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# What is the Pressure in Chronic Subdural Hematomas? A Prospective, Population-Based Study

Terje Sundstrøm, Christian André Helland, Mads Aarhus, and Knut Wester


## Abstract

Surgery for chronic subdural hematoma (CSDH) is performed to relieve brain displacement and high intracranial pressure (ICP). However, the intraoperative impression is often that the pressure inside the CSDH is low, despite marked clinical symptoms. We wanted to quantify the CSDH pressure and relate this to radiological and clinical characteristics. This prospective, population-based study of unilateral CSDHs was conducted over a 3-year period. CSDHs that were secondary to other conditions, re-operations, or CSDHs requiring other procedures than burr hole craniostomy under local anesthesia were excluded. Subdural pressure registration was performed via a simple manometric technique, and full compliance with a standardized protocol was mandatory. Sixty patients were included (mean age 76.2 years; for men, 77.4, and for women, 72.9). The mean pressure in the CSDHs was 15.2 cm $H_2O$ (range, 0–40) with no gender difference. Men had significantly larger volumes (mean 158.1 vs. 103.2 $cm^3$) and midline shifts (mean 1.04 vs. 0.68 cm) than did women. Large hematomas with large midline shifts had higher pressures and more often required repeat surgery. With a patient's increasing age, the volumes and midline shifts seemed to become larger, whereas the pressures became lower. We did not find an association between repeat surgery and pressure or age. Our results are generally in line with those of previous studies reporting quantitative pressure registrations. However, there are important disparities regarding methodology, not least when comparing with various subjective scales that are widely used in clinical practice. A mean subdural pressure of 15.2 cm $H_2O$ is probably within the range of a normal ICP.


**Key words:** clinical management of CNS injury; CT scanning; geriatric brain injury; head trauma; ICP

## Introduction

Chronic subdural hematoma (CSDH) is a common disorder in neurosurgical practice with an incidence of ~5 per 100.000 per year in the general population (Matsumoto et al., 1999; Santarius and Hutchinson, 2004). It is most frequently encountered in elderly people, and a substantial rise in incidence is expected to follow the progressively ageing population (Baechli et al., 2004; Santarius et al., 2009). Various surgical approaches are used, but they all have the same primary objectives: to reduce brain displacement and to alleviate what appears to be a high intracranial pressure (ICP).

It is generally assumed that the pressure within the CSDH cavity is higher than the pressure in the rest of the intracranial compartment, and that this pressure gradient is causing the symptoms. Preoperative neuroimaging, showing compression of the underlying cortex and midline shift, supports this notion, as does what is believed to be the typical intraoperative finding of a hematoma fluid that shoots out of the burr hole once the dura and the hematoma membrane are opened. However, it is also our clinical experience that there sometimes is a mismatch between the preoperative clinical and neuroimaging and the intraoperative findings, as the hematoma fluid only slowly runs out of the burr hole, apparently under very low pressure.

Several studies on CSDH have included pressure registrations (Gaab et al., 1979; Gjerris and Sørensen, 1980; Merlicco et al., 1995; Santarius et al., 2009; Tabaddor, 1979; Takeda et al., 2006; Tanaka et al., 1997). The methods used in these studies have varied, however, and the pressures have often been classified in a subjective manner such as "high", "medium", or "low". The interpretation of these data and their significance and implications for various treatment recommendations is therefore uncertain.

As a consequence of this lack of absolute measurements, we wanted to directly quantify the opening hematoma pressure, and relate this parameter to neuroimaging characteristics such as hematoma volume and architecture, and the midline shift. The significance of age with respect to these parameters was also investigated.

Department of Neurosurgery, Haukeland University Hospital, and Department of Surgical Sciences, University of Bergen Bergen, Norway.

## Methods

This prospective and population-based study was conducted in the Department of Neurosurgery at Haukeland University Hospital on the West Coast of Norway in the period from January 1, 2006 to December 31, 2008. For CSDH, our department has a catchment population of ~700.000; and ~ 60 operations for CSDH are performed each year.

Patients referred to our department and accepted for primary surgery of a symptomatic, unilateral hemispheric CSDH were consecutively recruited to the study. See Table 1 for details on inclusion and exclusion criteria. In total, 203 patients were operated on for a CSDH during the study period; 60 of these patients were eligible for inclusion and underwent successful pressure measurements in full compliance with the protocol. Several on-duty neurosurgeons performed the operations.

