid weakness, but 15 (83%) exhibited new biceps weakness, 8 (44%) had triceps weakness, and 2 (11%) had hand intrinsic muscle weakness. The mean time until onset of palsy was 4.6 days (range 2–14 days). Two patients (11%) complained of shoulder pain preceding weakness; 3 patients (17%) had sensory loss. Recovery to grade 4/5 deltoid strength occurred in 89% of the patients. No patient had intraoperative loss of somatosensory or motor evoked potentials or abnormal intraoperative C5 electromyography activity.

CONCLUSIONS Postoperative C5 nerve root dysfunction appears in a delayed fashion, is predominantly a motor deficit, and weakness is frequently appreciated in the biceps and triceps muscles in addition to the deltoid muscle. Preoperative cord signal change/myelomalacia at C4–5 was a significant risk factor. No patient had a detectable deficit in the immediate postoperative period or changes in intraoperative neuromonitoring status. Neurological recovery to at least that of grade 4/5 occurred in nearly 90% of the patients.

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KEYWORDS C5 nerve palsy; cervical fusion; complications; myelomalacia; myelopathy; T2 cord change; deltoid weakness

PARESIS of the C5 nerve is a well-recognized complication of decompressive cervical spine surgery involving the C4–5 level and is known to occur following both anterior and posterior approaches.^{1–4} The syndrome is characterized by predominantly motor deficits

appearing in a delayed fashion in the days or weeks following surgery. Numerous studies have investigated its incidence and possible causes, but most do not provide detail about the specific pattern of neurological deficits seen and the time course and character of neurological recovery. In

ABBREVIATIONS ACDF = anterior cervical discectomy and fusion; BMI = body mass index; CLF = cervical laminectomy and fusion; EMG = electromyography; MEP = motor evoked potential; mJOA = modified Japanese Orthopaedic Association; PEEK = polyetheretherketone; SSEP = somatosensory evoked potential.

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this investigation, we assessed the preoperative characteristics of C5 palsy that developed in patients after surgery, looking at the incidence and factors in patient history or imaging findings associated with the complication. Particular focus, however, was directed at carefully reviewing the specific details of the neurological examination and narrative descriptions of the symptoms of these patients, both in the hospital progress notes and office records.

Methods

After institutional review board approval, we conducted a retrospective review of records of consecutive patients who underwent decompressive cervical spine surgery for spondylosis, disc herniation, or ossification of the posterior longitudinal ligament, which included the C4–5 level, treated by a single surgeon over a 15-year period, and we identified cases complicated by C5 nerve palsy. Patients with acute spinal cord injury from trauma and those with active spinal infection/discitis were excluded. Data collected included patient age, sex, diabetes and smoking status, body mass index (BMI), surgical levels, and approach; in each patient, a pre- and postoperative modified Japanese Orthopaedic Association (mJOA) score was calculated.

Surgery

Cervical laminectomy and fusion (CLF) was the surgical procedure used for all posterior surgeries, employing a previously published decompression technique⁵ in which a rough 6-mm extra-coarse ball diamond burr (Midas Rex, Medtronic Power Tools) is used to thin the lamina at the junction of the medial facet, layer by layer, until the inner cortex was just traversed. The ligamentous attachments were then cut with Metzenbaum scissors, while the “floating” laminae were elevated with toothed forceps. All patients had internal fixation with cervical lateral mass screw/rod fixation. Pedicle screws were used for C7 fixation in some patients and in all those in whom fixation extended to the upper thoracic spine. In cases of anterior cervical discectomy, we used a machined allograft block graft or polyetheretherketone (PEEK) cages filled with demineralized bone graft; in cases involving corpectomy, we used PEEK cages filled with local bone. Plate fixation was performed in all anterior-approach cases. Intraoperative neuromonitoring was used in all surgeries and included somatosensory (SSEP) and motor evoked potential (MEP) monitoring and free-running electromyography. We did not routinely use perioperative intravenous steroids.

