Surgical Neurology International

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Original Article

Suboccipital craniotomy for Chiari I results in evoked potential conduction changes

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Received: 18 May 2012 Accepted: 28 August 2012 Published: 31 December 12

This article may be cited as:
Chen JA, Coutin-Churchman PE, Nuwer MR, Lazareff JA. Suboccipital craniotomy for Chiari I results in evoked potential conduction changes. Surg Neurol Int 2012;3:165.
Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2012/3/1/165/105277

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Abstract

Background: Management of Chiari I is controversial, in part because there is no widely used quantitative measurement of decompression. It has been demonstrated that brainstem auditory evoked responses (BAER) and somatosensory evoked potentials (SSEP) have decreased conduction latencies after wide craniectomy. We analyzed these parameters in a suboccipital craniectomy/craniotomy procedure.

Methods: Thirteen consecutive patients underwent suboccipital decompression for treatment of symptomatic Chiari I. Craniectomy was restricted to the inferior aspect of the nuchal line, and in most cases the bone flap was replaced. Neuronal conduction was monitored continuously with median nerve somatosensory evoked potentials (M-SEP), posterior tibial nerve somatosensory evoked potentials (T-SEP), BAER, or a combination. The M-SEP N20, T-SEP P37, and BAER V latencies were recorded at four milestones – preoperatively, following craniotomy, following durotomy, and following closure.

Results: Five males and eight females, with average age of 9 years, were studied. Clinical improvement was noted in all 13 patients. M-SEP N20 latency decreased from a mean of 18.55 at baseline to 17.75 ms after craniotomy ($P = 0.01$); to 17.06 ms after durotomy ($P = 0.01$); and to 16.68 ms after closing ($P = 0.02$). T-SEP P37 latency did not change significantly. BAER V latency decreased from a mean of 6.25 ms at baseline to 6.14 ms after craniotomy ($P = 0.04$); to 5.98 ms after durotomy ($P = 0.01$); and to 5.95 ms after closing ($P = 0.45$).

Conclusion: Significant improvements in conduction followed both craniectomy and durotomy. Bone replacement did not affect these results.

Key Words: Chari malformation, craniotomy, intraoperative neurophysiological monitoring

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.105277

Quick Response Code:

INTRODUCTION

A great deal of uncertainty exists regarding the Chiari type I malformation. Its pathophysiology is poorly understood, its natural history is unpredictable, and its response to various treatment methods has not been supported with robust evidence. This uncertainty manifests itself in myriad ways, but the most pressing

issue for surgeons is the practice variation of the management of Chiari type I malformations.

Currently there are two major approaches to patients with Chiari type I malformation. One is the sectioning of the filum terminale that is based on the principle that the Chiari malformation can result from caudal traction or craniocervical growth collision.[24] And the other, more widely accepted treatment is based on the concept that the Chiari malformation is in essence a phenomenon resulting from a disproportion between cerebellar tissue and the volume of the posterior fossa.[19] This concept leads to the idea that the best treatment would be enlarging the boundaries of the posterior fossa.

A large number of variations of posterior fossa decompression have been used, further increasing the heterogeneity of surgical intervention. Some surgeons perform a large occipital craniectomy,[26] others add duroplasty,[9,17] and some cauterize or excise the cerebellar tonsils.[14] In essence each of the methods has been shown to be successful. This situation requires an objective way of determining the length and the extent of the posterior fossa decompression. The ideal method is one that can be used intraoperatively to guide management and in recent times there have been reports of the usefulness of somatosensory evoked potentials (SSEP) and brainstem auditory evoked responses (BAER) in patients with Chiari type I.[2,29] In these studies, the authors have observed improvement of these values after the removal of the bone. Nonetheless the extent of the craniectomy has never been clearly defined by the authors who performed intraoperative SSEP. It is well known that in some cases when the craniectomy is very extensive the cerebellar tissue may slump into the craniectomy site and the cervical canal.[11]

It is our practice to perform craniectomy of a defined size bounded superiorly by the nuchal line.[7] Furthermore, we also have been replacing the bone flap in a fashion that does not defeat the purpose of achieving a decompression of the posterior fossa. Other authors have also reported similar procedures.[27] In order to increase our understanding of the effect of bone decompression over the cerebellum we decided to test with SSEP and BAER during our regulated craniectomy. As our practice is to replace the bone flap we also wanted to establish if such a procedure altered the neurophysiological measurements.