A standardized protocol using a simple and reproducible technique was used (Fig. 1). The patients, all operated on under local anaesthesia, were placed horizontally in the supine to lateral position with level head and body, ensuring a minimum of positional obstruction of cranial venous outflow. The head was positioned in such a way that the burr hole (14 mm) overlying the approximate center of the hematoma was at the top of the cranial vault. The dura was then punctured with a 21 G needle connected to an Optidynamic spinal fluid manometer (Mediplast AB, Sweden). Thirty seconds after the fluid column had stabilized, pressure was registered as the height from the burr hole to the fluid level in the manometer. The operation was subsequently completed according to the surgeon's own preferences (with or without postoperative drainage).

As most of the patients were referred to us on the basis of a CT scan, and as CT and MRI scans are difficult to compare for the purposes of this study, we included only patients with a preoperative CT scan in the study. Preoperative CT scans were investigated and side, architecture, and midline shift were recorded. The internal architecture of the hematoma was classified as proposed by Nakaguchi et al. into four types: homogenous, laminar, separated, and trabecular (Nakaguchi et al., 2000). The midline shift was measured as the distance from the cranial midline to the anterior attachment of the septum pellucidum. The hematoma volume was calculated using the validated ABC/2 technique (Sucu et al., 2005). We used the formula shown to give the closest estimation of the hematoma volume: [(Depth) × (Maximum length) × (Maximum width on any slice)] divided by 2.

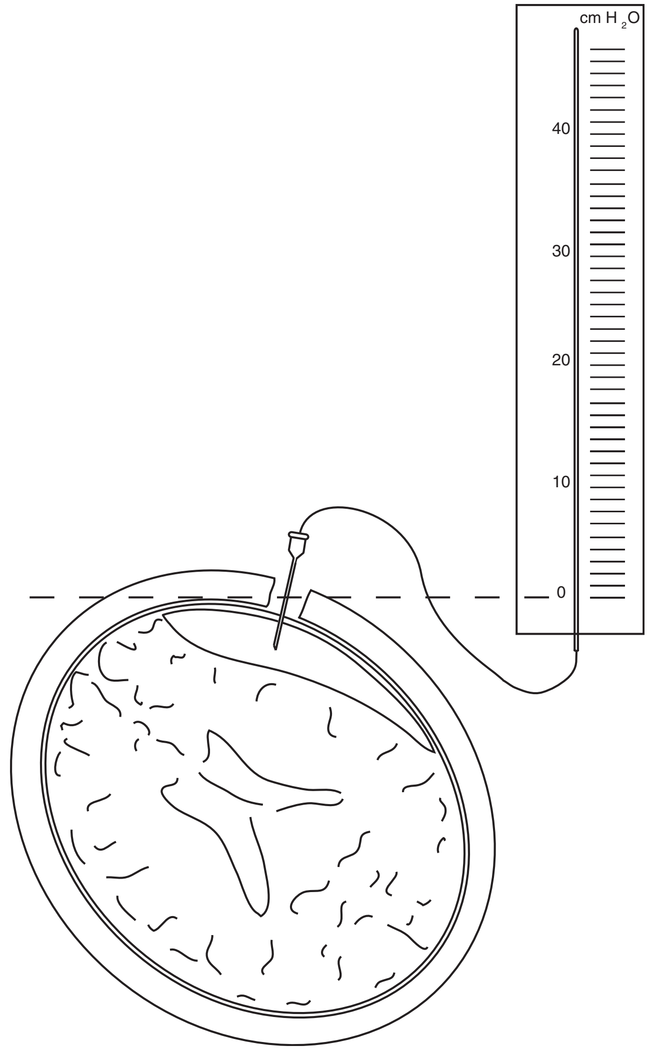


**FIG. 1.** Pressure recording procedure. Before dura puncture, the head is rotated to ensure that the burr hole is placed in the horizontal plane. At registration, the zero level of the manometer was kept in line with the burr hole. See text for details.

