

# The surgical management of chronic subdural hematoma

Andrew F. Ducruet · Bartosz T. Grobelny · Brad E. Zacharia · Zachary L. Hickman ·  
Peter L. DeRosa · Kristen Anderson · Eric Sussman · Austin Carpenter ·  
E. Sander Connolly Jr.

Received: 3 December 2010 / Revised: 2 April 2011 / Accepted: 15 May 2011 / Published online: 10 September 2011  
© Springer-Verlag 2011

**Abstract** Chronic subdural hematoma (cSDH) is an increasingly common neurological disease process. Despite the wide prevalence of cSDH, there remains a lack of consensus regarding numerous aspects of its clinical management. We provide an overview of the epidemiology and pathophysiology of cSDH and discuss several controversial management issues, including the timing of post-operative resumption of anticoagulant medications, the effectiveness of anti-epileptic prophylaxis, protocols for mobilization following evacuation of cSDH, as well as the comparative effectiveness of the various techniques of surgical evacuation. A PubMed search was carried out through October 19, 2010 using the following keywords: “subdural hematoma”, “craniotomy”, “burr-hole”, “management”, “anticoagulation”, “seizure prophylaxis”, “antiplatelet”, “mobilization”, and “surgical evacuation”, alone and in combination. Relevant articles were identified and back-referenced to yield additional papers. A meta-analysis was then performed comparing the efficacy and complications associated with the various methods of cSDH evacuation. There is general agreement that significant coagulopathy should be reversed expeditiously in patients presenting with cSDH. Although protocols for gradual resumption of anti-coagulation for prophylaxis of venous thrombosis may be derived from guidelines for other neurosurgical procedures, further prospective study is necessary to determine the optimal time to restart full-

dose anti-coagulation in the setting of recently drained cSDH. There is also conflicting evidence to support seizure prophylaxis in patients with cSDH, although the existing literature supports prophylaxis in patients who are at a higher risk for seizures. The published data regarding surgical technique for cSDH supports primary twist drill craniostomy (TDC) drainage at the bedside for patients who are high-risk surgical candidates with non-septated cSDH and craniotomy as a first-line evacuation technique for cSDH with significant membranes. Larger prospective studies addressing these aspects of cSDH management are necessary to establish definitive recommendations.

**Keywords** Subdural hematoma · Chronic · Anticoagulation · Antiplatelet · Antiepileptic · Burr-hole · Craniotomy

## Introduction

As the average age of the population in Western countries continues to rise, chronic subdural hematoma (cSDH) has become an increasingly prevalent neurological disease encountered by a wide variety of both general and specialized health-care practitioners. The incidence of cSDH increases greatly with age and ranges from approximately 3.4 per 100,000 in patients younger than 65 years of age, to 8 to 58 per 100,000 in those older than 65 years [4, 7, 12]. This age dependence is particularly relevant, as the latter population accounted for 35.9 million people in the United States or 12% of the overall population in 2003 [34], a number which is projected to rise to 72 million people (an estimated 20% of the overall population) by the year 2030 [34]. In fact, the incidence of cSDH in the United States is expected to double in slightly over 25 years, with

A. F. Ducruet (✉) · B. T. Grobelny · B. E. Zacharia ·  
Z. L. Hickman · P. L. DeRosa · K. Anderson · E. Sussman ·  
A. Carpenter · E. S. Connolly Jr.  
Department of Neurological Surgery, Columbia University,  
College of Physicians & Surgeons,  
710 West 168th Street,  
New York, NY 10032, USA  
e-mail: afd12@columbia.edu

similar trends worldwide. The increasing incidence of this disease underscores the need for an improved understanding of both the pathophysiology of cSDH as well as the various available treatment options [9]. Although the treatment goals for cSDH are well-established, several important aspects of clinical management remain controversial. To this end, we review the existing literature addressing the epidemiology, pathophysiology, and current treatment techniques for cSDH evacuation.