Postoperative Assessment

Narrative descriptions of the character and course of the patient's neurological examination were reviewed from the hospital progress notes and all office follow-up visits. Any surgical complications were noted. The minimum follow-up duration was 12 months. C5 palsy was defined as new weakness of one or more grades on manual muscle testing in the deltoid muscle without a worsening of myelopathic symptoms. A short course of steroids was administered to all patients with C5 palsy, typically a standard course of 4-mg oral methylprednisolone tablets tapering from 24 mg to 0 mg over 6 days. For those still in the inpatient setting,

we used 4-mg intravenous infusions of dexamethasone every 6 hours for 3 days. All patients were ordered to undergo physical therapy that included range of motion exercises intended to retard the development of a frozen shoulder. In the initial years of the study period, all patients underwent MRI following the appearance of the deficit; in subsequent years, however, we did not image patients with a C5 palsy unless they had associated deterioration of underlying myelopathic symptoms.

Statistical Analysis

Statistical analysis was done using commercially available software (GraphPad Prism 4, GraphPad Software) using the Student t-test, Mann-Whitney U-test, Fisher's exact test, or the chi-square test, with $p < 0.05$ considered statistically significant.

Results

A total of 642 patients met the inclusion criteria, 18 of whom developed C5 palsy (2.8%). Data describing clinical characteristics of the patients suffering from C5 palsy and comparisons of those with and without C5 palsy can be viewed in Tables 1 and 2. The C5 palsy complication rate was significantly lower in patients who underwent anterior surgery, occurring in 6 of 441 cases (1.4%) following the anterior approach and in all anterior cervical discectomy and fusion (ACDF) cases, compared with 12 of 201 (6.0%) cases following cervical laminectomy and fusion ($p < 0.001$). There were 10 men and 8 women, whose mean age was 66.7 years (range 54–76 years); their mean preoperative mJOA score of 11.4 improved to 15.6 at the latest follow-up visit. Compared with those not developing a C5 palsy, the patients with palsy did not differ significantly with respect to sex, age, number of levels treated, or pre- and postoperative mJOA score. In the patients who developed palsy, there were no non-C5-palsy-related neurological complications or wound infections, but one patient developed a deep vein thrombosis and one patient suffered anginal chest pain that required an urgent stenting procedure. Fifteen of these 18 patients (83%) had signal change on preoperative T2-weighted imaging/myelomalacia, compared with 436 of 624 (70%) patients without palsy; however, looking specifically at signal change/myelomalacia at the C4–5 level, it was present in 12 of 18 (67%) with C5 palsy, a significantly higher rate than that seen in the 149 of 624 (24%) without palsy ($p < 0.00003$). Paresis was unilateral in 16 (89%) and bilateral in 2 (11%) of the patients with palsy. All patients with C5 palsy presented with weakness of the deltoid muscle (mean 1.33 on the 0–5 manual muscle testing scale), but other muscles were affected as well, with 15 patients (83%) also demonstrating new weakness in the biceps (a mean score of 3.08), 8 (44%) demonstrating new weakness in the triceps (a mean score of 3.83), and 2 patients (11%) demonstrating new weakness in the hand intrinsic muscles (a mean score of 3.5). The mean time until onset of motor deficits was 4.6 days (range 2–14 days). Two patients (11%) complained of shoulder pain that preceded the appearance of weakness. Only 3 (17%) of 18 patients had any detectable cutaneous sensory loss. In 14 (78%) of 18 patients, maximal weakness seemed to appear

TABLE 1. Comparison of clinical data for patients who underwent C4–5 decompression with and without the complication of C5 nerve palsy