Table 1. Inclusion and Exclusion Criteria. All Inclusion Criteria Had To Be Fulfilled. Patients with One or More Exclusion Criterion Were Not Included in the Study. Anticoagulant Use Was Allowed

| *Inclusion criteria* | *Exclusion criteria* |
|---|---|
| Chronic subdural hematoma | Fresh bleeding |
| Unilateral | Bilateral |
| Hemispheric/convexity | 2 or more burr holes or craniotomy |
| Primary operation | Accidental dural lesion |
| Local anesthesia | Preoperative MRI (no CT scan) |
| Burr hole craniostomy (14 mm) | Subarachnoid hemorrhage |
| Standardized pressure measurement according to protocol (See Fig. 1) | Intracerebral hemorrhage |
| Preoperative CT scan | Arachnoid cyst |
| | Ventriculoperitoneal shunt |
| | Other space-occupying lesions |

The study was approved by the Regional Committee for Medical Research Ethics (REK Vest), and performed according to directives from the Norwegian Social Science Data Services (NSD). We did not have any sponsors or funding.

### Statistical analyses

Statistical analyses were conducted with SPSS version 18.0 for Mac (SPSS Inc., Chicago, IL). A probability value of $<0.05$ was considered significant. Group comparisons were done with Student's *t*-test for normally distributed continuous variables. Contingency tables were analyzed with Fisher's exact test for dichotomized variables or $\chi^2$ statistics. Pearson's correlation was used to reflect the degree of linear relationship between two variables.

## Results

A total of 60 patients were included, 44 (73.3 %) men and 16 (26.7 %) women. The mean age was 76.2 years (median 78.0 years; range, 37–97 years), for men and women 77.4 years and 72.9 years, respectively ($p = 0.18$). The CSDHs were not associated with any hemisphere (33 left, 27 right), and this applied to both genders.

### Pressure

The pressure inside the hematoma ranged from 0 to 40 cm $H_2O$, with a mean of 15.2 cm $H_2O$, and a median of 15.3 cm $H_2O$ (Table 2, Fig. 2). We found no pressure difference between left- and right-sided CSDHs or between the two genders; neither was there any significant difference between older and younger patients (Fig. 3a).

### Hematoma volume

The hematoma volume, as measured by the ABC/2 formula ranged from 53 to 264 $cm^3$, with a mean of 144 $cm^3$, and a median of 147 $cm^3$ (Table 2). Men had significantly larger volumes than women. We observed a significant correlation between age and volume ($p < 0.0001$) (Fig. 3b); older patients had larger volumes. There was no difference in hematoma volume between the two sides.

### Midline shift

The midline shift ranged from 0.3 to 1.9 cm (mean 0.94 cm, median 0.90 cm) and was significantly larger in men than in women (Table 2). There was no difference related to age (Fig. 3c).

### Correlations between hematoma pressure and hematoma volume and midline shift

Hematoma pressure correlated significantly with volume ($p = 0.03$) (Fig. 4a). Similarly, midline shift correlated significantly with hematoma pressure ($p = 0.04$) (Fig. 4b).

### Repeat surgery

A repeat surgery was performed on 12 of the 60 patients. The indication was based on the judgement of the responsible neurosurgeon, and generally included clinical worsening in addition to radiological recurrence. The hematoma volume was significantly larger in the patients who needed a second procedure (180 $cm^3$) than in the patients who did not have a repeat procedure (135 $cm^3$; $p = 0.005$). Also, the midline shift differed significantly between patients who needed a second procedure and those who did not, 1.2 cm and 0.9 cm, respectively ($p = 0.023$). The hematoma pressure, age, and amount of irrigation fluid did not differ between these two groups.

