Working Toward Rational and Evidence-Based Treatment of Chronic Subdural Hematoma

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Chronic subdural hematoma (CSDH) is one of the most common neurosurgical conditions that can usually be treated with relatively simple and effective surgical procedures. It affects primarily the elderly, a rising population worldwide. Together with improved awareness among the medical profession and greater access to modern imaging facilities, the incidence of CSDH is set to rise significantly. Maximization of the efficiency of management of CSDH is therefore a priority. To this end, a review of the findings of clinical and laboratory research underpinning the basis of the modern management of CSDH has been carried out. This review focuses on the pathophysiology and briefly discusses the epidemiology, clinical presentation, and surgical treatments of CSDH, concluding that a one-for-all management strategy is not appropriate. Creating rational bases for the selection of an ideal treatment strategy for an individual patient should be the target. This can be achieved through better understanding of the nature of the condition through systematic basic science research, ascertaining the merits of different surgical techniques in well-designed and rigorously executed clinical trials, using advances in imaging techniques to classify CSDH (a subject not addressed here), and training in and ongoing refinement of clinical acumen and surgical skills of individual surgeons.

Chronic subdural hematoma (CSDH) is one of the most common neurosurgical conditions that can usually be treated with relatively simple and effective surgical procedures. However, its management is not always straightforward. The preferred surgical method continues to attract debate, and the time for an evidence-based approach is now overdue.

CSDHs affect primarily the elderly. According to the 2001 Report of the US Census Bureau, the proportion of people $\geq$ 65 years of age is expected to double worldwide between 2000 and 2030.[1] A corresponding rise in the incidence of CSDH is expected, particularly with a more active aging population. Improved awareness among the medical profession, together with greater access to modern imaging facilities, has improved the detection of CSDH; hence, the neurosurgical burden from CSDH is set to increase significantly.

Here, we review the findings and highlight the targets of clinical and laboratory research that underpin the basis of the modern management of CSDH. We focus on the pathophysiology and briefly discuss surgical treatments (the latter was comprehensively reviewed by Weigel et al[2]). The epidemiology and clinical presentation of CSDH are also included because they are important for understanding the nature of CSDH.

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ANATOMY: THE SUBDURAL SPACE

CSDHs are found between the dura mater and the arachnoid. Under normal conditions, however, a space or cavity does not exist at the junction between the dura and the arachnoid. Instead, a layer of cells with unique morphological features and a propensity to shear open is found at this point (Figure 1).[3-6] This layer is called the dural border cell (DBC) layer.[5,6] It is characterized by a relative paucity of tight junctions and enlarged extracellular space containing nonfilamentous, amorphous material. This layer therefore lacks strength and can easily be dissected, for example, by a surgeon during the elevation of dura or blood from shearing of a vein traversing the DBC layer.[7] The veins are anchored firmly within the arachnoid and the dural layers but less so within the DBC layer. With increasing brain atrophy, the arachnoid is pulled toward the center, whereas the dura remains attached to the skull. The resultant force stretches the DBC layer and veins traversing through it. Only a minor additional force may be required to cause shearing of a vein and leakage of blood that will further dissect the DBC layer, creating a subdural cavity. Indeed, this has been observed in experimental models.[8] Similarly, a traumatic tear of the arachnoid can cause a hygroma, which can later transform into a CSDH (see more below).

PATHOPHYSIOLOGY

Although it is likely that CSDHs were treated by ancient civilizations in many parts of the world,[9-13] the first medical description of CSDH is probably that of Johannes Wepfer[14] in