Characteristic	C5 Palsy	No C5 Palsy	p Value
No. of patients	18 (2.8%)	624 (97.2%)	
Age, yrs	66.3 ± 7.59 (54–76)	63.4 ± 5.05 (22–91)	0.12
Male/female ratio	10:8	315:309	0.67
Follow-up, mos	20 ± 10.7 (12–40)	51.4 ± 39.6 (12–152)	0.007
Diabetes	4 (22%)	112 (18%)	0.64
BMI, kg/m ²	27.7 ± 4.99 (20–38)	28.6 ± 7.65 (18–47)	0.30
Preop mJOA score	11.4 ± 1.68 (8–13)	11.7 ± 1.62	0.60
Postop mJOA score	15.4 ± 1.38 (13–18)	15.6 ± 1.39 (10–18)	0.61
Intraop C5 neuromonitoring changes	0 (0%)	0 (0%)	
No. of surgical levels	3.16 ± 1.04 (1–4)	2.94 ± 1.01 (1–6)	0.18
Anterior/posterior approach	6/12	435/189	<0.001
Preop increased T2 cord signal intensity	15 (83%)	436 (70%)	0.23
Preop increased T2 cord signal intensity at C4–5	12 (67%)	149 (24%)	<0.00003

Values are presented as the mean ± SD (range) or as the number (%) of patients. Boldface type indicates statistical significance.

abruptly rather than developing gradually, but in the remaining 4 (22%), only mild weakness was initially noticed that progressively worsened to a maximum deficit over 48 hours. Weakness began to improve at a mean of 11.7 weeks (range 4–50 weeks), but 50% had begun to show signs of improvement at 8 weeks, and 89% ultimately had recovery at least to grade 4/5 strength in the deltoid muscle by the last regular follow-up visit at 12 months following surgery. There was no instance of intraoperative loss of SSEPs or MEPs or any sustained or abnormal intraoperative C5 electromyography (EMG) activity. No patient with C5 palsy underwent an exploration or other surgical treatment for the complication.

Illustrative Cases

Case 1

A 74-year-old woman with a medical history of poorly controlled non–insulin-dependent diabetes, hypertension, and morbid obesity (BMI of 45 kg/m²) presented with an 8-month history of bilateral arm pain, numbness in the hands, and a feeling of gait imbalance. There were no complaints of sphincter dysfunction, however. Neurological examination revealed weakness of the bilateral triceps, hand intrinsic muscle strength of grade 4/5, and nondermatomal sensory loss in the hands bilaterally. The deep tendon reflexes were 1+/4 in the upper extremities and ankles, but 3+/4 at the knees. The toes were downgoing, but a left Hoffmann sign was present. Cervical spine MRI (Fig. 1) demonstrated disc/osteophyte at C4–5 and C5–6 causing spinal cord compression. She underwent a C4–6 ACDF, without intraoperative complications, changes in motor or sensory monitoring, or unusual EMG activity. Following surgery, she noticed subjective improvement in hand sensation bilaterally, and the weakness in the triceps and hand intrinsic muscles had resolved. On postoperative day 2, however, she began to complain of new shoulder pain on the left side. The following morning, she had new deltoid and biceps weakness (grade 1/5 and 4/5, respectively)

on the left. There was no sensory loss or return of any distal upper-limb weakness. Intravenous steroid therapy was initiated but was discontinued later the same day because her serum glucose levels were elevated (> 400 mg/dl). Cervical spine MRI (Fig. 1) revealed a satisfactorily decompressed spinal cord at the surgical levels, but evaluation of the foramina was limited by the presence of an artifact from the titanium endplate coating of the PEEK cage and the plate. CT scanning (Fig. 1) showed a satisfactory decompression of the spinal canal but persistent foraminal stenosis on the left side. A posterior foraminotomy was discussed with the patient, but prior to intervention, she developed chest pain that led to the placement of coronary

TABLE 2. Clinical characteristics of patients with postoperative C5 nerve palsy

Characteristic	Value
Following anterior surgery	6/441 (1.4%)
Following laminectomy & fusion	12/201 (6.0%)
Preop increased T2 cord signal intensity	15 (83%)
Preop increased T2 cord signal intensity at C4–5	12 (67%)
Bilateral palsy	2 (11%)
Muscle weakness	
Deltoid	18 (100%)
Biceps	15 (83%)
Triceps	8 (44%)
Hand intrinsic	2 (11%)
Mean time to onset of weakness, days	4.6 (2–14)
Sensory loss	3 (17%)
Pain preceding appearance of weakness >24 hrs	2 (11%)
Abrupt onset of maximal weakness	14 (78%)
Mean time to initial recovery of weakness, wks	11.7 (4–50)
Late recovery to grade ≥4/5 deltoid strength	16 (89%)

Values are presented as the number (%) of patients or as the mean (range).