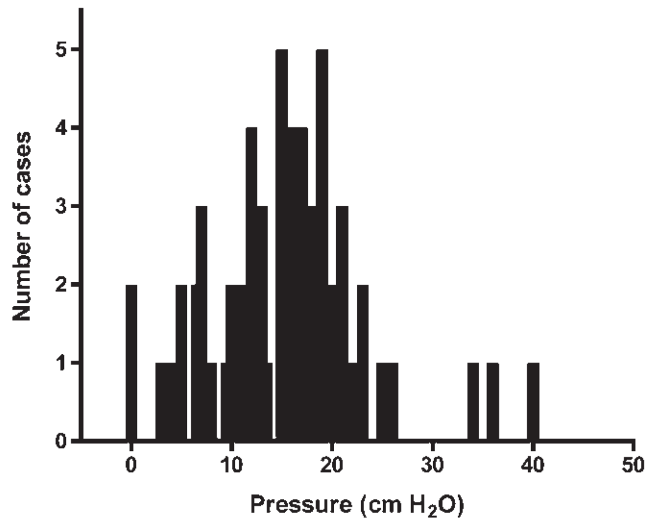


**FIG. 2.** Distribution of pressure registrations. The mean intracavitary pressure in chronic subdural hematomas was 15.2 cm $H_2O$ (median, 15.3 cm $H_2O$).

### Hematoma architecture

Fifty percent of the hematomas were homogenous, followed by trabecular (33.3 %), separated (15 %), and laminar (1.7 %) architecture. All hematoma types had an equal occurrence in the two genders. Homogenous hematomas were smaller (average volume 120 $cm^3$) than trabecular (volume 167 $cm^3$; $p = 0.01$) and separated (volume 167 $cm^3$; $p = 0.01$), and with less midline shift (0.9 cm vs. 1.0 and 1.2 cm, respectively). There was, however, no significant difference in hematoma pressure between the subtypes (homogenous 14.8 cm $H_2O$, trabecular 14.7 cm $H_2O$, separated 18.1 cm $H_2O$).

## Discussion

Our main objective with this prospective, population-based study was to directly quantify the pressures in unilateral hemispheric CSDHs. The registrations were standardized and full compliance with the protocol was mandatory for inclusion.

For the whole group, the average hematoma pressure just exceeded 15 cm $H_2O$. Although normality for ICP is poorly defined, this result is within the limits of what is considered a normal ICP (Albeck et al., 1991; Friden and Ekstedt, 1983; Minns, 1984). We find our technique to be reliable and reproducible, and our results are in line with those of previous

Table 2. Values for Volume, Midline Shift, and Hematoma Pressure

| | *Range* | *Mean* | *Median* | *Male (mean)* | *Female (mean)* | P (t-*test*) |
|---|---|---|---|---|---|---|
| Volume ($cm^3$) | 52.9–264 | 143.5 | 147.2 | 158.1 | 103.2 | $< 0.0001$ |
| Midline shift (cm) | 0.3–1.9 | 0.95 | 0.90 | 1.04 | 0.68 | = 0.001 |
| Hematoma pressure (cm $H_2O$) | 0–40 | 15.2 | 15.3 | 14.9 | 16.1 | = 0.6 |