## Methods

An inclusive PubMed search was performed through October 19, 2010 using the following keywords: “subdural hematoma”, “craniotomy”, “burr-hole”, “management”, “anticoagulation”, “antiplatelet”, “seizure prophylaxis”, “mobilization”, and “surgical evacuation”; alone and in combination. Relevant articles were identified and back-referenced to yield additional papers. We also included previously unpublished data from our institution's retrospective series of patients with cSDH who underwent surgical evacuation. In this series, good outcome was defined as a discharge modified Rankin Score (mRS) of 2 or better.

Our initial search yielded 85 references. Of these studies, 45 specifically reported complications, mortality, recurrence and reoperation, good outcome, and neurological improvement rates according to the method of surgical intervention employed. These studies were included in our meta-analysis (Table 1), with the patients grouped by outcome and surgical technique. The range and mean percentage of patients fulfilling the outcome in each group were calculated. Fisher's exact test was used to evaluate differences between groups. *P* values of <0.05 were considered significant. All analyses were carried out using JMP 8.0.2 statistical software (SAS, 2009).

## Pathophysiology of cSDH

A subdural hematoma (SDH) occurs by the accumulation of hematoma in the subdural space. Paradoxically, a true subdural space does not exist at baseline in healthy individuals. Rather, the dura and arachnoid meningeal layers are tethered together by a distinct layer of dural border cells [27, 28]. Following head trauma, hemorrhage into this potential space may produce an acute subdural hematoma (SDH). Most commonly, SDH occurs due to tearing of the bridging veins draining the surface of the brain into the dural sinuses [92]; however, less commonly (20–30% of cases), SDH may result from arterial rupture [20, 33, 54]. Patients with extensive brain atrophy, such as

the elderly or alcoholics, are particularly vulnerable to developing SDH, due to increased baseline stretch of the bridging vessels. Patients maintained on chronic anticoagulation are also at increased risk for SDH [75]. Although the mechanism for this increased risk is incompletely understood, it has been suggested that contained, asymptomatic “microbleeds” are common in elderly people and that anticoagulants impair the ability to control these bleeds, allowing progression to symptomatic hemorrhage [32, 72].

cSDH may evolve from a prior traumatic acute SDH, which is often asymptomatic, through a series of distinct pathologic processes [49, 50]. Following the initial trauma and development of a subdural space hemorrhage, fibrin deposition occurs. This is followed by organization, enzymatic fibrinolysis, and liquefaction of the subdural blood clot [48]. The ensuing subdural collection triggers an inflammatory reaction, resulting in dural collagen synthesis and fibroblast spread over the inner surface of the dura to form a thick outer hematoma membrane. This process, as well as the formation of extensive subdural neo-membranes characterized by in-growth of fragile neo-capillaries, occurs over approximately 2 weeks [13, 55, 77]. In the longer term, fibrous overgrowth continues and these neo-membranes may calcify [18, 55].

Alternatively, cSDH may develop following hemorrhage into a subdural hygroma. A subdural hygroma develops at the site of lowest pressure in the cranial cavity and begins by separating the dura-arachnoid junction [49]. In patients with severe baseline atrophy, hygromas develop passively through an accumulation of cerebrospinal fluid. The development of a hygroma, much like a hematoma, induces the formation of neo-membranes accompanied by neo-vascularization, and these fragile vessels promote repeated microhemorrhage and eventual development of cSDH [48].

cSDH tend to gradually enlarge; however, the previously accepted hypothesis that expansion occurs secondary to osmotic gradients has been disproven [92, 96]. Rather, hematoma expansion is thought to result from repeated micro-hemorrhage of fragile neo-membranes [48]. cSDH expansion, either acute or gradual, may lead to clinical signs and symptoms from increased intracranial pressure or direct compression of vital neurologic structures. The presenting symptoms associated with cSDH are protean. Patients may present with headaches, light-headedness, cognitive impairment, apathy, somnolence, focal neurologic deficits, or seizures. In addition to this varied presentation, clinical recognition and diagnosis is hindered by the fact that symptoms may not become evident until weeks following the initial injury, and the inciting trauma may be so minor as to go unnoticed.