PATIENTS AND METHODS

Patients

Between March 2011 and April 2012, 13 consecutive children presenting with symptomatic Chiari type I malformation were treated with suboccipital craniotomy [Table 1]. The patients ranged in age from 2 to 17 years; the mean age was 9 years. Eight were female and five were male. Three patients had syringomyelia secondary to the Chiari type I malformation. Preoperatively, patients received magnetic resonance imaging (MRI) and clinical assessment. Presenting symptoms that were indications for surgery included headache, neck pain, pain of the upper or lower extremities, scoliosis, sleep apnea, lack of coordination, and seizures. Twelve patients were followed intraoperatively with Median Nerve SSEPs (M-SEP). Of these, seven had additionally Posterior Tibial nerve SSEPs (T-SEP) and BAERs. Two had only T-SEPs; one had only BAERs.

Surgical technique

Suboccipital craniotomy was performed in all patients by one surgeon (JAL), as previously described, with the additional use of SSEP and BAER monitoring.[7] With the patient in the prone position and the head flexed, the occipital bone was exposed. Two small burr holes were made just below the lip of the nuchal line, each approximately 2 cm lateral of midline. A rectangular bone flap was then removed by extending the burr holes down to the rim of the foramen magnum [Figure 1]. C1 laminectomy was performed in three cases. The dura was incised in a "Y" shape and patched with a triangular piece of Dura Guard [Figure 2a]. The bone flap was flipped in order to take advantage of its concavity; furthermore, a Leibinger plate was connected to the inner table of the bone flap and the outer table of the skull for attachment, thus creating additional space in the posterior fossa [Figure 2b]. In three cases this was not feasible, because the bone flap was too thin or too thick to replace. The wound was then closed in layers.

Neurophysiological recording

Intraoperatively, M-SEPs, T-SEPs, and BAERs were continuously recorded to functionally assess the neural tissue at risk during the surgery, as well as for the purposes of this study. Needle electrodes were inserted after anesthesia induction at Fz, C3', Cz', C4', A1, A2, and at the level of C5 spine (C5s) for recording. Amplifier gain was 10 μV/div. Bandpass filters were 30–1500 Hz for SEPs and 100-3000 Hz for BAERs. The following channels were used: Ci'-Fz, Cc'-Ci', and C5s-Fz for M-SEPs; Cz'-Fz, Ci'-Cc'c, and C5s-Fz for T-SEPs; Ai-Cz and Ac-Cz for BAERs (i = ipsilateral, c = contralateral to the stimulated side). Continuous electroencephalography (EEG) from the same channels was continuously recorded and displayed as control. Monitoring was continued until the end of the surgery, typically 40-60 minutes after replacement of the bone flap.

Neurophysiological stimulation

Electrical pulses of 200 ms duration and with intensity of 20 mA were delivered to each nerve with a rate of 4.19 Hz through needle electrode twisted pairs applied

Table 1: Patient description

Patient	Age	Gender	Presentation	Surgical procedure†	Evoked potential modality‡	Postoperative status
1	4	F	headache, leg pain	CE+D+P+T+CO	M-SEP	Improved headache, pain, and balance at 6 mo
2	12	F	headache, seizures	CE+D+P+T+CO	M-SEP, T-SEP, BAER	Tonsils ascended at 3 days. Improved headache and seizures
3	7	M	lack of balance, penmanship	CE+L+D+CO	M-SEP, T-SEP, BAER	Fine motor skills dramatically improved at 2 mo
4	7	F	headache	CE+L+D+CO	M-SEP, T-SEP, BAER	Improved headaches, tonsils ascended at 6 mo
5	10	M	occipital pain, behavior problems, syringomyelia	CE+L+D+CO	BAER	Clinically improved but no radiological evidence ascertained yet
6	14	F	scoliosis, syringomyelia	CE+D+P+CO	M-SEP, BAER	Improved headache, smaller syrinx at 2 mo
7	7	M	sleep apnea	CE+D+CO	M-SEP, T-SEP, BAER	Improvement in clinical condition
8	5	F	headache, leg pain	CE+D+CO	M-SEP, T-SEP	Improved headache at 2 mo
9	17	F	neck pain, arm paresthesia	CE+D+CO	M-SEP, T-SEP	Asymptomatic, tonsils ascended at 3 mo
10	13	F	papilledema, absence seizures	CE+CO	M-SEP, T-SEP, BAER	Seizures discontinued at 2 mo
11	4	F	syringomyelia	CE+D+P+T	M-SEP, T-SEP, BAER	Clinically improved but no radiological evidence ascertained yet
12	2	M	sleep apnea, headache	CE	M-SEP	Headache improved at 1 mo. Sleep apnea controlled at 9 mo
13	12	M	neck/shoulder pain, ataxia	CE	M-SEP, T-SEP, BAER	Improved headache and balance at 1 mo