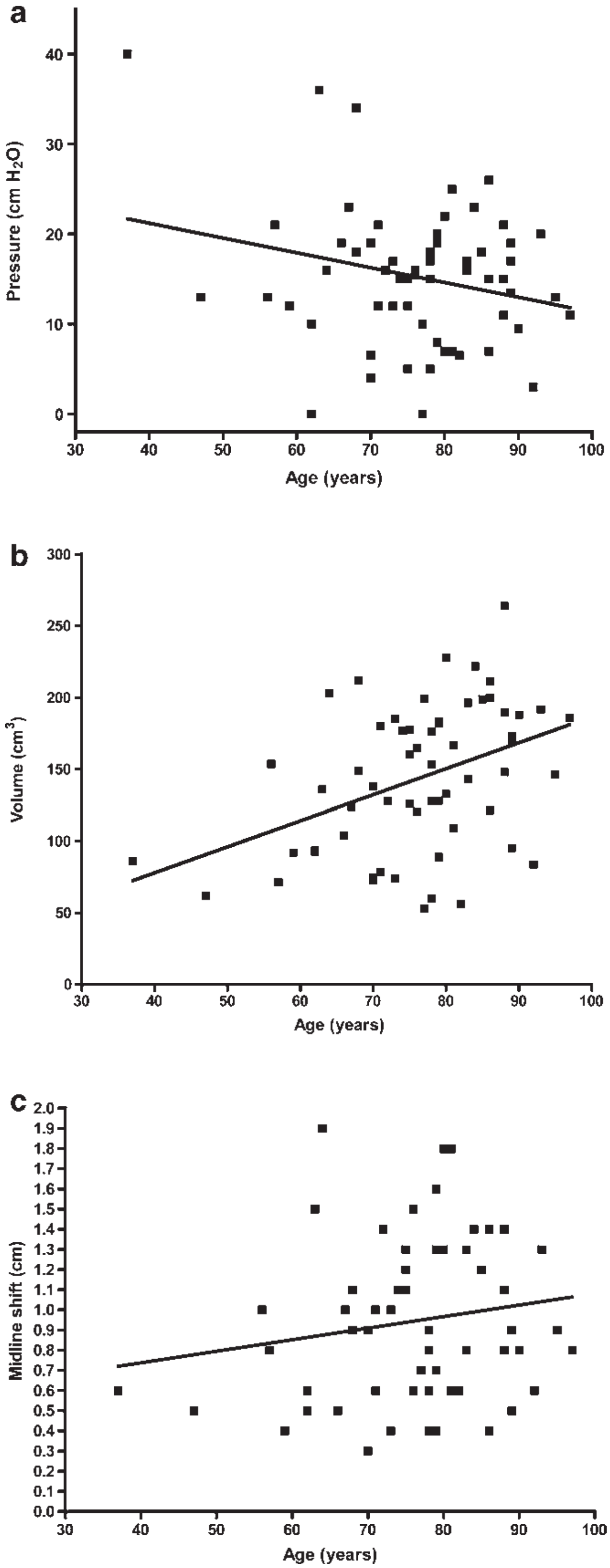


**FIG. 3.** Correlations between age and pressure, volume and midline shift. (**a**) There was no significant correlation between age and pressure. (**b**) Older patients had significantly larger hematoma volumes ($p<0.0001$). (**c**) There was no significant difference in midline shift related to age.

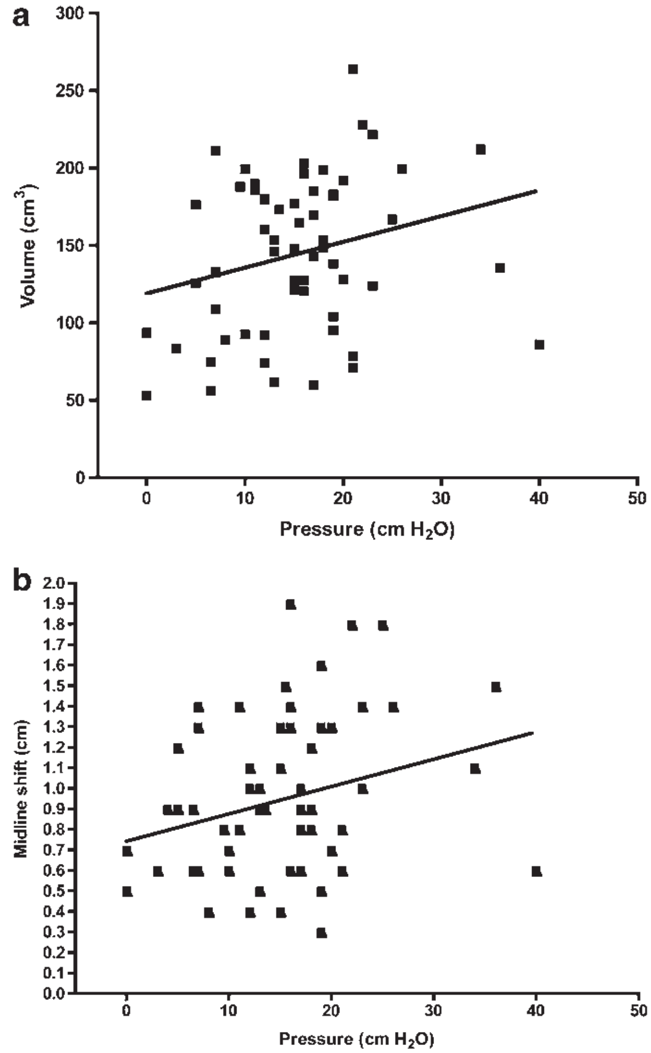


**FIG. 4.** Correlations between hematoma pressure and volume and midline shift. Both volume **(a)** and midline shift **(b)** correlated significantly with hematoma pressure, $p=0.04$ and $p=0.03$, respectively.

studies reporting quantitative pressure registrations (Gaab et al., 1979; Gjerris and Sørensen, 1980; Tabaddor, 1979; Takeda et al., 2006). The methods have, however, often varied in these previous studies and the pressures have often been classified in a subjective manner (Merlicco et al., 1995; Santarius et al., 2009), or the pressure measuring has often not been the primary aim of the studies. Therefore, the interpretation of the data from these studies, and above all their significance and implications for various treatment recommendations, remains uncertain.