Definitive diagnosis of cSDH relies on computed tomography (CT) imaging of the head [25]. Using this

**Table 1** Outcomes for surgical treatment of cSDH, individual studies

| Author                 | Year | <i>n</i> | Design | Twist drill |    |    |   | Burr hole |       |       |    | Craniotomy |    |    |    |
|------------------------|------|----------|--------|-------------|----|----|---|-----------|-------|-------|----|------------|----|----|----|
|                        |      |          |        | I/G         | Re | C  | M | I/G       | Re    | C     | M  | I/G        | Re | C  | M  |
| Richter                | 1984 | 120      | R      |             |    |    |   |           |       | 89    | 4  |            |    |    |    |
| Robinson               | 1984 | 133      | R      |             |    |    |   | 85        |       |       | 2  |            |    |    |    |
| Camel                  | 1986 | 114      | R      | 86          | 11 |    | 8 |           |       |       |    |            |    |    |    |
| Wakai <sup>a</sup>     | 1990 | 38       | P      |             |    |    |   |           | 5–33  |       |    |            |    |    |    |
| Hamilton <sup>b</sup>  | 1993 | 92       | R      |             |    |    |   | 86        |       | 15    |    | 86         |    | 15 |    |
| Benzei                 | 1994 | 111      | R      |             |    |    |   | 90        | 11    |       |    |            |    |    |    |
| Smely                  | 1997 | 66       | R      |             | 18 | 0  | 6 |           | 33    | 18    | 9  |            |    |    |    |
| Ernestus               | 1997 | 104      | R      |             |    |    |   | 72        | 19    |       |    | 70         | 13 |    |    |
| Beatty                 | 1999 | 23       | R      |             |    |    |   |           |       |       |    | 91         | 0  |    | 9  |
| Hennig                 | 1999 | 77       | R      |             |    |    |   |           | 3–33  |       |    |            |    |    |    |
| Matsumoto              | 1999 | 121      | R      |             |    |    |   |           | 8     |       |    |            |    |    |    |
| Reinges                | 2000 | 118      | P      | 67          |    |    |   |           |       |       |    |            |    |    |    |
| Nakaguchi              | 2000 | 135      | R      |             |    |    |   |           | 13    |       |    |            |    |    |    |
| Kwon                   | 2000 | 175      | R      |             |    |    |   |           | 4     |       |    |            |    |    |    |
| Williams <sup>a</sup>  | 2001 | 62       | R      | 36          | 64 |    |   | 84–93     | 7–11  |       |    |            |    |    |    |
| Tanikawa               | 2001 | 49       | R      |             |    |    |   |           | 12    |       | 3  |            | 0  |    | 0  |
| Oishi                  | 2001 | 116      | R      |             |    |    |   |           | 9     |       |    |            |    |    |    |
| Mori                   | 2001 | 500      | R      |             |    |    |   | 89        | 10    | 5     | 1  |            |    |    |    |
| Rohde                  | 2002 | 376      | R      |             |    |    |   | 69        | 32    | 16–21 | 13 |            |    |    |    |
| Okada                  | 2002 | 40       | R      |             |    |    |   |           | 5–25  |       |    |            |    |    |    |