FIG. 1. Case 1. Preoperative (A) and postoperative (B) sagittal T2-weighted MR images of the cervical spine obtained in a 74-year-old morbidly obese, diabetic woman with cervical myelopathy, showing compression of the spinal cord at C4–5 and C5–6 that was successfully relieved by C4–6 ACDF. Postoperative CT scans showing decompression of the canal on the sagittal reconstruction (C) but persistent foraminal stenosis on the axial image through C4–5 (D).

artery stents, which required double antiplatelet therapy for 6–12 months. The weakness resolved at approximately 3 months following surgery.

Case 2

A 62-year-old woman presented with no history of any medical illness and a 2-year history of progressive deterioration in gait stability, with changes becoming more rapid and pronounced over 3 months. She also complained of loss of hand sensation and strength in her left, more than right, upper extremity, without any changes in her bowel or bladder function. Neurological examination revealed grade 4/5 left triceps strength and hand intrinsic muscle weakness, sensory loss in the bilateral hands in a nondermatomal distribution, diffusely enhanced deep tendon reflexes, and a bilateral Hoffmann sign. Her toes were down-going. Cervical spine MRI (Fig. 2) revealed multilevel spondylosis with cord compression at every level from the C3–4 disc space through the C7–T1 disc space. Increased cord signal was noted at more than one level, especially prominent at C7–T1. She underwent a C3–T1 laminectomy and fusion with the placement of instrumentation that extended to T2 (Fig. 3); there were no intraoperative complications or changes in motor or sensory monitoring or unusual EMG activity. Prior to closure, an O-arm multidimensional scan (Medtronic Spine) verified satisfactory placement of all hardware. On postoperative day 3, the patient was discharged to a subacute rehabilitation facility with resolution of her preoperative left arm weakness and improvement in her preoperative gait dysfunction. On postoperative day 10, however, she abruptly developed left arm weakness and was transferred back to the acute care

hospital for evaluation. She complained of mild incisional pain but no arm pain, and she maintained that her hand numbness was much better than it had felt preoperatively. Neurological examination revealed weakness of grade 3/5 strength in the left deltoid, 4/5 in the left biceps, 3/5 in the left triceps, and only 2/5 in the intrinsic muscles of the left hand. No new sensory loss or gait problems were noted. Follow-up MRI (not shown) demonstrated a satisfactory decompression of the spinal cord and no significant foraminal stenosis. At 10 weeks following surgery, the patient noted improvement in the arm strength, and she was found to be neurologically intact at a 13-week follow-up visit.

Discussion

Incidence and Surgical Approach

The reported incidence of C5 palsy following cervical laminectomy and fusion ranges from 0% to as high as 30%.^{5–9} A particularly large number of published papers on C5 palsy pertain to laminoplasty, and the complication is widely thought to be more prevalent following posterior approaches than anterior approaches.¹⁰ This assumption, however, is contradicted by the findings of Nassr et al., who assessed a population of patients treated surgically in North America in which the overall rate of C5 nerve palsy was 6.7%, without statistically significant differences between those undergoing anterior versus posterior surgery.³ In the experience detailed in this investigation, all posterior surgeries were CLFs, and we found the complication to be more frequent in posterior procedures compared with the anterior procedures, consistent with the findings of a

FIG. 2. Case 2. Sagittal T2-weighted preoperative MR image obtained in a 62-year-old woman with cervical myelopathy, demonstrating multilevel spondylosis, spinal cord compression, and myelomalacia, particularly severe at the C7–T1 level.