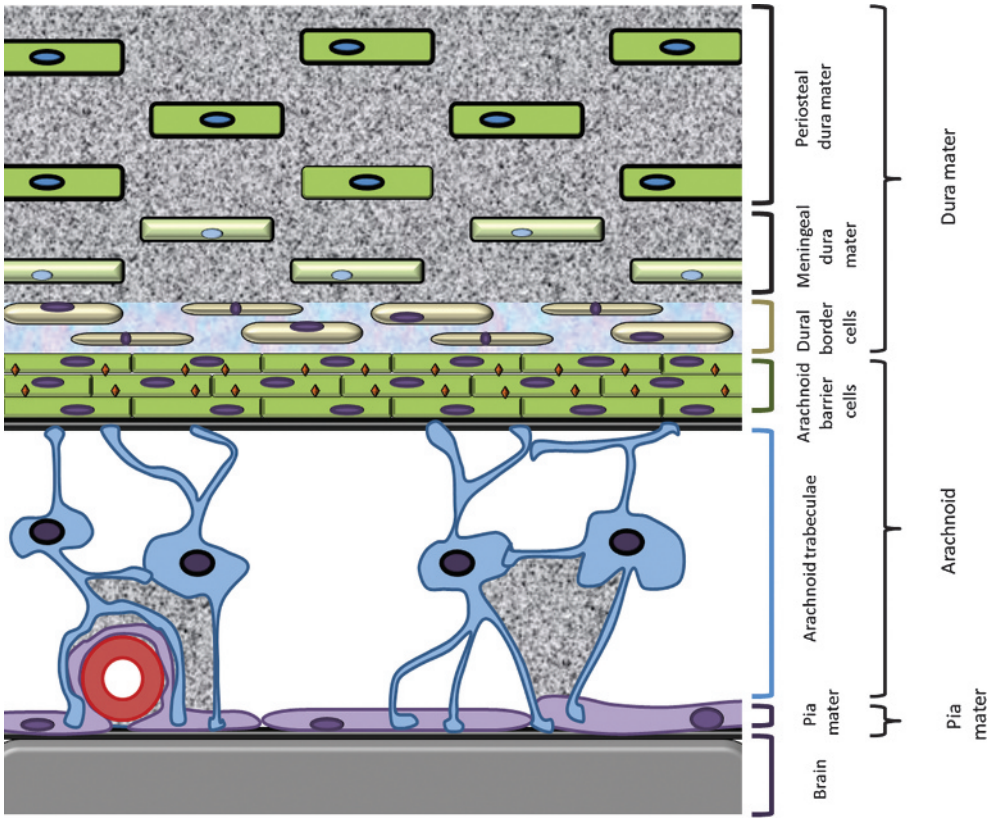

FIGURE 1. Schematic representation of the ultrastructure of the meninges (adapted from Haines et al[5]) The dura mater is composed of fibroblasts and a large amount of collagen. The arachnoid barrier cells are supported by a basement membrane and bound together by numerous tight junctions (red diamonds). The dural border cell layer (light blue) is formed by flattened fibroblasts with no tight junctions and no intercellular collagen. It is therefore a relatively loose layer positioned between the firm dura matter and arachnoid. The subdural space is a potential space that can form within the dural border cell layer.

his *Observationes Anatomicae* published in 1675. Uncertain of its nature, Wepfer described the fluid found in one of his postmortem cases as ''serum.'' In his 1857 article ''Das Hämatom der Dura mater,'' Virchov called CSDH *pachymeningitis haemorrhagica interna,* recognizing both its inflammatory and hemorrhagic elements.[15]

In 1914, Trotter[16] suggested the role of trauma to the bridging veins in the pathogenesis of the subdural hemorrhagic cyst, and Yamashima and Friede[17] later demonstrated, using electron microscopic data, that human bridging veins have thin walls of variable thickness, circumferential arrangement of collagen fibers, and a lack of outer reinforcement by arachnoid trabecules, all likely contributory factors to the subdural portion of the vein being more fragile than its subarachnoid portion.

Today, it is widely accepted that CSDHs are a result of the failure of acute subdural hematomas to heal. Indeed, transformation of acute subdural hematoma is observed by neurosurgeons in daily practice, and it has been documented in patients followed up with serial computed tomography.[18] However, in some cases, CSDH seems to have developed from an initial subdural hygroma.[19-26] Some authors maintain that, in fact, the majority of CSDHs develop from subdural hygromas rather than acute subdural hematomas.[20] Park et al[25] studied 145 cases of post-traumatic subdural hygroma of whom 13 developed into CSDH. The proportion of CSDHs arising from hygromas was even higher (6 out of 24) in a series by Yamada et al.[27] Kristof et al[28] found β-trace protein in subdural fluid in 90% of CSDHs, and β-trace protein was predictive of recurrence. It is possible that leakage of cerebrospinal fluid into the CSDH, at least in some cases, may play a role in hematoma growth.