Only three studies seem to present objective results that can be compared with ours; one found lower pressures (Takeda et al., 2006) and two found higher pressures (Gaab et al., 1979; Tabaddor, 1979) than we did in our patients. The discrepancies between these studies and ours may be because of the different measuring techniques used or the type of anesthesia (local or general), or that the recruitment of the patients differed. Takeda and associates measured the opening hematoma pressure using a seemingly similar technique to ours when trying out a novel therapeutic method for treating CSDH with oxygen replacement (Takeda et al., 2006). They found an average pressure of 9.21 cm $H_2O$ (range, 0–20 cm $H_2O$). Tabaddor described subdural pressures ranging between 11 and 28 mm Hg (equivalent to ~15 and 38 cm $H_2O$, respectively) (Tabaddor, 1979), and Gaab and associates noticed that despite large mass effects of CSDHs, the ICP did not exceed 25 mm Hg (~ 34 cm $H_2O$) (Gaab et al., 1979). In addition to these studies, Gjerris and Sørensen measured the intraventricular or epidural pressure on the opposite side of the hematoma prior to surgery in eight patients (Gjerris and Sørensen, 1980). The mean pressure was 22.6 mm Hg (~ 30 cm $H_2O$) ranging between 15 and 32 mm Hg (equivalent to ~ 20 and 44 cm $H_2O$, respectively). Unfortunately, they have not stated which method they used in each case. Tanaka and associates used a transducer-tipped catheter placed in the epidural space over the CSDH in 15 patients (Tanaka et al., 1997). The mean pressure before evacuation was 19.4 mm Hg (~ 26 cm $H_2O$). Devices for epidural pressure registrations are generally regarded as less accurate and reliable (Bratton et al., 2007). We therefore find the results difficult to compare with our intracavitary pressure measurements.

If the hematoma pressure is only moderately increased compared with the normal ICP, what is then the mechanism underlying the neurological deficits observed in the CSDH patients? Using a similar manometric technique as in the present study, our group has previously studied another intracranial, expansive condition: arachnoid cysts (Helland and Wester, 2007). Our observations in arachnoid cysts may have implications for the present study. We found a mean intracystic pressure of 13.1 cm $H_2O$, therefore slightly lower than the pressure measured in the CSDHs presented here. Even if the intracystic pressure did not exceed what is considered a normal ICP, the cysts caused symptoms, and these were more pronounced in patients with higher intracystic pressure than in patients with low pressure. Therefore, it appears probable that even a pressure gradient created by a moderately increased intracavitary pressure may give symptoms. Neuroimaging studies have shown that the neural tissue adjacent to an arachnoid cyst has a reduced metabolic activity that seems to normalize after decompression (De Volder et al., 1994; Horiguchi and Takeshita, 2000; Sgouros and Chapman, 2001; Tsurushima et al., 2000; Zaatreh et al., 2002).

Only a few articles have tried to investigate the cortex underlying a CSDH in a similar way. Tanaka and associates (1997) and Ishikawa and associates (1992) found that CSDHs compromised cerebral blood flow (CBF) not only on the affected side, but on the contralateral side as well, and that there was a slower CBF normalization than one would expect from the rapid clinical improvement. A hyperperfusion has also been observed immediately after a rapid decompression of CSDHs (Ogasawara et al., 2000).

Therefore, it appears likely that it is not the pressure per se that causes the neurological deficits in CSDH patients, but the fact that the pressure and/or the displacement of the affected cortex may interfere with the cerebral perfusion in the underlying cortex. We do not know how high the pressure has to be to compromise the perfusion, but from our results, it seems reasonable to believe that this may occur rather easily in an ageing, vulnerable brain.