†CE: Craniectomy, L: C1 laminectomy, D: Duroplasty, P: Pial cauterization over the tonsils, T: Reduction of the tonsils, CO: Craniotomy with replacement of the bone flap.
‡M-SEP: Median somatosensory evoked potentials, T-SEP: Posterior tibial somatosensory evoked potentials, BAER: Brainstem auditory evoked responses

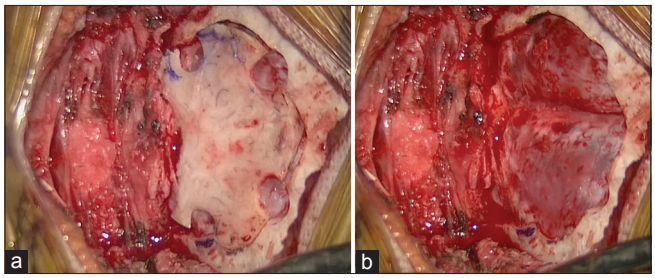

Figure 1: Removal of the bone flap as part of the posterior fossa decompression. (a) a 3 cm x 3 cm bone flap was created, (b) the bone flap is removed, exposing the dura covering the cerebellar tonsils

over the median nerve at the wrist and the posterior tibial nerve behind the medial malleolus. Monoaural clicks with an intensity of 80 dB nHL delivered through insert tube earphones at a rate of 11.1 Hz were used for acoustic stimulation for BAERs.

Data analysis

Between 500 and 750 responses were averaged for SEPs and 2000 for BAERs. Timebase was 15 ms for BAERs, 50 ms for M-SEPs, and 100 ms for T-SEPs. Responses were collected in the following sequential cycle: M-SEPs (bilateral alternating)/T-SEPs (bilateral alternating)/Left BAER/Right BAER. The time span for collecting each full set of responses was between 5 and

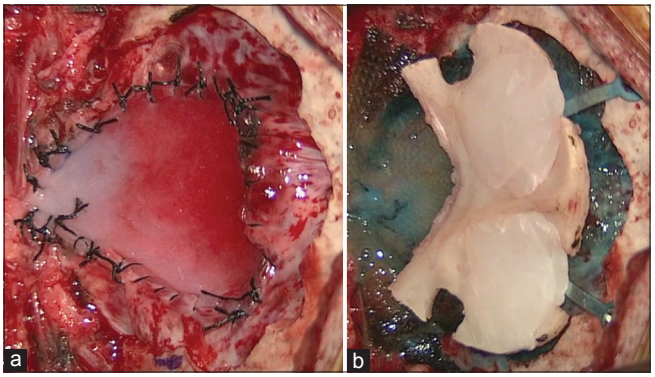

Figure 2: Completion of duroplasty and replacement of the bone flap. (a) dural patching with synthetic dural replacement, (b) the bone flap is inverted and lifted over the craniotomy site using Leibinger plates. The posterior fossa is enlarged in this manner

8 minutes, except for intervals of intense electrocautery when updates took longer due to artifact rejection. Cortical M-SEP N20 latency, cortical T-SEP P37 latency, and BAER wave V latency were followed through the case. The spinal component (N13 for M-SEP, N30 for T-SEP) was also used as reference.

The stage of surgery at which the measurement was taken - before craniotomy (baseline, BL), immediately after bony decompression (BN), immediately after opening

the dura (DUR), and immediately after replacement of the bone (END) – was used as the independent variable. Latency measurements taken at each stage (average of right and left sides) were considered the dependent variable. Data was analyzed using a repeated measures design two-way analysis of variance (ANOVA), with rejection threshold α of 0.05. Statistical analysis was performed using MATLAB 7.0 R14 (The MathWorks Inc., Natick, MA).