| Lee <sup>c</sup>       | 2004 | 172      | R      |             |    |    |   |           | 16–18 |       | 5  |            | 23 |    | 5  |
| Muzii                  | 2005 | 47       | P      |             | 33 |    |   |           | 68    |       |    |            |    |    |    |
| Gelabert-Gonzales      | 2005 | 1,000    | R      |             |    |    |   |           | 6     | 14    | 0  |            |    |    |    |
| Stanisic               | 2005 | 99       | R      |             |    |    |   |           | 15    |       |    |            |    |    |    |
| Horn <sup>b</sup>      | 2006 | 79       | P      | 84          | 18 | 18 | 7 |           |       |       |    | 74         | 8  | 25 | 13 |
| Gurelik                | 2007 | 80       | P      |             | 11 |    |   |           | 19    |       |    |            |    |    |    |
| Santos-Ditto           | 2007 | 213      | R      |             | 8  | 9  |   |           |       |       |    |            |    |    |    |
| Abouzari               | 2007 | 84       | P      |             |    |    |   |           | 2–19  |       |    |            |    |    |    |
| Kiyamaz                | 2007 | 50       | R      |             |    |    |   |           | 7–29  |       |    |            |    |    |    |
| Gokmen                 | 2008 | 70       | P      |             | 3  |    |   |           | 5     |       |    |            |    |    |    |
| Ramnarayan             | 2008 | 42       | R      | 88          |    |    | 5 |           |       |       |    |            |    |    |    |
| Taussky <sup>c</sup>   | 2008 | 76       | R      |             |    |    |   |           | 5–29  | 0–9   |    |            |    |    |    |
| Zakaria                | 2008 | 82       | R      |             |    |    |   | 83–88     | 10–14 | 6     | 0  |            |    |    |    |
| Torihashi              | 2008 | 337      | R      |             |    |    |   |           | 18    |       |    |            |    |    |    |
| Lindvall               | 2009 | 71       | R      |             |    |    |   |           | 17    |       |    |            | 14 |    |    |
| Mondorf                | 2009 | 193      | R      |             |    |    |   | 64        | 14    |       | 2  | 41         | 28 |    | 5  |
| Zumofen                | 2009 | 147      | R      |             |    |    |   |           | 13    | 11    | 3  |            |    |    |    |
| Han <sup>c</sup>       | 2009 | 180      | R      |             |    |    |   |           | 6     |       |    |            |    |    |    |
| Yu                     | 2009 | 97       | R      |             |    |    |   |           | 7     |       |    |            |    |    |    |
| Santarius <sup>a</sup> | 2009 | 205      | P      |             |    |    |   | 72        | 17    |       | 13 |            |    |    |    |
| Rughani                | 2010 | 42       | R      |             | 26 |    | 7 |           | 15    |       | 4  |            |    |    |    |
| Lega                   | 2010 | 129      | R      |             |    |    |   |           | 9     | 3     | 9  |            |    |    |    |
| Kansai <sup>c</sup>    | 2010 | 267      | R      |             |    |    |   |           | 12    |       |    |            |    |    |    |
| White                  | 2010 | 205      | R      |             |    |    |   | 83        | 18    |       | 8  | 73         | 20 |    | 17 |
| Ducruet <sup>d</sup>   | –    | 77       | R      |             |    |    |   | 58        |       |       | 1  |            |    |    |    |

