Published in final edited form as:

J Neurosurg Spine. 2014 October ; 21(4): 559–564. doi:10.3171/2014.6.SPINE13950.

Effect of craniovertebral decompression on CSF dynamics in Chiari malformation Type I studied with computational fluid dynamics:

Laboratory investigation

Svein O. Linge, Ph.D.^{1,2}, Kent-A. Mardal, Ph.D.², Anders Helgeland, Ph.D.^{2,3}, John D. Heiss, M.D.⁴, and Victor Haughton, M.D.^{2,5}

¹Telemark University College, Porsgrunn ²Center for Biomedical Computing, Simula Research Laboratory, Lysake ³Norwegian Defense Research Establishment (FFI), Kjeller, Norway ⁴Surgical Neurology Branch, National Institutes of Health, Bethesda, Maryland ⁵Department of Radiology, University of Wisconsin Hospitals and Clinics, Madison, Wisconsin

Abstract

Object—The effect of craniovertebral decompression surgery on CSF flow dynamics in patients with Chiari malformation Type I (CM-I) has been incompletely characterized. The authors used computational fluid dynamics to calculate the effect of decompression surgery on CSF flow dynamics in the posterior fossa and upper cervical spinal canal.

Methods—Oscillatory flow was simulated in idealized 3D models of the normal adult and the CM-I subarachnoid spaces (both previously described) and in 3 models of CM-I post–craniovertebral decompressions. The 3 postoperative models were created from the CM model by virtually modifying the CM model subarachnoid space to simulate surgical decompressions of different magnitudes. Velocities and pressures were computed with the Navier-Stokes equations in Star-CD for multiple cycles of CSF flow oscillating at 80 cycles/min. Pressure gradients and velocities were compared for 8 levels extending from the posterior fossa to the C3–4 level. Relative pressures and peak velocities were plotted by level from the posterior fossa to C3–4. The heterogeneity of flow velocity distribution around the spinal cord was compared between models.

Results—Peak systolic velocities were generally lower in the postoperative models than in the preoperative CM model. With the 2 larger surgical defects, peak systolic velocities were brought closer to normal model velocities (equal values at C-3 and C-4) than with the smallest surgical defect. For the smallest defect, peak velocities were decreased, but not to levels in the normal

©AANS, 2014

Address correspondence to: Svein Linge, Ph.D., Telemark University College, P.O. Box 203, N-3901 Porsgrunn, Norway. sveinlin@simula.no.

Author contributions to the study and manuscript preparation include the following. Conception and design: all authors. Acquisition of data: Linge, Mardal, Helgeland, Haughton. Analysis and interpretation of data: all authors. Drafting the article: Linge, Mardal, Helgeland, Haughton. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Linge.

model. In the postoperative models, heterogeneity in flow velocity distribution around the spinal cord increased from normal model levels as the degree of decompression increased.

Pressures in the 5 models differed in magnitude and in pattern. Pressure gradients along the spinal canal in the normal and CM models were nonlinear, with steeper gradients below C3–4 than above. The CM model had a steeper pressure gradient than the normal model above C3–4 and the same gradient below. The postoperative models had lower pressure gradients than the CM model above C2–3. The most conservative decompression had lower pressure gradients than the normal model above C2–3. The two larger decompression defects had CSF pressure gradients below those in the normal model above C2–3. These 2 models had a less steep gradient above C-3 and a steeper gradient below.

Conclusions—In computer simulations, craniovertebral surgical defects generally diminished CSF velocities and CSF pressures.

Keywords

Chiari malformation; decompression surgery; cervical; computer simulation

The effect of craniovertebral decompression on CSF fluid dynamics has not been fully characterized. Investigators using phase-contrast MRI in patients with Chiari malformation Type I (CM-I) before and after craniovertebral decompression^{2,4,5,10,15,16} have shown both reduced and increased CSF velocities at the craniovertebral junction. These studies record velocities at only one spinal level, usually at the tip of the tonsils, and sometimes only qualitatively. Results differ from one patient to another. The limited spatial and temporal resolutions of phase-contrast (PC) MRI diminish the value of these studies.²¹ Measuring velocities at one level alone does not suffice because the velocity of CSF flow varies along the length of the upper cervical spinal canal in patients with CM and probably to a different extent in healthy adults,^{14,18} since spinal canal dimensions differ in CM patients and controls.^{6,9} Measurements of CSF pressures after craniovertebral decompression are also limited. Phase-contrast MRI does not provide a measure of pressure. To measure pressures, investigators have canalized the subarachnoid space in patients with CM-I before and after surgery.⁷ Because these studies are invasive, they are not frequently performed, and they are only rarely performed at multiple locations.