RESULTS

Somatosensory evoked potentials

M-SEP N20 latency improved in all 12 patients (100%), while T-SEP P37 latency improved in 7 of the 9 patients (78%) for which this procedure was performed [Figure 3]. Patients 2 and 10 had a slightly increased N20 latency (increases of 2.7% and 0.27%, respectively). The improvements were consistent in direction between right and left recordings in each patient; however, patients sometimes had a greater magnitude of improvement unilaterally. Representative SSEP recordings from one of our patients are shown in Figure 4. On average, the latency times were reduced at each stage of the surgery. The M-SEP N20 latency decreased from 18.55 ± 0.34 ms (mean ± standard error) at preoperative baseline to 17.75 ± 0.26 ms after removal of the bone flap (P = 0.01); further decreasing to 17.06 ± 0.19 ms after opening of the dura (P = 0.01); and again decreasing to 16.68 ± 0.20 ms after closing (P = 0.02). Improvements in the neurophysiological parameters were sustained for the duration of the procedure. Interaction terms of the ANOVA were demonstrated insignificant at the α =0.05 level.

Similarly, the T-SEP P37 latency decreased from 35.65 ± 0.78 ms at preoperative baseline to 34.61 ± 0.91 ms after removal of the bone flap (P = 0.14); slightly decreasing to 33.91 ± 0.80 ms after opening of the dura (P = 0.30); and slightly decreasing to 33.07 ± 0.89 ms after closing (P = 0.18). However, none of these changes were statistically significant. Interaction terms of the ANOVA were demonstrated insignificant at the α = 0.05 level.

Brainstem auditory evoked responses

An analogous pattern of improvement in BAERs following the surgical procedure was seen in eight of nine patients (89%) [Figure 3]. Patient 10 had a slightly increased BAER wave V latency (increase of 0.79%). The improvements were consistent between right and left recordings in each patient. Representative BAER recordings from one of our patients are shown in Figure 5. At baseline, the BAER wave V latency averaged 6.25 ± 0.10 ms. Following removal of the bone flap ± laminectomy, this value decreased to 6.14 ± 0.09 ms (P = 0.04). Following opening of the dura, the wave V latency further decreased to 5.98 ± 0.09 ms (P = 0.01). After closing, the wave V latency remained stable at 5.95 ± 0.07 ms (P = 0.45). Interaction terms of the ANOVA were demonstrated insignificant at the α =0.05 level.

Craniotomy versus craniectomy

In three cases the bone flap was not replaced (craniectomy), and in nine cases it was (craniotomy). A similar general trend in decreasing latencies was observed in the M-SEP, T-SEP, and BAER measurements for craniotomy compared with craniectomy [Figure 6]. Notably, the BAER wave V latency remained stable between opening of the dura and closing in craniotomy patients (increasing slightly from 5.99 to 6.00 ms), but continued to improve in craniectomy patients (decreasing from 5.95 to 5.82). Statistical analysis was not performed due to the limited sample size of the groups.

Clinical evaluation

None of the patients experienced postoperative complications or required additional decompression, and all improved clinically following surgery [Table 1]. Syrinx size was found to be decreased in one patient on follow-up MRI. In two other patients, the MRI results were not yet available.