Both blood and cerebrospinal fluid are likely to shear open the DBC layer and create a subdural collection. It is possible that hemorrhage from a blood vessel traversing the DBC occurs even in the case of hygroma as the primary

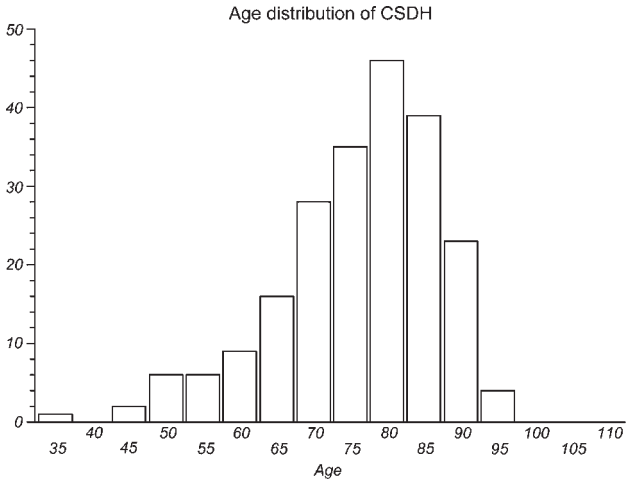

FIGURE 2. Age distribution of chronic subdural hematoma of the cohort of patients published by Santarius et al.[88]

pathology. As in other tissue injuries, this triggers a complex reparatory response that aims to heal the tissues. Whatever the primary insult, it triggers an early inflammatory reaction characterized by proliferation of the DBCs, formation of granulation tissue with collagen fibers, and deposition of macrophages.[6,29,30] New blood vessels form that supply cellular and noncellular components necessary for tissue remodeling and final healing. It is likely that such a process is successfully completed in many cases, especially in young and healthy patients with nonatrophic brain.[20,31-33] However, in a proportion of cases, the repair process fails to achieve healing, and CSDH ensues.

The CSDH consists of a subdural fluid surrounded by a membrane arising within the DBC layer.[29,30] Its visceral part is adjacent to the arachnoid and is relatively thin and avascular.[29,30] The parietal membrane is composed of multilayered tiers and clusters of cells derived from the DBCs, transfixed by multiple capillaries, with collagen fibrils and elastic fibers between them (Capillaries and collagen fibrils are absent from the normal DBC layer).[29,30]

What causes CSDH hematoma to enlarge? All surgeons have observed a great variety of subdural fluid types, ranging from bright red liquid through to thick engine oil to light, serous fluid. Similarly, some CSDHs contain a very think parietal membrane, whereas in others the membrane is hardly visible with a naked eye. Therefore, it is likely that multiple factors are responsible for the maintenance and enlargement of CSDH, the relative importance of which varies from case to case.

In 1925, Putnam and Cushing[34] proposed rebleeding from thin-walled sinusoidal blood vessels in the outer neomembrane. Since then compelling evidence for repeated hemorrhages as an important cause of for CSDH enlargement has been accumulated. Ito et al[35] infused 50 CSDH patients with 51 Cr- labeled erythrocytes and studied their concentration in a fluid obtained during craniotomy performed between 6 and 24 hours later. They estimated that the new hemorrhages accounted on average for 6.7% of the hematoma content. Similarly, in an experimental mouse model of CSDH, Aikawa et al[36] observed fresh hemorrhages surrounded by hemosiderin-laden macrophages in the outer membrane. Friede and Schachenmayr[29] reported that the loose and irregular deposition of collagen and DBCs provides little mechanical support for the sprouting new capillaries, which are fragile and bleed easily. Yamashita et al[37] studied the ultrastructure of the microcapillaries using an electron microscope. They reported that the endothelial cells have numerous large gap junctions (6-8 μm) and the basement membrane is either thin or absent, rendering the capillaries fragile and susceptible to bleeding. Moreover, erythrocytes and plasma were observed in the gap junctions, providing direct evidence of blood leakage. Murakami et al[38] identified high levels of plasma soluble thrombomodulin in the subdural fluid, an indication of an ongoing injury to the sinusoidal capillaries, thus demonstrating another potential mechanism of rebleeding. Thrombomodulin itself can inhibit coagulation by forming a complex with thrombin and activating protein C[111].