Computational fluid dynamics (CFD), an engineering tool for the study of fluid dynamics, has been used effectively to study CSF pressures and velocities in adjacent segments of the spinal canal.^{8,11,12,14,20} The primary goal of this study was to apply CFD to compute the effect of craniovertebral decompression on CSF pressures and velocities in the posterior fossa, foramen magnum, and cervical spinal canal. As a first step toward computational modeling of surgery, idealized models of the craniovertebral decompression were used. Also previously, idealized models have been used effectively to justify studies in patient-specific models.^{3,13,22} Since craniovertebral decompression has not been standardized between surgical services or between patients, we chose to study models with decompressions of different sizes.

Methods

Models of Subarachnoid Space Geometries

Cervical CSF flow was simulated in 5 idealized models of the subarachnoid space, one model of a normal adult, one model of CM-I, and 3 models representing different-sized craniovertebral decompressions in a patient with CM.

The normal and CM models (Fig. 1) were identical, except for the presence of descended tonsils in the CM model. The tonsils were 4 cm in length, encompassing the posterior fossa to the C3–4 level approximately. A 2-cm extension was added to each end of the models to permit the use of plug-shaped velocity profiles at inflow/outflow boundaries. Both of these models have been validated and described previously.^{11,12}

The 3 surgery models (hereinafter referred to as s1, s2 and s3, in order of increasing degree of decompression) were constructed by modifying the outer boundary of the subarachnoid space in the CM model to mimic craniovertebral decompression. The sagittal and axial sections (at the foramen magnum) in Fig. 2 illustrate how the subarachnoid space was modified (note that the spinal cord diameter is 1 cm). In each case, the surgical defect covered (superiorly-inferiorly) the posterior fossa and the tonsils, and comprised the posterior part of the subarachnoid space, that is, posterior to the coronal plane. In the most conservative model (s1), the CSF depth posterior to the spinal cord was increased by 0.5 cm at the foramen magnum. In s2 and s3, this measurement was increased to 1.0 cm and 1.5 cm, respectively. In all models, the left-right width of the expansion was about 2 cm at this level. Dimensions were then gradually reduced to presurgical values superiorly and inferiorly, as well as laterally. The shape and dimensions of the tonsils were not altered. Common to all 3 surgery models was that anatomical structures within the dura mater were left unchanged, as they would be for a surgical procedure that left the dura intact. The volume increase implied by s3, however, would require the insertion of a dural graft.

Computational meshes for the 3 surgery models were very similar to the mesh for the CM model—hexahedral meshes with 600,000 nodes and a smallest distance between nodes in any cell ranging from 0.15 to 1.25 mm. A tetrahedral mesh with 60,000 nodes was used for the normal model, with the smallest distance between nodes in each cell varying between 0.1 and 5.0 mm. As for the normal and CM models, the outer boundaries of the subarachnoid space, the spinal cord, and the tonsils were all modeled as rigid and immobile. Star-CD (User Guide and Methodology, version 3.26, Computational Dynamics Ltd., 2007) was used for model construction and simulations.

Modeling Cyclic CSF Flow

All models were exposed to the same boundary conditions, corresponding to pulsating CSF flow at 80 heartbeats per minute. A laminar fluid flow of 1-ml stroke volume¹ was assumed. An asymmetrical sinusoidal waveform was applied, with the upward flow (diastole) lasting twice as long as the downward flow (systole), and having half the amplitude. The fluid had the properties of water at 37°C, with a kinematic viscosity of $0.70 \times 10^{-6} \text{ m}^2/\text{sec}$. The cranial flow direction was chosen as positive (that is, diastolic velocities were defined as

positive), while systolic velocities were negative. No-slip boundary conditions were chosen at the spinal cord and dural boundaries.

The fluid was at rest when simulations were started, but the initial 6 flow cycles were skipped, using only data from when maximum velocities at the foramen magnum differed by less than 1 mm/sec and distributions were observed to be similar by visual inspection. Calculations of flow velocities and pressures were performed for each computational point at 0.001-second intervals.