meta-analysis by Shou et al., who found a prevalence of C5 palsy of 5.8% following posterior cervical surgery and 3.3% following ACDF.¹¹ In addition, we found that 89% of cases were unilateral, similar to the results of Sakaura et al., who reported unilateral deficits in 92% and bilateral deficits in 8% of cases.⁹

Mechanism of Injury

The precise mechanism involved in the pathogenesis of postoperative C5 palsy is unknown. The greatest focus of attention in the literature has been directed at a theory of dorsal migration of the cord, placing tension on the anatomically short C5 nerve root that is located inferiorly in the foramen, which leads to neuropraxia on the basis of stretch injury;^{12–17} this phenomenon may be aggravated by tethering of the C5 root from foraminal stenosis at C4–5 and the fact that the superior articular process of C5 protrudes more anteriorly than at other levels.^{10,12,18} For CLF, attempts to associate the appearance of a deficit with the width of the laminectomy have produced conflicting results, with Bydon et al. finding a relationship with the appearance of C5 palsy and Klement et al. finding no correlation.^{7,19} In the laminoplasty literature, the palsy appears to be more frequent on the gutter side as opposed to the hinge side and is seen more frequently in the presence of preoperative foraminal stenosis.²⁰ To explain the development of the deficit following the anterior approach,

FIG. 3. Case 2. Postoperative lateral (left) and anteroposterior (right) cervical spine radiographs following C3–T1 laminectomy and fusion with instrumentation.

it has been proposed that anterior expansion of the dura may lead to increased tension along the ventral rootlets,²¹ and it has been suggested that a narrower anterior decompression may be protective.²² Some of these postulated mechanisms may be of particular relevance to the patient described in this report's first illustrative case, in which the authors believe that persisting foraminal stenosis may have contributed to the nerve palsy.

Additional proposed causes of injury include rotation of the cord, reperfusion injury, and C5 root tension from traction placed on the shoulders during operative positioning.^{23–26} A thermal mechanism in which heat generated from the high-speed drill causes neural injury has also been proposed, stemming from an observation of Takenaka et al. that the palsy may be common with use of the diamond drill.²⁷ They have thus advised the use of chilled irrigation during surgery.²⁸ The authors of this investigation, however, find the hypothesis of a thermal mechanism of injury hard to reconcile with the typical delayed appearance of deficit. Indeed, a thermal injury to the C5 root should be an intraoperative injury expected to be clearly identifiable on intraoperative neuromonitoring and apparent clinically in the recovery room immediately following surgery, something not seen in any patient in this series. Following consideration of the various potential causes, we are attracted to the thoughts expressed by Jack et al., who proposed that cases of C5 palsy may not be a uniform group explainable by a single pathophysiological mechanism, but, rather, the observed deficit may be the end result of a conglomeration of factors.¹

Preoperative T2 Cord Signal Change and Myelomalacia

In our study, there was no difference in preoperative mJOA scores or the magnitude of neurological improvement following surgery when comparing those who developed C5 palsy and those who did not. The presence of preoperative cord signal change/myelomalacia specifically at the C4–5 level, however, was found in this study to be strongly associated with the appearance of postoperative C5 palsy (Fig. 4). There are conflicting reports as to whether the presence of increased signal intensity on preoperative MRI is a negative risk factor with regard to neurological outcome.²⁹ Looking past the immediate post-

FIG. 4. Preoperative sagittal T2-weighted MR images obtained in a 54-year-old man with ossification of the posterior longitudinal ligament (A), a 74-year-old woman with spondylosis (B), and a 76-year-old man with spondylosis (C), all of whom underwent cervical laminectomy and fusion for signs and symptoms of myelopathy; the surgery improved myelopathy-related neurological deficits, but their course was complicated by C5 nerve palsy. In each case, prominent high cord signal intensity was observed at the C4–5 level.