Under normal circumstances, capillary leaks would be stopped with blood clots. However, subdural and the parietal membrane are awash with profibrinolytic and anticoagulation factors. Indeed, subdural fluid was shown to accelerate fibrinolysis, a finding consistent with the clinical observation of a liquid, nonclotting contents of CSDH.[39] High levels of tissue plasminogen activator were measured in the subdural fluid and parietal membrane.[8,40-44] This enhanced fibrinolytic activity is further documented by the high concentration of fibrin degradation products in both the subdural fluid and the parietal membrane.[43,45,46] Moreover, Katano et al[42] have found tissue plasminogen activator to be predictive of recurrence, and Rughani et al[47] reported a patient with plasminogen activator inhibitor type I deficiency whose recurrent CSDH healed only after a course of aminocaproic acid. Relative reduction in the concentration of the profibrinolytic agents in the subdural fluid has frequently been used to explain the highly effective therapeutic burr hole drainage.[33,40,48,49]

In addition to coagulation factors, the role of inflammatory and growth factors in CSDH has been investigated. High concentrations of vascular endothelial growth factor (VEGF) were found in subdural fluid and high expression of the VEGF receptor subtype 1 (FLT1) in the cells of the parietal membrane.[50-52] Hohenstein et al[50] found a markedly higher level of VEGF mRNA expression in cells floating in the hematoma fluid compared with cells obtained from the outer membrane. Thus, the hematoma fluid itself appears to be a strong promoter of ongoing angiogenesis and hyperpermeability in CSDH rather than a mere reflection of an abnormally high demand for healing-associated angiogenesis. VEGF is also known to increase the permeability of capillaries and thus contribute directly to the increase in the volume of CSDH.

Weigel et al[53] analyzed 310 patients, 81 of whom were taking angiotensin-converting enzyme (ACE) as a treatment for hypertension. Hypothesizing that hyperangiogenesis plays an important role in the pathogenesis of CSDH, the authors studied retrospectively the recurrence rate of patients with and without concurrent treatment with ACE inhibitors, agents that had been shown to inhibit angiogenesis.[53] Indeed, the recurrence rate was 5% in patients taking and 18% in those not taking ACE inhibitors ($P = .003$). Moreover, the VEGF content was significantly lower ($P = .012$) in the hematomas of patients taking than those not taking ACE inhibitors.

High concentrations of various molecules known to play a role in inflammation such as platelet-activating factor, interleukin-6 and -8, and bradykinins have also been found in subdural fluid.[55-59] Because of the important role of both inflammation and angiogenesis in CSDH, corticosteroids have been proposed in the management of CSDH. Steroids inhibit

tissue plasminogen activator activity[60] and interleukin-6 and -8, and VEGF expression can be inhibited with corticosteroids.[61-63] Despite the fact that corticosteroids are used to treat CSDH, especially if conservative management is adopted, surprisingly few systematic studies of their role in CSDH have been published. Glover and Labadie[64] have shown that corticosteroids inhibit the growth CSDH membrane. Although the use of steroids in the management of CSDH has been reported.[65-71] treatment with steroids has never been compared in a meaningful way with no treatment (in the case of nonoperative management). Similarly, we are not aware of a study that provides useful guidance about the role of steroids in surgically treated CSDH. Thus far, the rationale for using steroids has largely been based on theory, and clearly, more research into their treatment of CSDH is warranted.