Assessing Effect of Surgical Decompression

Velocity and pressure distributions in space and time were displayed in Star-CD and inspected. For all models, peak systolic velocities were plotted and compared for 8 equidistant axial levels from 1 cm above (Level 1) the craniovertebral junction to 3 cm below (Level 8). These levels correspond to the posterior fossa (Levels 1 and 2), the craniovertebral junction (Level 3), and the cervical spine from C-1 (Level 4) to C3–4 (Level 8). Also, peak systolic velocity, as percentage excess over the corresponding normal model peak velocity, was computed and plotted for each level in the CM and the postoperative models. Finally, distributions of peak systolic velocities around the spinal cord were also plotted and compared, using axial sections corresponding to local Levels 2 and 4, respectively.

Peak pressure gradients (superior-inferior) over Levels 1–8 were computed, tabulated, and compared for all models. The pressure gradient G was computed as $G = (p_t - p_b)/h$, where p_t is the pressure at the top (Level 1), p_b is the pressure at the bottom (Level 8), and h is the distance (superior-inferior) between Level 1 and 8 (0.04 m).

In addition, for each model, the pressure at each level relative to the pressure at Level 8 was computed. Comparison was done by plotting these relative pressures by level for all models in a single plot.

Results

In the 5 models, the peak CSF velocities increased nonlinearly from the posterior fossa (Level 1) to C-3 (Level 7). In the CM model, peak velocity increased from 4.3 cm/sec at the foramen magnum to 7.5 cm/sec at C-4; in the normal model it increased from 2 cm/sec to 7.2 cm/sec over the same region. Peak pressure gradients occurred at 2 times in the cycle for all models, corresponding to the time when flow changed direction.

Effect of Decompression on Velocities

The postoperative models had intermediate peak systolic velocities, that is, values that were between those obtained in the normal and CM models (Fig. 3). The CM model had faster CSF velocity in the posterior fossa, foramen magnum, and upper cervical spinal canal than the normal model during caudal flow (Figs. 3 and 4). The two larger surgical defects, s2 and s3, had reduced CSF velocities in the posterior fossa, foramen magnum, and cervical spinal canal. They normalized velocities at C-2 and C-3. The smaller defect, s1, reduced velocities to a lesser extent but not to values in the normal model. Peak systolic velocities in the

posterior fossa of the CM model exceeded normal model velocities by 65% to 120% (Fig. 4), while in the postoperative models, they exceeded normal by 30% to 70%. At C-2, the CM model and s1 had velocities 18% to 23% above normal, while s2 and s3 had velocities that did not exceed those in the normal model.

Flow patterns showed greater heterogeneity in the CM model than in the normal model, and heterogeneity increased with the size of the surgical defect. Near the foramen magnum, at the time of peak systolic flow, the difference between peak velocities anterior and posterior to the spinal cord (Fig. 4) increased with increasing size of the surgical defect. The normal model did not show the same heterogeneity in flow distribution. For example, with anterior peak velocities in s3 (Fig. 4), peak velocities diminished to approximately 3.2 cm/sec and 3.7 cm/sec for the superior and inferior axial sections, respectively. Corresponding numbers from the normal model (Fig. 4) were 2.1 cm/sec and 3.4 cm/sec. Posteriorly, s3 peak velocities diminished to 1.1 cm/sec and 2.6 cm/sec (for the superior and inferior axial sections, respectively). Corresponding velocities in the normal model were approximately 1.7–2.0 cm/sec and 3.0–3.5 cm/sec. Anterior peak velocities for s3 were 50% above normal values, whereas posteriorly they were 50% below normal values.

Effect of Decompression on Pressure

The CM model had a 15% higher peak pressure gradient than the normal model (Table 1), while all 3 surgery models had peak gradients within 8% of the normal value. Pressure gradients were nonlinear (Fig. 5). The graph of the most conservative surgical defect (that is, s1) most closely follows the relative pressure gradient in the normal model. Below Level 7, neither the CM model nor the surgery models had relative pressures that differed more than 15% from the normal model. For the same region, all surgery models had gradients that were closer to normal than the CM model. At Level 7, s2 and s3 had relative pressures that were higher (increased by less than 34%) than what was found in the other models.

Discussion

In the present computer simulation study, an idealized model of a patient with Chiari malformation Type I (CM-I) was exposed to 3 different degrees of idealized decompression. Peak systolic velocities and pressure gradients were generally lower and closer to normal model values in all postoperative models, compared with those found in the preoperative CM model. Peak systolic velocities were virtually identical in s2 and s3, and also closer to normal model values than those observed in the most conservative model s1. Although flow velocities decreased, the heterogeneity in velocity distribution around the spinal cord showed an increase as the magnitude of decompression increased. Pressure gradients were normalized by small surgical defects and reduced to levels below the pressures in the normal model with larger surgical defects.