operative period, however, there is some evidence that patients with myelomalacia fare poorer in terms of long-term neurological function. A hypothesized reason for this is that age-related deterioration of the nervous system is more likely to manifest clinically in those who have already sustained permanent damage to the spinal cord.³⁰ There has been limited study of any cumulative effects of repeated spinal cord trauma and any associated changes over time, though this has been studied extensively in cases of head injury. It may be reasonably predicted that the spinal cord and brain, both being part of the central nervous system, may have parallels with regard to responses to injury.^{31,32} A concept of “loss of neurologic reserve” is thought to be helpful in understanding late-onset deterioration in cognitive and motor function following cardiac surgery and repetitive head injury, and late decline of a similar nature has been seen subsequent to spinal cord injury, even though at the time of initial injury only mild or entirely subclinical neurological impairment was seen.^{33–36} Thus, we postulate that the presence of segmental neuronal loss and gliosis at the C4–5 level, reflected by the presence of myelomalacia on preoperative imaging, may predispose to the development of clinical weakness and that stress, without regard to the character of the underlying mechanism, may make it more likely to result in an overt deficit.

Time to Onset of Symptoms and Recovery

One of the most striking features of C5 palsy is its delayed appearance, which may not even be present while a patient is still confined to the hospital, a pattern at odds with deficits apparent immediately following surgery, which would indicate an intraoperative injury.^{4,11,37,38} We

found a mean time to deficit onset of 4.6 days with all deficits occurring between 2 and 14 days. As has been postulated by authors looking at mechanisms of injury after both anterior and posterior procedures, this may be best explained by persisting root tension that gradually leads to neuropraxic injury. Time to recovery has been reported to vary, with most reports indicating that recovery occurs over a period of 3–12 months. The description of the time course to recovery in our report provides additional granularity to the specific time course of the improvement.^{4,11,37,38} The finding of a mean lag time to initial recovery of 11.7 weeks would suggest that clinicians should counsel patients with postoperative C5 palsy that they should expect that the start of recovery may take at least 3 months.

Utility of Intraoperative Neuromonitoring

Jimenez and colleagues reported a reduction in the incidence of C5 palsy when using continuous EMG.³⁹ In this series, however, intraoperative MEP and SSEP or EMG monitoring did not provide any useful information in terms of the prediction of a C5 palsy, consistent with the findings of Currier.⁴⁰ Indeed, palsies of the C5 nerve of the sort reported by Fan et al. that were detectable with intraoperative EMG and MEP monitoring seem to be true intraoperative injuries apparent when the patient awakens from anesthesia, and a different type of injury, such as the C5 palsy, which is the subject of this investigation, that presents in a typical delayed fashion.^{9,24,37} We did not encounter any instances of false-positive changes on EMG indicative of C5 injury in those patients who did not exhibit the C5 paresis syndrome, but there were two instances of loss of bilateral SSEPs that, in each case, were not cor-

related with loss of MEPs and proved to be false-positive warnings.

Pattern of Motor and Sensory Deficits

Motor loss is the defining characteristic of the C5 palsy syndrome, and it has been described in some investigations as “dissociated motor loss.”³⁴¹ While some authors have reported up to half of their patients developing sensory loss in the shoulder or arm,⁹ our results in this 18-patient series were that sensory loss was present only in 17%, speaking for a phenomenon characterized primarily by motor deficit.

The deltoid muscle is most prominently affected by a C5 palsy, but most of patients in this series also had muscle groups affected other than the deltoid. The C5 nerve root may make a significant contribution to the biceps muscle that might readily explain biceps weakness, but many patients in our series also had detectable triceps weakness, a muscle that typically is predominantly innervated by the C7 nerve root.⁴² It has been shown that motor and sensory innervation may differ from individual to individual due to the patterns formed within the brachial plexus and the presence of intradural connections between adjacent roots.⁴³ Thus, variability of the precise pattern of innervation in different individuals may provide some explanation for the pattern of motor deficits seen with postoperative C5 palsy, particularly in cases of functional loss that can be attributed to an adjacent root; however, we found that ascribing a deficit to C5 root palsy was not a satisfying explanation for the patient with profound postoperative hand intrinsic muscle weakness detailed in the illustrative case 2, in whom preoperative MRI had shown multilevel increased signal intensity (Fig. 2).