*R* retrospective design, *P* prospective design, *I/G* clinical improvement or good outcome (%), *Re* recurrence (%), *C* complications (%), *M* mortality (%)

<sup>a</sup> Burr hole with or without drain

<sup>b</sup> Burr hole and craniotomy group combined

<sup>c</sup> One or two burr holes

<sup>d</sup> The authors' unpublished series

modality, cSDH appear as a crescent-shaped, isodense, or hypodense hemispheric collection layered over the cerebral convexity (Fig. 1). Associated membranes may enhance with intravenous contrast, which can be useful in preoperative planning [70]. The radiographic characteristics of cSDH have been previously extensively reviewed [40, 41, 99].

### Treatment of anti-coagulated patients presenting with cSDH

One issue frequently faced in the management of cSDH involves the treatment of the anti-coagulated patient. This is increasingly common, as a large proportion of the elderly population are chronically anti-coagulated, and the incidence of cSDH has risen significantly with the increased utilization of anticoagulant therapy [9]. According to a recent retrospective review, anticoagulants significantly increase the risk of developing cSDH (up to 42.5 times) relative to the general population [75]. In another study, 41% of all cSDH patients admitted to a neurosurgical department in Switzerland were chronically anti-coagulated [5].



**Fig. 1** Representative CT scan image of a right frontal cSDH with mass effect suggested by effacement of underlying sulci and midline shift

### Reversal of anticoagulation

In addition to increasing the risk of developing cSDH, coagulopathy complicates the treatment of this disease. In our own series of patients with cSDH ( $n=88$ ), those presenting on anticoagulant medications exhibited a significantly longer median hospital stay (11 vs. 7.5,  $p=0.040$ ). However, the proportion of patients experiencing a good discharge outcome ( $mRS \geq 2$ ) did not differ between patients who presented on anticoagulation and those who did not (50.0% vs. 60.7%) [unpublished results]. There exists a consensus that patients presenting with symptomatic cSDH while on anticoagulant medications require rapid reversal of anticoagulation in order to prevent hemorrhage expansion and to facilitate potential neurosurgical intervention [32]. Even in patients with compelling indications for anti-coagulation—such as patients with prosthetic heart valves—reversing anticoagulation is still necessary, as arresting intracranial bleeding is paramount.

The most common anticoagulant medication implicated in the development of cSDH is orally administered warfarin [2]. Reversal of warfarin prior to surgery has traditionally been accomplished through the use of fresh frozen plasma (FFP) transfusion, which replenishes all coagulation factors. However, the required volume of FFP necessary to reverse warfarin can promote fluid overload in cSDH patients, as these patients often present with impaired renal or cardiac function [46]. Alternative to FFP for the reversal of warfarin include prothrombin complex concentrate, which permits complete reversal with a bolus infusion over a period of three minutes [93]. Another emerging alternative for the reversal of anticoagulation is recombinant Factor VIIa (rFVIIa). We have found this agent to be helpful in reversal of intraoperative coagulopathy that proves recalcitrant to traditional methods of reversal. However, the role of rFVIIa remains unclear given the known side-effects of administration coupled with its extremely high cost [51, 56, 57].

Given the potential for adverse thrombotic events associated with reversal of coagulopathy [43, 93], a more gradual reversal technique, such as the administration of vitamin K, can be employed in cases where immediate reversal is not critical, such as patients in whom initial conservative management is planned [32]. This is accomplished by administering up to 10 mg of vitamin K via slow intravenous infusion, followed by oral supplementation of 1 or 2 mg [2].

### Timing of resumption of anticoagulant therapy

The timing of resumption of anticoagulation therapy, particularly following surgical intervention for cSDH, poses a significant management dilemma. Caution must be

exercised to balance the increased risk of thromboembolic complications due to prolonged discontinuation of anticoagulation against the increased risk of hemorrhage if anticoagulation therapy is reinstituted promptly after surgical intervention. Definitive recommendations are lacking in the literature, and only a few retrospective studies have addressed this issue. These studies reveal that 3–33% of hematomas recurred post-operatively in patients initially taking anticoagulant medications [17, 23, 42, 84, 90], relative to 3–15% of patients who were not anti-coagulated on presentation [17, 23, 84, 90]. In those patients whose anticoagulant medications were restarted post-operatively, recurrence rates ranged from 6% to 10% [23, 42], while in those who were not restarted, the rate was 22% [23]. Additionally, the rates of thrombotic complications for patients off anticoagulation (0–10%) did not vary significantly from patients without baseline anticoagulation requirements [23, 42]. It is difficult to determine the true incidence of thrombotic events in this patient population, as only 34 patients required chronic anticoagulation in the study [23]. Furthermore, none of the comparisons reported in these studies were statistically significant.

These investigations suggest that cSDH patients who undergo reversal of anticoagulation prior to surgical treatment do not experience additional post-operative morbidity relative to patients without a requirement for anticoagulation and that those patients in whom anticoagulation is resumed following surgical intervention do not experience increased rates of recurrence [42, 84]. Similar data have derived from studies of anticoagulation reversal and resumption in patients on oral antithrombotics who underwent surgical evacuation for related hemorrhagic processes. In a study of 27 patients suffering from warfarin-related intracerebral hemorrhage, Kawamata et al. [42] found that early resumption of anticoagulant therapy (within 3 days) did not result in intracranial rebleeding in any post-operative patient. The authors recommend resuming anticoagulants after an interval of 3 days in those patients with mechanical heart valves.