A once popular explanation for the maintenance and growth of CSDH, the osmotic theory, introduced by Gardner[72] in 1932 and later modified by Zollinger and Gross,[73] was largely abandoned after Weir's[74,75] 1971 and 1980 publications. The osmotic theory was based on the premise that fluid is attracted into the subdural space along the osmotic gradient created by the breakdown of blood products of the acute hematoma. However, Weir[74] compared the osmolality of the subdural hematoma fluid, venous blood, and cerebrospinal fluid and found no significant difference between them. Weir[75] also found no significant difference between oncotic pressures of the fluid from subdural hematoma and venous blood, whereas the oncotic pressure of subdural hygroma was significantly lower. Similarly, Markwalder et al[48] found that the concentration of proteins in CSDH fluid is similar to that of blood. Recently, Heula et al[76] and Sajanti and Majamaa[77] found very high concentrations of the propeptides of type I and type III protocollagens in the subdural fluid (relative to their concentrations in serum), suggesting sustained collagen synthesis. These concentrations were similar to those observed in wound fluid during the first few days after a surgical operation.[78-80] The authors suggest that the increased synthesis of the components of the extracellular matrix and their deposition in the subdural fluid may lead to the increase in its oncotic pressure.[77] This notion also seems to be in line with data suggesting that exudation plays an important role in CSDH. Fujisawa et al[81] and Tokmak et al[82] have observed a significant uptake of ^{99m}Tc-labeled human serum albumin into the subdural fluid, thus demonstrating that exudation takes place.

EPIDEMIOLOGY

The age distribution of patients with CSDH varies somewhat between published series, depending on the population from which it is derived. In a series of 2300 patients from Tivandrum, India, by Sambasivan[83] (patients treated between 1966 and 1996), the most frequent decade of presentation was 41 to 50 years. Similar to most other published series,[84-87] our recently published cohort[88] was substantially older (Figure 1). CSDH has a strong male preponderance, with a male-to-female ratio approximately 3:1.[84-87] To the best of our knowledge, a satisfactory explanation of this striking gender difference has not been published, nor has its biological basis been thoroughly investigated.

PRESENTATION

Most commonly, the presentation is subacute or chronic with gait disturbance, mental deterioration, headache, and limb weakness. In 10% to 20% of cases, patients present acutely with depressed level of consciousness. The relative frequency of presenting symptoms varies in different series, depending on the way the symptoms are categorized, the historical and cultural context, and the accuracy with which these were documented (Table 1). History of trauma can usually be elicited in 50% to 70% cases.[26,86-89]

Numerous articles have listed predisposing factors identified in the studied populations. These are listed in Table 2.

MANAGEMENT

Surgical drainage is well recognized as an effective treatment of CSDH. Drainage can be achieved via craniotomy, burr hole craniostomy (BHC; 5-30 mm in diameter according to Weigel et al[2]) or twist drill craniostomy (TDC; $<$ 5 mm in diameter). General or local anesthesia can be used, and the procedure can be performed in the operating theater or at bedside. Numerous variations of each technique have been developed and are practiced (see also recent reviews by Weigel et al[2] and Lega et al.[90]

Burr Hole Craniostomy

Probably the most widely practiced treatment is evacuation via burr holes.[83,84,86,87,89,91-93] Both the systematic review by Weigel et al[2] and the decision analysis model based on the previously published data by Lega et al[90] have identified BHC as the most efficient choice to treat an ''uncomplicated'' CSDH because it balances a low recurrence rate against morbidity and mortality better than craniotomy and TDC.

Over the last 2 decades, evidence has been emerging that the usage of drains with BHC is associated with lower recurrence rates.[84,87,89,94-98] In the review by Weigel et al,[2] the use of drains was endorsed with Type B recommendation. The results of a Monte Carlo simulation in the 2009 article by Lega et al[90] suggest a trend toward better outcomes with insertion of an in-dwelling drain. Many surgeons remain unconvinced about the role of drains in burr hole evacuation. Results of a survey commissioned by the Society of British Neurological Surgeons in 2006 showed that most neurosurgeons in the United Kingdom and the Republic of Ireland do not use drains most of the time.[93] The perceived risk, surgeons' experience of a patient with complications, and insufficient or a perception of insufficient evidence might play a part in their decision.