The use of postoperative drainage has been shown to be safe and is associated with reduced recurrence rates (Santarius et al., 2009). This practice is advocated as a standard procedure. The observed pressure in a CSDH may give some useful information about the probability that the brain will expand after the surgical decompression and whether a continuous postoperative drainage is warranted or not. Patients with high subdural pressures usually show the most rapid brain expansion (Markwalder et al., 1981). It is also our clinical experience that a continuous postoperative drainage often is unnecessary when the brain expands rapidly.

Low CSDH pressure in the elderly is a known clinical phenomenon and has been associated with increased recurrence rates (Merlicco et al., 1995; Takeda et al., 2006). One could hypothesize that postoperative drainage would be more beneficial in the elderly, and especially when the pressure is low. In our study, patients with large hematomas and midline shifts more often had repeat surgery. However, we failed to demonstrate a significant association between repeat surgery and pressure or repeat surgery and age.

Men had a mean volume that was more than 50 $cm^3$ larger than in women. The midline shift was also larger in men; their mean midline shift was 0.36 cm larger than in the women in our study. Women did, however, have somewhat higher hematoma pressures, and they were also a few years younger than their male counterparts. A possible explanation for some of these disparities between the genders could be that cerebral atrophy is more pronounced in men, as they were older, therefore allowing a larger volume to be present without dramatic effect on the pressure. The following observation supports this: with increasing age, the hematoma volumes and midline shifts were larger, but the hematoma pressures were lower.

## Conclusion

One half of our patients with unilateral CSDHs had a hematoma pressure of ~15 cm $H_2O$ or below. This is probably within the range of a normal ICP. Despite the relatively low pressure, patients may develop severe neurological deficits. Affected cerebral perfusion of the cortex underlying the CSDH may explain the discrepancy between the relatively low pressure and the pronounced deficits.

Large hematomas and midline shifts were associated with higher hematoma pressures and repeat surgery. There was a tendency toward older patients having lower pressures than younger patients, despite larger volumes and midline shifts. However, we did not find an association between repeat surgery and pressure or repeat surgery and age.

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## Author Disclosure Statement

No competing financial interests exist.

## References

Albeck, M.J., Børgesen, S.E., Gjerris, F., Schmidt, J.F., and Sørensen, P.S. (1991). Intracranial pressure and cerebrospinal fluid outflow conductance in healthy subjects. J. Neurosurg. 74, 597–600.

Baechli, H., Nordmann, A., Bucher, H.C., and Gratzl, O. (2004). Demographics and prevalent risk factors of chronic subdural haematoma: results of a large single-center cohort study. Neurosurg. Rev. 27, 263–266.

Bratton, B.L., Chestnut, R.M., Ghajar, J., McConnell Hammond, F.F., Harris, O.A., Hartl, R., Manley, G.T., Nemecek, A., Newell, D.W., Rosenthal, G., Schouten, J., Shutter, L., Timmons, S.D., Ullman, J.S., Videtta, W., Wilberger, J.E., and Wright, D.W. (2007). Guidelines for the management of severe traumatic brain injury: VII - Intracranial pressure monitoring technology. J. Neurotrauma 24, S45–S54.

De Volder, A.G., Michel, C., Thauvoy, C., Willems, G., and Ferriere, G. (1994). Brain glucose utilisation in acquired childhood aphasia associated with a sylvian arachnoid cyst: recovery after shunting as demonstrated by PET. J. Neurol. Neurosurg. Psychiatr. 57, 296–300.

Friden, H.G., and Ekstedt, J. (1983). Volume/pressure relationship of the cerebrospinal space in humans. Neurosurgery 13, 351–366.

Gaab, M., Pflughaupt, K.W., Wodarz, R., and Baumgartner, U. (1979). Pathogenesis of chronic subdural haematoma: ICP, morphological, and biochemical findings. Fourth International Symposium of Intracranial Pressure. Neurosurgery 4, S476.

Gjerris, F., and Sørensen, S.C. (1980). Colloid osmotic and hydrostatic pressures in chronic subdural haematomas. Acta Neurochir. 54, 53–60.

Helland, C.A., and Wester, K. (2007). Intracystic pressure in patients with temporal arachnoid cysts: a prospective study of preoperative complaints and postoperative outcome. J. Neurol. Neurosurg. Psychiatr. 78, 1129–1135.