The question of when to restart anticoagulation following surgical drainage of a cSDH remains a matter of debate. The American College of Chest Physicians provides evidence-based clinical practice guidelines regarding protocols for interruption and resumption of anticoagulant therapy in both low- and high-risk patients [35]. These authors recommend optimal use of intermittent pneumatic compression devices combined with a pharmacologic method (low-dose unfractionated heparin or low-molecular weight heparin) for high-risk post-operative neurosurgical patients. These pharmacologic strategies are recommended to begin from 12 to 24 h post-surgery and when there is “adequate hemostasis”. The recommendations for the resumption of full-dose anticoagulation in high-risk patients

involve considering the anticipated bleeding risk and adequacy of post-operative hemostasis in individual patients, instead of resuming at a fixed time in all patients. The practice at our institution generally follows this philosophy. We generally favor resumption of venous thrombo-embolism prophylactic-dose anticoagulation following removal of the closed system drainage catheter, provided that the follow-up CT scan reveals no significant recurrent or acute hemorrhage. However, in terms of the resumption of full-dose anticoagulants, we believe that every case should represent an individually tailored clinical decision and advocate for the neurosurgeon to assess the hemostatic function of the patient during the operation to make a gross estimation of the appropriate length of time that anticoagulant drugs should be withheld to prevent recurrence [15]. Risks and benefits of anticoagulation are weighed with particular attention to the patient's reason for anticoagulation, history of thrombosis, coagulation function, serial radiographic evaluation of the cSDH after surgical intervention, and clinical examination.

A larger, prospective study that examines SDH recurrence as well as thrombotic events is warranted in order to establish more definitive conclusions. If varying surgical treatments are employed, separate analyses should be undertaken for each treatment group. In these patients, there should also be a standard protocol applied to reverse anticoagulation prior to surgery, and laboratory values should be followed to determine coagulation status.

### Antiplatelet therapy in the setting of cSDH

Antiplatelet agents are another important class of medications that complicate the management of patients presenting with cSDH. Our series ( $n=88$ ) showed that cSDH patients with antiplatelet medications neither had a significantly different median hospital stay (7 vs. 9 days), nor proportion of patients experiencing a good discharge outcome (50.0% vs. 60.7%) [unpublished results]. Patients that suffer from cSDH while receiving antiplatelet agents generally have their medication discontinued immediately in an attempt to prevent hemorrhage expansion. Most commonly, patients planned for subsequent evacuation of the cSDH undergo reversal of antiplatelet agents prior to intervention. This can be accomplished through platelet transfusion [31] and/or desmopressin administration [68]; however, no studies have rigorously analyzed the efficacy of such therapy in the setting of cSDH. Similarly, little evidence exists to determine the optimal timing of post-operative resumption of antiplatelet therapy in these patients. In a recent study, Torihashi et al. [90] restarted antiplatelet medications 1 week after surgical intervention. These authors observed no significant difference in the frequency of cSDH recurrence



between those patients with a history of antiplatelet medications and those without such a history [90]. In contrast, a single-center study of 81 patients presenting with cSDH who underwent evacuation of their lesion found that a significantly higher proportion of patients with a history of aspirin (39%) underwent reoperation for recurrent cSDH than did those patients either on warfarin (21%) or without any history of anticoagulation/antiplatelet medication (28%) [75]. A third study found no statistically significant difference in recurrence of hematoma in patients who did (32%) and did not (13%) receive antiplatelet agents preoperatively [17]. Given the dearth of published literature on this topic, further studies must be performed examining the potential thrombotic and hemorrhagic consequences of management of antiplatelet medications in the setting of cSDH.