This CSF study with CFD showed decreases in CSF velocities similar to those found in studies in which PC MRI was used. For example, Dolar et al.⁵ found that the average peak systolic velocities at the foramen magnum decreased from an average of 3.4 cm/sec before surgery to 2.4 cm/sec after surgery. In a pediatric study, Iskandar et al.¹⁰ found that peak systolic velocities were lowered in most patients following decompression. Nevertheless,

clinical studies have not consistently reported decreases in CSF velocities from decompression surgery. Sivaramakrishnan et al.¹⁹ found average maximum CSF velocities to be relatively unaffected by surgery. Armonda et al.² and Bhadelia et al.⁴ reported an increase in velocities to the posterior and anterior side of the spinal canal, respectively. Differences in methods may explain some of these inconsistencies. It should also be noted, however, that the reduction in CSF velocities found in the present study is consistent with predictions from theoretical fluid dynamics—surgical widening of the flow channel (subarachnoid space) makes CSF velocities go down while volume flux remains constant. The reduction in CSF pressure gradients found in our postoperative models is consistent with the clinical study of Heiss et al.,⁷ who measured cervical and lumbar pressure before and after decompression surgery.

The present work has several limitations. Our surgical craniovertebral defects may deviate from what is achieved by surgery. In particular, the superior extension of the defect may be less than is common practice. In theory, the effect of this difference on velocity and pressure characteristics would be small. The use of rigid subarachnoid space boundaries, including an immobile spinal cord, represents model simplification that does affect CSF velocity and pressure characteristics. This could, for example, alter the degree of heterogeneity observed in CSF velocity distribution around the cord, which increased with increasing degree of decompression. A stronger validation of the applied models would be preferable; however, idealized models are inherently difficult to validate, since they do not represent any particular patient from which measurement data could be collected for comparison. Computer-based simulations in patient-specific geometries have previously been compared with flow measurements using MRI.^{17,22} While more detailed validation and development of more accurate computational models are warranted, these studies demonstrate that the main flow is often well captured. The main flow is strongly coupled to the pressure distribution, suggesting that prediction of computational pressure distribution is feasible in patient-specific decompression models.

The pressures found in our simulation approach revealed a nonlinear relationship between pressure gradient and level. In the models, it was also found that peak velocities and pressure gradients diminished differently at different levels in the cervical spine. The relevance of this observation to the reduction of syringomyelia due to craniovertebral decompression needs additional study. Our work has clinical relevance, representing the first step toward patient-specific models for individual decompression surgery planning. This modeling technique could lead to a better understanding of the relative effects of different amounts of craniocervical decompression on CSF flow dynamics in patients with CM-I. The amount of expansion of the CSF pathways that is sufficient to treat patients with CM-I is controversial, and some neurosurgeons prefer craniocervical decompression and duraplasty to bony decompression alone in patients with syringomyelia, based on reports that syrinx resolution occurs more frequently when duraplasty is included with the bony decompression. The modeling described here explains the relative effects of different amounts of expansion of the subarachnoid space on CSF flow dynamics in these patients. On the other hand, if a syrinx, cervicomedullary sign, or other objective measure is not present before surgery, the outcome of surgery is primarily judged by the degree of postoperative symptomatic improvement. If a symptom such as headache were to persist

after craniocervical decompression, this modeling technique could predict whether the amount of expansion of the subarachnoid space produced by the craniocervical decompression was enough to effectively normalize CSF flow dynamics, and another cause of headache could then be sought if the amount of expansion was demonstrated to be sufficient. Alternatively, if the CSF flow dynamics were abnormal after surgery, this modeling could predict whether the headache could be improved by enlarging or reducing the size of the decompression.

Conclusions

The computer simulations in the present study show that CSF dynamics in the upper cervical spine depend on the type or amount of the surgical defect created. In our simulations, velocities tended toward normal model levels as the surgical defect increased in size. Pressure gradients diminished to or below normal mode levels with the addition of the craniovertebral decompression defect.

Acknowledgments

Disclosure

This study was supported by a Center of Excellence grant from the Norwegian Research Council to the Center for Biomedical Computing at Simula Research Laboratory.