Horiguchi, T., and Takeshita K. (2000). Cognitive function and language of a child with an arachnoid cyst in the left frontal fossa. World J. Biol. Psychiatry 1, 159–163.

Ishikawa, T., Kawamura, S., Hadeishi, H., Suzuki, A., Yasui, N., Shishido, F., and Uemura, K. (1992). Uncoupling between CBF and oxygen metabolism in a patient with chronic subdural haematoma: case report. J. Neurol. Neurosurg. Psychiatr. 55, 401–403.

Markwalder, T.M., Steinsiepe, K.F., Rohner, M., Reichenbach, W., and Markwalder, H.J. (1981). The course of chronic subdural haematomas after burr hole craniostomy and closed-system drainage. Neurosurgery 55, 390–396.

Matsumoto, M., Sakata, Y., Yamazaki, T., Endo, G., Ohishi, H., and Takasu, N. (1999). Local coagulofibrinolysis in the postsurgical recovery of patients with chronic subdural haematoma. Acta Neurochir. (Wien) 141, 177–181.

Merlicco, G., Pierangeli, E., and di Padova, P.L. (1995). Chronic subdural hematomas in adults: prognostic factors. Analysis of 70 cases. Neurosurg. Rev. 18, 247–251.

Minns, R.A. (1984). Intracranial pressure monitoring. Arch. Dis. Child. 59, 486–488.

Nakaguchi, H., Tanishima, T., and Yoshimasu, N. (2000). Relationship between drainage catheter location and postoperative recurrence of chronic subdural hematoma after burr-hole irrigation and closed-system drainage. J. Neurosurg. 93, 791–795.

Ogasawara, K., Ogawa, A., Okuguchi, T., Kobayashi, M., Suzuki, M., and Yoshimoto, T. (2000). Postoperative hyperperfusion syndrome in elderly patients with chronic subdural hematoma. Surg. Neurol. 54, 155–159.

Santarius, T., and Hutchinson, P.J. (2004). Chronic subdural haematoma: time to rationalize treatment? Br. J. Neurosurg. 18, 328–332.

Santarius, T., Kirkpatrick, P.J., Ganesan, D., Chia, H.L., Jalloh, I., Smielewski, P., Richards, H.K., Marcus, H., Parker, R.A., Price, S.J., Kirollos R.W., Pickard J.D., and Hutchinson P.J. (2009). Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet 374, 1067–1073.

Sgouros, S., and Chapman, S. (2001). Congenital middle fossa arachnoid cysts may cause global brain ischaemia: a study with 99Tc-hexamethylpropyleneamineoxime single photon emission computerised tomography scans. Pediatr. Neurosurg. 35, 188–194.

Sucu, H.K., Gokmen, M., and Gelal, F. (2005). The value of XYZ/2 technique compared with computer-assisted volumetric analysis to estimate the volume of chronic subdural hematoma. Stroke 36, 998–1000.

Tabaddor, K. (1979). ICP and compliance in chronic subdural haematoma. Fourth International Symposium of Intracranial Pressure. Neurosurgery 4, S476.

Takeda, N., Sasaki, K., Oikawa, A., Aoki, N., and Hori, T. (2006). A new simple therapeutic method for chronic subdural hematoma without irrigation and drainage. Acta Neurochir. (Wien) 148, 541–546.

Tanaka, A., Nakayama, Y., and Yoshinaga, S. (1997). Cerebral blood flow and intracranial pressure in chronic subdural hematomas. Surg. Neurol. 47, 346–351.

Tsurushima, H., Harakuni, T., Saito, A., Tominaga, D., Hyodo, A., and Yoshii, Y. (2000). Symptomatic arachnoid cyst of the left frontal convexity presenting with memory disturbance—case report. Neurol. Med. Chir. (Tokyo) 40, 339–341.

Zaatreh, M.M., Bates, E.R., Hooper, S.R., Palmer, G., Elmenshawi, E.E., Courvoisie, H.E., and Greenwood, R.S. (2002). Morphometric and neuropsychologic studies in children with arachnoid cysts. Pediatr. Neurol. 26, 134–138.

Address correspondence to:
*Terje Sundstrøm, M.D.*
*Department of Neurosurgery*
*Haukeland University Hospital*
*N-5021 Bergen*
*Norway*

*E-mail:* terje.sundstrom@gmail.com