TABLE 1. Presentation of Patients With Chronic Subdural Hematoma

Country Year	Santarius et al[88] England 2009	Mori and Maeda[86] Japan 2001	Sambasivan[83] India 1997	Krup and Jans[141] Germany 1995	McKissock et al[142] England 1960
Average age	77	69	49[a]	65	49[a]
Series size	215	500	2300	212	216
Gait disturbance or falls	54	63	…	…	…
Mental deterioration	33	25	…	…	…
Limb weakness	33	59	…	45	22
Acute confusion	31	…	…	18	38
Headache	17	38	15	41	81
Drowsiness or coma	9	17	15	65	47
Speech impairment	6	2	…	18	6
Nonspecific deterioration	3	…	…	…	…
Collapse	1	…	…	…	…
Seizures	1	2	12	6	9
Incontinence	1	17	…	…	-
Visual disturbance	1	…	12	…	13
Vomiting	1	3	…	11	30
Vertigo	…	…	…	12	6
Strokelike symptoms[b]	…	…	29	…	…
Behavioral disturbance[b]	…	…	18	…	…

The series were selected arbitrarily to illustrate the presentation of patients with chronic subdural hematoma in different series. Only series with > 200 cases and those with clinical presentations specifically listed were considered.

[a]These series included pediatric cases.

[b]Naming and classification of symptoms and signs differed between series. They were made to fit categories used in our recent publication.[88] Only when this was not possible were the original categories retained.

Recently, we published results of a randomized controlled trial of the use of drains versus no drains after BHC for primary (nonrecurrent) CSDH in adults.[88] The primary end point was recurrence (reoperation) rate. The secondary outcomes were mortality at 30 days and 6 months and functional status (modified Rankin scale[99]) at discharge and 6 months (Table 3). At 6 months, recurrence occurred in 10 of 108 patients (9.3%) treated with a drain and 26 of 107 (24%) without a drain ($P = .003$). There was lower mortality in the drain group at both 30 days and at 6 months, although only at 6 months was the difference statistically significant (Table 3). Patients left the hospital in better functional status if they were treated with a drain, and this advantage was maintained at 6 months (Table 3). Medical and surgical complications were much the same between the study groups. In accordance with the previously published studies, the results of this trial provide strong evidence for the use of drains with BHC.

Several groups have demonstrated benefit from intraoperative irrigation.[100-103] Only Kuroki et al found > 6 times higher recurrence ($P = .49$) in cases with (5 of 45, 11.1%) than without (1 of 55, 1.8%) irrigation. Conversely, Aoki in a study of TDC (without the use of postoperative drains) found a lower recurrence rate in the cases when intraoperative irrigation was used (1 of 15 versus 7 of 24; $P = .096$). Collectively, these findings provide little support for the hypothesis that a reduction in the concentration of profibrinolytic factors in the subdural fluid is the basis for the curative effect of the drainage procedure (see above). It has to be pointed out that none of the studies cited here were sufficiently powered to demonstrate such a difference. In contrast, 2 studies in which postoperative continuous irrigation was used showed that it is associated with a lower recurrence rate.[105,106]

Most surgeons use 2 burr holes, mainly because doing so allows better washout of the subdural cavity. Taussky et al[107] have found higher recurrence rate if 1 rather than 2 burr holes was used. In contrast, in the series by Han et al,[108] the recurrence rate was 1 of 51 (2%) if 1 burr hole and 9 of 129 (7%) if 2 burr holes were used. This difference was not statistically significant ($P = .19$). Both studies were retrospective series, and a number of factors, including the decision making as to when 1 or 2 burr holes should be drilled, may have been responsible for obtaining such disparate results. If drains are used, it seems to be more advantageous to insert them via frontal burr hole because this technique was found to be associated with lower recurrence.[109,110]

Craniotomy

Until the mid-1960s, craniotomy was the prevailing technique used to evacuate CSDH.[111,112] In 1964, Svien and Gelety[112] published a series of 69 patients with primary CSDH