Abbreviations used in this paper

CFD	computational fluid dynamics
CM	Chiari malformation
CM-I	CM Type I
PC	phase contrast

References

1. Alperin N, Sivaramakrishnan A, Lichtor T. Magnetic resonance imaging-based measurements of cerebrospinal fluid and blood flow as indicators of intracranial compliance in patients with Chiari malformation. *J Neurosurg.* 2005; 103:46–52. [PubMed: 16121972]
2. Armonda RA, Citrin CM, Foley KT, Ellenbogen RG. Quantitative cine-mode magnetic resonance imaging of Chiari I malformations: an analysis of cerebrospinal fluid dynamics. *Neurosurgery.* 1994; 35:214–224. [PubMed: 7969828]
3. Bertram CD. Evaluation by fluid/structure-interaction spinal-cord simulation of the effects of subarachnoid-space stenosis on an adjacent syrinx. *J Biomech Eng.* 2010; 132:061009. [PubMed: 20887034]
4. Bhadelia RA, Bogdan AR, Wolpert SM, Lev S, Appignani BA, Heilman CB. Cerebrospinal fluid flow waveforms: analysis in patients with Chiari I malformation by means of gated phase-contrast MR imaging velocity measurements. *Radiology.* 1995; 196:195–202. [PubMed: 7784567]
5. Dolar MT, Haughton VM, Iskandar BJ, Quigley M. Effect of craniocervical decompression on peak CSF velocities in symptomatic patients with Chiari I malformation. *AJNR Am J Neuroradiol.* 2004; 25:142–145. [PubMed: 14729545]

6. Hammersley J, Haughton V, Wang Y, del Rio AM. Tapering of the cervical spinal canal in patients with scoliosis with and without the Chiari I malformation. *AJNR Am J Neuroradiol.* 2012; 33:1752–1755. [PubMed: 22499845]
7. Heiss JD, Suffredini G, Bakhtian KD, Sarntinoranont M, Old-field EH. Normalization of hindbrain morphology after decompression of Chiari malformation Type I. Clinical article. *J Neurosurg.* 2012; 117:942–946. [PubMed: 22978540]
8. Hentschel S, Mardal KA, Løvsgren AE, Linge S, Haughton V. Characterization of cyclic CSF flow in the foramen magnum and upper cervical spinal canal with MR flow imaging and computational fluid dynamics. *AJNR Am J Neuroradiol.* 2010; 31:997–1002. [PubMed: 20223887]
9. Hirano M, Haughton V, Munoz del Rio A. Tapering of the cervical spinal canal in patients with Chiari I malformations. *AJNR Am J Neuroradiol.* 2012; 33:1326–1330. [PubMed: 22403772]
10. Iskandar BJ, Quigley M, Haughton V. Foramen magnum cerebrospinal fluid flow characteristics in children with Chiari I malformation before and after craniocervical decompression. *J Neurosurg.* 2004; 101(2 Suppl):169–178. [PubMed: 15835104]
11. Linge SO, Haughton V, Løvsgren AE, Mardal KA, Helgeland A, Langtangen HP. Effect of tonsillar herniation on cyclic CSF flow studied with computational flow analysis. *AJNR Am J Neuroradiol.* 2011; 32:1474–1481. [PubMed: 21596806]
12. Linge SO, Haughton V, Løvsgren AE, Mardal KA, Langtangen HP. CSF flow dynamics at the craniovertebral junction studied with an idealized model of the subarachnoid space and computational flow analysis. *AJNR Am J Neuroradiol.* 2010; 31:185–192. [PubMed: 19729542]
13. Loth F, Yardimci MA, Alperin N. Hydrodynamic modeling of cerebrospinal fluid motion within the spinal cavity. *J Biomech Eng.* 2001; 123:71–79. [PubMed: 11277305]
14. Mardal KA, Rutkowska G, Linge S, Haughton V. Estimation of CSF flow resistance in the upper cervical spine. *Neuroradiol J.* 2013; 26:106–110. [PubMed: 23859177]
15. Panigrahi M, Reddy BP, Reddy AK, Reddy JJM. CSF flow study in Chiari I malformation. *Childs Nerv Syst.* 2004; 20:336–340. [PubMed: 15085382]
16. Pinna G, Alessandrini F, Alfieri A, Rossi M, Bricolo A. Cerebrospinal fluid flow dynamics study in Chiari I malformation: implications for syrinx formation. *Neurosurg Focus.* 2000; 8(3):E3. [PubMed: 16676926]
17. Rutkowska G, Haughton V, Linge S, Mardal KA. Patient specific 3D simulation of cyclic CSF flow at the craniovertebral region. *AJNR Am J Neuroradiol.* 2012; 33:1756–1762. [PubMed: 22517282]
18. Shah S, Haughton V, del Río AM. CSF flow through the upper cervical spinal canal in Chiari I malformation. *AJNR Am J Neuroradiol.* 2011; 32:1149–1153. [PubMed: 21511868]
19. Sivaramakrishnan A, Alperin N, Surapaneni S, Lichtor T. Evaluating the effect of decompression surgery on cerebrospinal fluid flow and intracranial compliance in patients with Chiari malformation with magnetic resonance imaging flow studies. *Neurosurgery.* 2004; 55:1344–1351. [PubMed: 15574215]
20. Støverud KH, Langtangen HP, Haughton V, Mardal KA. CSF pressure and velocity in obstructions of the subarachnoid spaces. *Neuroradiol J.* 2013; 26:218–226. [PubMed: 23859246]
21. Wentland AL, Wieben O, Korosec FR, Haughton VM. Accuracy and reproducibility of phase-contrast MR imaging measurements for CSF flow. *AJNR Am J Neuroradiol.* 2010; 31:1331–1336. [PubMed: 20203113]
22. Yiallourou TI, Kröger JR, Stergiopulos N, Maintz D, Martin BA, Bunck AC. Comparison of 4D phase-contrast MRI flow measurements to computational fluid dynamics simulations of cerebrospinal fluid motion in the cervical spine. *PLoS ONE.* 2012; 7:e52284. [PubMed: 23284970]