### Prophylactic anticonvulsants following cSDH

There also remains a lack of consensus regarding the prophylactic administration of antiepileptic drugs (AEDs) in patients presenting with cSDH. The reported rate of seizures in this patient population is substantial but varies widely from 2% to 19% [63, 64]. The administration of AEDs is reasonable, since patients with cSDH frequently exhibit some extent of underlying traumatic brain injury, a well-established risk factor for seizures [89]. However, anticonvulsant medications have been shown to increase the risk of falling in patients aged 65 and older, which itself represents a primary risk factor for SDH [16]. Therefore, the added risk of recurrent hemorrhage following anticonvulsant treatment must be outweighed by the benefits of AED prophylaxis. Unfortunately, the efficacy of seizure prophylaxis in the setting of both treated and untreated cSDH has been addressed only through a small number of retrospective studies [24, 64, 73, 76].

Two studies reported no significant difference in seizure rate secondary to the prophylactic administration of AEDs [64, 73]. Both groups concluded that the morbidity of AED administration outweighs the benefits except in patients at particularly high risk for seizures, such as alcoholics [64, 73]. In contrast, Sabo et al. [76] demonstrated that while 32% of the patients receiving inadequate prophylaxis developed seizures, only 2% of patients receiving appropriate prophylaxis developed new seizures. This study also demonstrated a significant correlation between seizure activity and both morbidity and mortality. Although these three studies reach seemingly contradictory conclusions, their experimental designs varied significantly, and concrete conclusions are difficult to extrapolate. A study recently published by our group, on the other hand, examined the effect of timing of prophylactic AED initiation. We found

that pre-operative initiation of AEDs was the only independent predictor of decreased post-operative seizure incidence [24]. However, this study found no effect of the timing of AED initiation on discharge outcomes [24]. These data suggest that if AEDs are to be administered, the anticonvulsant benefit may be maximized by prophylactic administration prior to surgical intervention.

Further investigation of this topic is necessary prior to establishing definitive recommendations. A prospective, randomized study with long-term follow-up must clarify the impact of the timing of seizure prophylaxis initiation, choice of surgical intervention, and co-morbid factors on patient outcomes. The duration of AED therapy must also be investigated in a standardized fashion. All possible adverse effects of the AEDs administered should be monitored, and the efficacy of different AEDs should be examined. In particular, AEDs with more favorable side effect profiles such as levetiracetam, should be compared to older agents such as phenytoin. Until more data is available, clinical judgment should be exercised, especially with patients who are at a high risk for either seizures (such as alcoholics or those with significant underlying traumatic brain injury) or for additional morbidity secondary to AED administration.

### Mobilization of patients following cSDH drainage

Mobilization of patients following drainage of cSDH is another important aspect of post-operative care. This is particularly relevant to the elderly patients who constitute the majority of patients treated for cSDH, as these patients are more susceptible to complications of immobility such as pneumonia and venous thrombosis. Despite the theoretical benefits of early mobilization, some investigators favor delayed mobilization following a period of recumbency with continuous closed system drainage, in the hopes of promoting brain expansion and thus prevent recurrence of cSDH [1, 11]. Previous literature on this topic has reached mixed conclusions about the influence of patient mobilization on cSDH recurrence.

Two prospective, single-center studies have analyzed recurrence rates of cSDH relative to post-operative positioning and have concluded that recurrence rates of cSDH are independent of post-operative posture. Nakajima et al. [62] conducted a prospective, randomized study of 46 patients with cSDH treated surgically using a single burr-hole without closed system drainage. They found no significant differences in rates of recurrence between patients who remained recumbent for 3 days post-operatively relative to those who assumed a sitting position on the day following the operation. In the second study, Kurabe et al. [45] undertook a prospective study of 182

patients aged 65 years and greater who underwent single burr-hole drainage. The incidences of complications and recurrences were compared between 91 patients who were maintained in a supine position with continuous drainage and 91 patients who were mobilized (sitting or walking) on the day of surgery, and whose drains were discontinued on the day after surgery. The authors found no difference between cohorts in the rates of recurrence, which occurred in 6 out of 91 patients in the delayed cohort (6.6%) and 8 of the 91 (8.8%) patients in the early cohort. However, the incidence of medical complications, such as pneumonia and urinary tract infection, was higher in the group undergoing delayed mobilization (26.4% vs. 12.1%,  $p=0.015$ ).