TABLE 2. Predisposing Factors for the Development and Recurrence of Chronic Subdural Hematoma[a]

Factor	Predisposing to Occurrence (Positive or Negative Association)	Predisposing to Recurrence (Positive or Negative Association)
Age	All articles	Positive[86,94]
Male sex	All articles	Positive[94]
History of head injury	All articles	
History of falls	Positive[133,143,144]	
Epilepsy	Positive[133]	Positive[145]
Diabetes mellitus		Negative[145]
Alcoholism	Positive[26,133,146]	Positive[147,148]
Anticoagulation/coagulopathies	Positive[133,68,146,147,149,150–154]	Positive[26,143]
		Neutral[155,156]
Antiplatelet agents	Positive[33,126,143,149,151,153,157]	
ACE inhibitors	Negative[53]	Negative[53]
Low-ICP states	Positive[33,158–162]	
High density on preoperative CT		Positive[163]
Width of hematoma on preoperative CT		Positive[145]
Midline shift > 5 mm on preoperative CT		Positive[164]
Multiplicity of hematoma cavities on preoperative CT		Negative[145]
Homogeneous type on preoperative CT		Negative[164]
Homogeneous type on preoperative CT		Negative[165]
Separated type on preoperative CT		Positive[56,164,165]
Evidence of cerebral infarction on preoperative CT		Positive[86,166]
Brain atrophy	Positive[48,159,167]	Positive[166,168]
Subdural hygroma	Positive[20,21,23,169]	
Poor brain expansion	NA	Positive[26,86,89,135,164]
Thick subdural membrane		Positive[89]
Preoperative irrigation		Positive[104,170]
Postoperative irrigation		Negative[105]
Subtemporal marsupialization		Negative[83]
Placement of subdural drains		Negative[84,88,89,94,96–98]
Placement of subgaleal/subperiosteal drains		Negative[87,95]
Frontal position of drain		Negative[109,171]
Postoperative bed rest		Negative1[40]
		Neutral1[39]
Postoperative air on CT		110,86,94,148,163,164,168,170,172,173
High postoperative volume on CT		Positive[171]
Higher GOS at discharge		Positive[163]
High levels of β-trace protein in subdural fluid		Positive[28]
High concentration of tPA in subdural fluid		Positive[42]

[a]ACE, angiotensin-converting enzyme; CT, computed tomography; GOS, Glasgow Outcome score; ICP, intracranial pressure; tPA, tissue plasminogen activator. This table lists factors that have been reported in the literature as associated with or predictive of development or recurrence of CSDH. There was a great variation in the quality of evidence for the role of individual factors. Whenever possible, we tried to preserve the original description of each factor. This resulted in a degree of redundancy because some listed factors represent different aspects or merely different description of the same phenomenon (ie, brain atrophy, age, width of hematoma, evidence of infarct on the CT, lack of preoperative or postoperative brain expansion, high postoperative volume on CT).

treated with either craniotomy or BHC. They found that the recurrence rate and functional outcome were better in patients treated with BHC than those who received craniotomy. Although the size of the series is rather small and the study falls short of today's expected methodological rigor, numerous prospective and retrospective case series that followed and 2 meta-analyses have confirmed the findings of Svien and Gekety.[2,90] Nevertheless, many surgeons use minicraniotomies as the method of choice for evacuation of CSDH, and good results with the use of craniotomy, in terms of both recurrence and functional outcome, have been reported by a number of teams.[111,113-115] Craniotomy and BHC have not been compared in an appropriately powered clinical trial. The results of the study by Tanikawa et al[116] suggest that hematomas with multilayer structure of hematoma membranes demonstrated by T2*-weighted magnetic resonance imaging are more

TABLE 3. Summary of Results of the Cambridge Chronic Subdural Haematoma Trial[88]