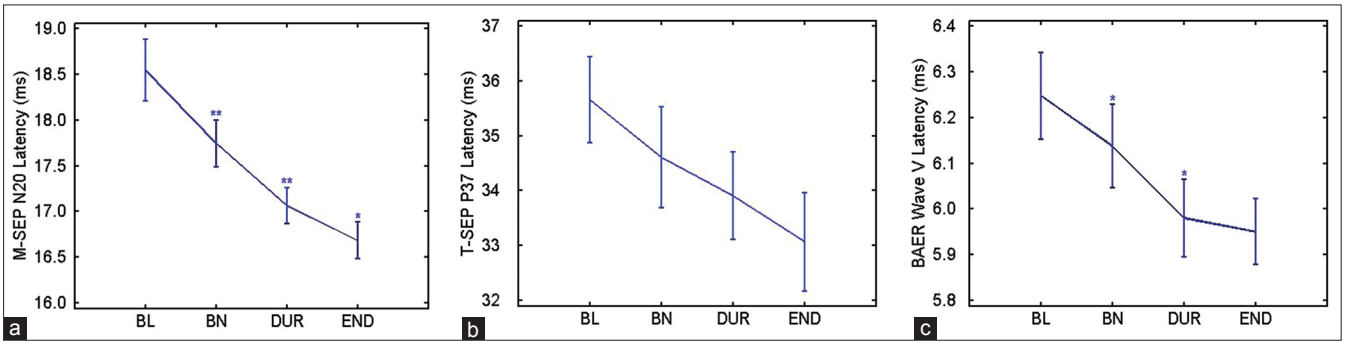

Figure 3. Progression of neurophysiologic parameters at each stage of decompressive craniotomy. (a) Median nerve SSEP N20 latency, (b) Posterior tibial nerve SSEP P37 latency, (c) BAER wave V latency. BL – preoperative baseline, BN – removal of the bone flap, DUR – opening of the dura, END – replacement of the bone flap at the end of the procedure. Values are reported as mean ± SEM. Significance: *-*P*<0.05 level, **-*P*<0.01

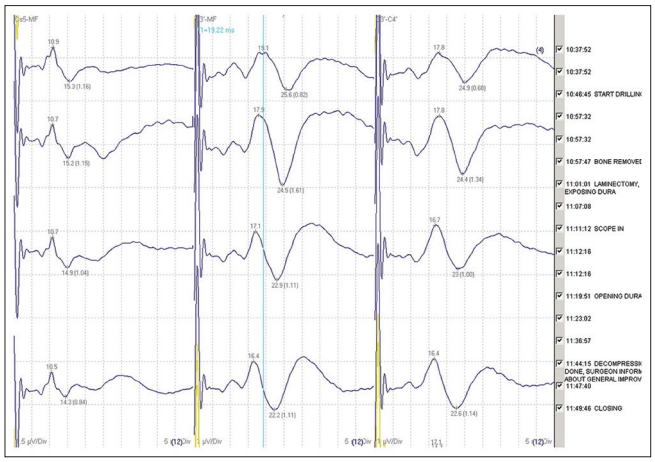

Figure 4: Representative M-SEP recording during the procedure. From top to bottom, curves represent baseline, craniectomy, durotomy, and the end of the procedure. N20 peaks are labeled; cyan line represents baseline N20 latency. The overall latency decrease from baseline was 14%

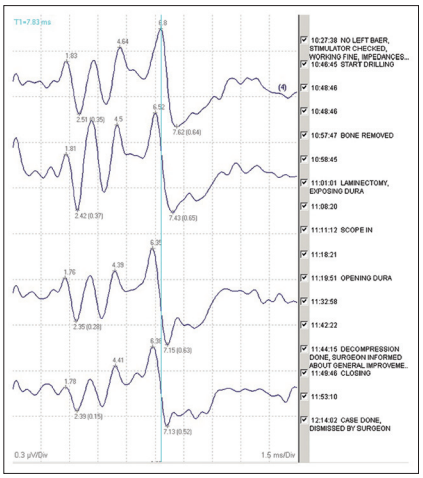

Figure 5: Brainstem auditory evoked responses recording of the same patient. From top to bottom, curves represent baseline, craniectomy, durotomy, and the end of the procedure. Wave V peaks are labeled; cyan line represents baseline V latency. The overall latency decrease from baseline was 6%