Fig. 1. 3D sketches of the normal model (**left**) and the CM model (**right**) with axial sections and one sagittal section. The sketches illustrate the differences in the subarachnoid spaces in the normal model and in the one to which descended tonsils were added. The tapering of the spinal cord in the sagittal section in the CM model is a result of selecting a paramidline location for the section to better illustrate the position of the tonsil.

Fig. 2. Sketches of the 3 postoperative models with progressively larger craniovertebral decompression. An axial section at the level of the decompression and a sagittal section are shown for each model. In the 3 models, the subarachnoid space posterior to the tonsils increases progressively in anteroposterior diameter.

Fig. 3.

Local superior-inferior peak systolic velocities at 8 levels in the 5 models. The region covered extends from 1 cm above the craniovertebral junction down to 3 cm below, with equidistant spacing. Velocities increase nonlinearly from Level 1 (posterior fossa) to Level 8 (C3–4). In the Chiari model, peak velocities exceed those of the normal model at all levels; in the postoperative models, peak velocities are intermediate. In the s2 and s3 models, velocities normalize below Level 4; in the s1 model, they normalize below Level 6. Values on the x-axis represent levels.

Fig. 4.

Axial sections at Level 2 (cisterna magna) and at Level 4 (C-2) illustrating the heterogeneous flow patterns in the 5 models at peak caudad velocity. In the normal model (A), velocities are greater anterior to the cord than posterior to it and greater at Level 4 (3.26–3.51 cm/sec) than at Level 2 (1.87–2.19 cm/sec). For this study, caudad flow was assigned a negative sign and upward flow a positive sign. The CM (Chiari) model (B) had qualitatively similar heterogeneity and greater velocities (3.96–4.12 cm/sec). The postoperative models s1, s2, and s3 (C) show heterogeneous flow patterns, with velocities intermediate between the CM and the normal model. s = second.

Fig. 5.

Relative pressures shown versus level in each of the 5 models. The pressure, in Pa (1 Pa = 0.0075 torr), is referenced to Level 8 to facilitate comparison. Pressures decrease nonlinearly across the 8 equidistant levels in the normal and CM models, with steeper gradients below C2–3 than above. The CM model has higher relative pressures than the normal model in the posterior fossa and at C-1 and C-2 and equal pressures below. The postoperative models have decreased relative pressures compared with the CM model at the upper 5 levels. The model with the smallest postoperative defect (s1) had relative pressures intermediate between the CM and the normal model at levels 1 through 5. This model had steeper gradients below C3–4 and less steep gradients above. The other 2 postoperative models (s2 and s3) had lower relative pressures (at or below normal). These 2 models have higher pressures than in the normal or CM or s1 models below C-3.

TABLE 1

Superior-inferior peak pressure gradients (Level 1–Level 8)

Model	Value (Pa/m)
Chiari	897
s1	812
s2	725
s3	737
normal	783