	Drain Group, n (%)	Nondrain Group, n (%)	*P*
Recurrence rate at 6 mo	10/108 (9.3)	26/107 (24)	.003
Mortality at 30 d	4/106 (3.7)	8/105 (7.6)	.191
Mortality at 6 mo	9/105 (8.6)	19/105 (18.1)	.042
Favorable modified Rankin scale at discharge	81/97 (84)	64/95 (67)	.009
Favorable modified Rankin scale at 6 mo	64/76 (84)	60/85 (71)	.040

effectively treated with craniotomy than BHC. Future studies of imaging in the selection of the optimal surgical technique will likely define the role of craniotomy in the management of CSDH. In the meantime, most surgeons would agree with the statement from Markwalder[33] in a 1981 review that craniotomy should be considered in those instances in which the subdural collection reaccumulates, there is solid hematoma, or the brain fails to expand and obliterate the subdural space.

TDC

In a recent paper, Rughani et al[117] reviewed the literature concerning TDC. The authors identified 16 articles published in English.[104,118-132] Weigel et al[2] have found that although the morbidity and mortality of TDC were similar to those of BHC, the recurrence rate was significantly greater than that of either BHC or craniotomy. The main advantage of TDC, however, is the possibility of performing it at bedside, which may be a consideration as the costs of operating theater time continue to rise.

Conservative Management

Our recent survey found that conservative management is rarely practiced in the United Kingdom and the Republic of Ireland.[93] Poorer outcome and prolonged hospital stay associated with conservative management are the main reasons for not pursuing this treatment option more often.[133-136] However, numerous cases of spontaneous resorption have been published,[137,138] and Delgado-López et al[67] reported nonsurgical (dexamethasone-based) healing of up to two-thirds of 101 carefully selected CSDHs. Because surgical treatment brings about rapid improvement in patients' clinical condition, overall, operative management is the treatment of choice, and conservative management tends to be reserved for patients who either are asymptomatic or have a high perceived operative risk.

Adjuvant Treatment

Approximately 55% of surgeons in Canada and the British Isles prescribe postoperative bed rest after the evacuation of CSDH.[91,93] This lack of consensus is consistent with the status of the available evidence. In a randomized trial, Nakajima et al[139] prospectively divided 46 patients into 2 groups of 23 patients. One group was kept flat for 3 days, and the other was allowed to sit up. The recurrence rates were 14.3% and 16%, respectively, and the difference was not significant. Abouzari et al[140] recently conducted a similar prospective randomized study with a 3-month follow-up and greater statistical power (84 patients). The recurrence rate was 2.3% in the supine group and 19% in the sitting group. There were significant differences in the incidence of atelectasis, pneumonia, bedsores, and deep venous thrombosis between the groups. These data suggest that there is a role for postoperative bed rest, but it is unclear whether the same reduction in risk of recurrence could be obtained with < 3 days of bed rest, as currently practiced by 93% of British and 99% of Canadian neurosurgeons.[91,93]

The potential role of corticosteroids and ACE inhibitors in the management of CSDH is discussed in the Pathophysiology section.

SUMMARY

Surgical drainage is a relatively safe and effective treatment for CSDH. Class II evidence exists for BHC being the treatment of choice for an uncomplicated primary CSDH. Together with previously published literature, our recent randomized controlled trial provides Class I evidence for the use of a drain with BHC. Craniotomy and TDC also play a role in the management of CSDH, but more clinical research is needed to refine their specific indications. Many other technical variations have been described, but they have largely been driven by tradition, hypotheses, personal or departmental experience, or case series. A similar level of evidence exists for nonsurgical management that, most would agree, is best reserved for patients who either are asymptomatic or have a high perceived operative risk. In addition to steroids, ACE inhibitors may also play a role in the management of CSDH.

A one-for-all management strategy is clearly not appropriate. Creating rational bases for the selection of an ideal treatment strategy for an individual patient should, in our opinion, be one of the targets in the process of improving the management of patients with CSDH. This can be achieved through better understanding of the nature of the condition through systematic basic science research, ascertaining the merits of different surgical techniques in well-designed and rigorously executed clinical trials, using advances in imaging techniques to classify CSDH (a subject not addressed here), and training and ongoing refinement of clinical acumen and surgical skills of individual surgeons.

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