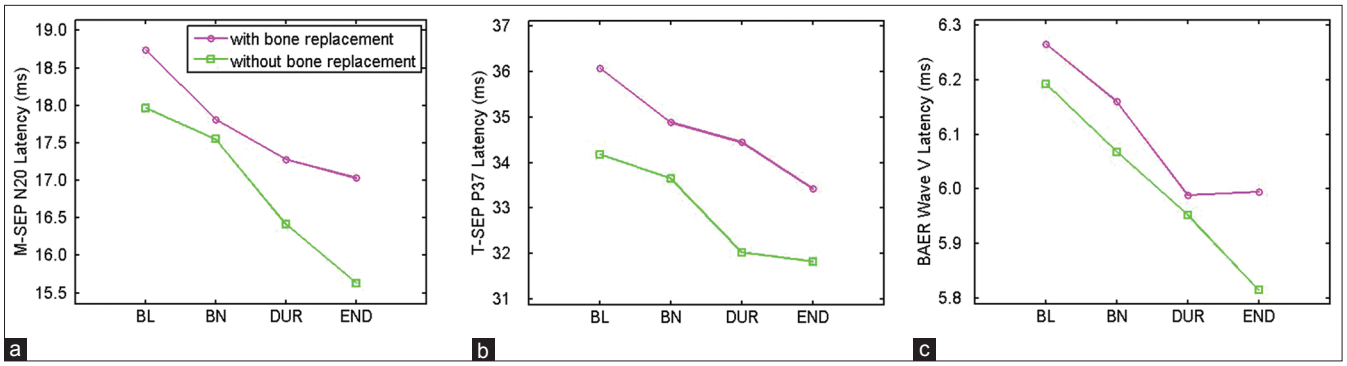

Figure 6. Comparison of craniotomy (*n* = 9) versus craniectomy (n = 2). (a) Median nerve SSEP N20 latency, (b) Posterior tibial nerve SSEP P37 latency, (c) BAER wave V latency

DISCUSSION

Since Penfield performed an occipital craniotomy on a "bookkeeper who could not wink", who he initially diagnosed with bilateral cerebellopontine angle tumor,[22] the neurosurgical community has been experimenting with different lengths, widths, and depths of decompression for Chiari malformation. In more recent times, many disparate methods have been shown to be successful. This raises the basic question of what is actually being corrected by the procedure, and how its extent can be measured.

Evoked potentials present one possible avenue to address these questions; their correlation with impulse conduction in sensory pathways forms a putative basis for changes seen in Chiari decompression. Any process compromising these tracts (e.g., demyelination, compression, or ischemia) will affect axonal function, decrease the conduction velocity, and consequently increase evoked potential latencies.[16] Intraoperative evoked potential interpretation relies on the principle that ischemia or compression of tracts will be reflected by adverse changes on evoked potentials almost immediately. This is what is classically described when acute compression/ischemia compromises the involved pathway, reflected as an increase of a given evoked potential latency 10% or more over baseline values when a surgical maneuver compromises the tract, while a decrease back to baseline values is seen when the compression/ischemia is relieved.[21] However, a change in the opposite direction, that is, immediate decreases in latency from baseline values due to the relief of chronic compression/ischemia, is much less frequently described.[2,29] Our results imply that chronic compression from Chiari I results in reversible deficits in axonal conduction through the brainstem.

Determining latency improvements for each step of the surgical treatment of Chiari malformation gives us a further understanding of the decompressive effects of certain procedures. Our craniotomy demonstrated clinically notable improvements in overall latency

times in both M-SEP N20 (by 1.87 ms) and BAER wave V latency (by 0.30 ms). The T-SEP pattern of decrease lacked statistical significance, likely due to the smaller sample size and greater inherent variability in the P37 latencies within the patient population. Anderson *et al*.[2] described similar positive changes in BAERs after bony decompression (0.31 ms decrease in wave I-V interpeak latency) but without significant additional improvement after duroplasty (0 ms decrease in wave I-V interpeak latency), using a wide craniectomy. They reported that SSEPs were stable. Zamel *et al*.[29] reported qualitatively similar results for BAERs as found by Anderson *et al*. Unlike previous authors, we have demonstrated a significant improvement in BAERs not only after bony decompression but also after duroplasty. Moreover, although those authors reported that SSEPs were stable, it is precisely the median nerve SSEPs that showed the most significant changes in our series for both bony decompression and duroplasty. Given that all patients improved clinically, and that all patients had improvements in median nerve SSEP, it seems to be most sensitive among tested evoked potentials for sufficient decompression.

Our procedure furthermore incorporated an additional step that was not used in previous studies, namely replacement of the bone flap. The improvements in M-SEP and BAER were retained following this step, suggesting that the technique we have devised does not compromise the decompressive effect of the surgery. The dorsoventral space created in this manner seems to be sufficient for decompression, while the lateral extent of the craniectomy is mostly preserved. Replacement of the bone flap was not feasible in three patients, providing an opportunity to compare craniotomy against craniectomy directly. The craniotomy procedure did not reverse the latency improvements seen in M-SEPs, T-SEPs, or BAERs, and a similar trend of improvements was observed in craniotomy versus craniectomy. Although this analysis lacks statistical power, it reflects our belief that replacement of the bone does not negate the objectives of decompression.