We think that it is reasonable to accept that the theory of dorsal cord migration is at least part of the underlying mechanism involved in the pathogenesis of most cases of C5 palsy and that the accompanying tension on the cervical roots may actually lead to clinical deficits referable to levels other than C5. These deficits, however, are less frequent and prominent than those referable to C5 and, as such, are infrequently independently noted or reported.⁴ An alternative explanation is advanced by Brown et al., who presented a series of 6 patients diagnosed with the Parsonage-Turner syndrome, a brachial plexitis possibly caused by a reactivation of a dormant viral agent from changes in the immune system related to the stress of surgery. They posited that this condition is a cause of delayed and predominantly motor deficit following decompressive cervical spine surgery that may involve multiple root dysfunction.³⁷ Indeed, Park and associates have suggested that this diagnosis may actually be responsible for many cases that had been diagnosed with C5 palsy.⁴⁴ We believe that it is possible that this condition may have been a cause of the deficit in the 2 patients in this series who had complained of severe shoulder pain that preceded the development of a motor deficit, a prominent feature of the Parsonage-Turner syndrome that is not usually present in the typical cases of C5 palsy, although this diagnosis had not been considered in either case at the time the complication was being managed.

Necessity of MRI in Evaluating Postoperative C5 Palsy

In the initial period covered in this investigation, the usual practice was to perform cervical spine MRI promptly following the appearance of the C5 palsy, as would typically be done in any case of a new and unexplained neurological deficit in the immediate postoperative period. In no instance, however, did we observe any actionable new finding in these studies, such as a compressive hematoma. Not seeing clinical utility, we eventually discontinued the routine practice of ordering MRI in a postoperative patient with new difficulty lifting the arm and fitting the pattern of a C5 palsy but without distal upper-extremity weakness or worsening of any of the other myelopathic symptoms. We reason that defects appearing in a delayed fashion, such as the formation of an epidural hematoma, abscess, cerebrospinal fluid fistula, or some other mass, would produce spinal cord compression with clinical findings that should manifest bilaterally and in all muscles below the level of compression; this is in stark contradistinction to the typical presentation of the C5 palsy. Additionally, compression of the neural elements introduced intraoperatively, such as from screw malposition, would be evident when the patient awakens from anesthesia and would likely be picked up by intraoperative neuromonitoring.

Study Limitations

This investigation is limited by its retrospective design and the potential for inaccuracies that may result from relying on information obtained from the hospital and office charts. In addition, the selection of a surgical treatment approach may have introduced a selection bias in terms of which patients were selected to undergo either anterior or posterior approaches. In addition, considering that only a small percentage of patients undergoing surgery develop the C5 palsy syndrome, the sample size of affected patients is, perforce, small.

Conclusions

The distinctive characteristics of the postoperative C5 root palsy syndrome are that it appears in a delayed fashion, that it is predominantly a motor deficit, and that weakness is frequently appreciated in the biceps and triceps in addition to the deltoid muscle. We found that preoperative cord signal change/myelomalacia at C4–5 is a significant risk factor. Neuromonitoring is not useful in detecting affected patients. Based on the observation that neurological recovery of deltoid strength to at least grade 4/5 occurs in nearly 90% of individuals, patients suffering the complication can be counseled that the prognosis for recovery of function is very favorable but requires tremendous patience because it may not occur for at least 3 months.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Houten. Acquisition of data: Houten, Collins. Analysis and interpretation of data: all authors. Drafting the article: Houten, Collins. Critically revising the article: Houten, Buksbaum. Reviewed submitted version of manuscript: Houten. Approved the final version of the manuscript on behalf of all authors: Houten. Statistical analysis: Houten, Collins. Administrative/technical/material support: Houten. Study supervision: Houten.

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