Taken together these findings suggest that our approximately 3 × 4 cm craniectomy restricted to the nuchal line can achieve a similar extent of decompression as a more extensive one, implying that sufficient space can be created with a less drastic approach. Our series also demonstrates improvements in M-SEPs and BAERs following both craniectomy and duroplasty, suggesting that in those instances duroplasty indeed had a decompressive effect. This finding could potentially explain the superior symptomatic improvements seen with duroplasty in the literature.[8,17] Duroplasty is effective for decompression in smaller craniectomies, while it may be superfluous with a larger craniectomy.

Interestingly, Caldarelli *et al*.[5] report a series of 30 children operated with a 2.0 × 2.5 cm craniectomy without duroplasty, with promising results. Intraoperative ultrasound was used to measure restoration of cerebrospinal fluid (CSF) flow, which presumably added confidence that the procedure was sufficient for each patient. Other authors also demonstrate success with craniectomies with dimensions of 3 × 3 cm or smaller in the pediatric population.[12,18] Considering the success of these limited procedures, it is reasonable to suggest that intraoperative neurophysiological monitoring may be used to perform suboccipital decompression in a step-by-step fashion, enlarging the craniectomy or adding additional procedures (laminectomy, duroplasty) until positive changes are observed.

Although our results are highly suggestive, some caution must be exercised in their interpretation. The use of SSEP and BAER as a real-time, intraoperative surrogate for surgical outcome or extent of decompression in Chiari type I malformation has been proposed, but not proven. The purpose of suboccipital craniectomy itself remains debatable, with various authors asserting relief of direct compression on neural structures,[1,3,4,6,15,23,28] improvements in CSF flow,[3,10,15,22,23,25,28] decompression of vascular structures,[23] and/or creation of a cisterna magna,[15,17] as principal objectives of the procedure. However, our study and others have shown that the neurophysiological parameters consistently improve following decompression. This improvement was discussed by Anderson *et al*. who postulated that compression of the descending motor and ascending sensory pathways at the cervicomedullary junction by the cerebellar tonsils resulted in increased BAER latencies.[2] The pattern of SSEP changes also suggests that the neurophysiologic changes following decompression are due to a local phenomenon. Nonspecific factors, such as a rise in patient temperature, may also result in improved conduction; however, in that case the longer latency T-SEP P37 would be expected to show roughly twice the improvement of the shorter M-SEP N20. Furthermore, in our experience it is rare to observe conduction improvements of such magnitude in other neurosurgical procedures.

The practice variation in the management of Chiari type I malformation suggests the need for a widely accepted quantitative measurement of decompression. While our data was analyzed in a retrospective manner, there have been previous reports of intraoperative diagnostic measurements used to guide surgical management of the Chiari type I malformation. In the recent past the use of intraoperative ultrasound imaging has been advocated to gauge the extent of the decompression.[15] Our paper is similar in that regard; by tiptoeing outside the beaten path, we suggest a new and modest means

of understanding the Chiari malformation. With further refinement, these techniques may help surgeons to better understand the decompressive effects of various procedures and to minimize the extent of surgical intervention to only what is required for satisfactory clinical outcome.

Essentially, the crux of the debate regarding the surgical treatment for Chiari malformation is the tradeoff between the extent of decompression and adverse effects.[13,20] Several studies have demonstrated a correlation between increasing craniectomy size and improvement in Chiari malformation,[20] though complications (most notably cerebellar ptosis) have been reported if the craniectomy is too large.[11,12] In this study we used intraoperative neurophysiological monitoring to illuminate the effects of surgical variations – namely a craniectomy that respects the nuchal line and with replacement of the bone – on the magnitude of decompression. Since we demonstrated similar improvements in decompression despite using arguably a more conservative approach, an understanding of what factors actually affect decompression may yield surgical procedures with similar rates of patient improvement but reduced adverse outcomes.

ACKNOWLEDGMENT

The authors thank Ms. Vanessa Marrero for administrative support. JAC is a student in the University of California, Los Angeles Medical Scientist Training Program and was supported in part by NIH NIGMS Training Grant GM08042.

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