In contrast, a single study determined that post-operative posture does influence the rates of recurrence. This study examined 84 patients with cSDH who underwent single burr-hole drainage and placement of a closed system drainage for a period of 48 h [1]. Patients were randomized to two cohorts, the first remaining supine for 3 days following the operation, and the second who assumed a sitting position beginning immediately post-operatively. The authors found that recurrence rates in the supine group were 2.3% (1/42) compared to 19% (8/42) in the sitting group, and this difference reached statistical significance ( $P=0.02$ ). Only one recurrence led to repeat surgery. There were no differences between cohorts in terms of complications, including atelectasis, pneumonia, decubitus ulcers, and deep venous thrombosis.

A meaningful comparison of these studies is complicated by significant study heterogeneity, as well as the small size of the cohorts. For instance, the study by Nakajima et al. differs fundamentally from the others in the lack of closed system drainage. Although the studies by Abouzari et al. and Kurabe et al. [1, 45] employed similar surgical paradigms, they differ significantly in the age of the patients they examine. The mean age of patients examined by Kurabe et al. [45] was 77.3 years, whereas the median age in the study by Abouzari et al. [1] was 56.5 years. This difference may contribute to the observed differences in recurrence rates. Further large-scale, prospective, randomized study of mobilization techniques following cSDH drainage, with closed system drainage, should be undertaken in order to distill definitive recommendations on this important aspect of management of cSDH.

### Surgical intervention

The decision to evacuate a cSDH is influenced both by the radiographic appearance of the lesion as well as by the patient's neurological exam. An asymptomatic patient with

a small cSDH is often best observed in a carefully monitored setting. The spontaneous resolution of cSDH of significant thickness has been reported in a small number of case series [22, 65]. In general, these reports describe patients with advanced age ( $>70$  years) with significant brain atrophy and without clinical or radiographic evidence of increased intracranial pressure. Because of the possibility of spontaneous resolution, although the size of a cSDH may play a role in the decision to operate, absolute size cutoffs should be avoided. It is generally accepted that in the presence of focal symptoms and/or significant changes in neurologic status, patients should undergo immediate operative evacuation.

### Techniques for surgical evacuation of cSDH

Multiple standard surgical techniques exist for the evacuation of cSDH, including twist drill craniostomy (TDC), burr-hole craniostomy (BHC), and craniotomy. In general, TDC produce the smallest openings of the skull ( $<10$  mm), while BHC carried out using a high-speed drill enable larger openings ( $<30$  mm in diameter). Removal of a substantial piece of bone ( $>30$  mm) that is replaced and fixed to the skull defect following evacuation constitutes a craniotomy [95]. While these distinctions may be helpful to understand the existing literature, we feel it is most relevant to segregate the available surgical options into the following categories: (1) craniotomy performed in the operating room, (2) burr-hole performed in the operating room, and (3) burr-hole performed at the bedside. In general, the radiographic characteristics of the hematoma dictate the use of a specific surgical technique. For instance, acute SDH and cSDH with significant thick and/or calcified membranes are best evacuated using craniotomy [3]. However, for the majority of cSDH without extensive membranes, evacuation is possible via any of the above techniques, and the method employed is often dictated by case specifics and surgeon preference.

### Craniotomy

Prior to modern imaging techniques, evacuation of a cSDH was accomplished primarily via craniotomy. This technique exposes the largest portion of the brain and thus provides the surgeon with the most expansive operative exposure. However, craniotomy is best performed under general anesthesia and is the most invasive of the options for treatment of cSDH, encompassing the greatest operative time as well as the greatest volume of blood loss. Despite the increased risks, craniotomy remains the best option for evacuation of organized, calcified, or cSDH with numerous thick membranes [37].

### *Burr-hole craniostomy*

BHC is the surgical treatment most frequently employed for cSDH [10, 71]. According to a 2005 survey of Canadian neurosurgeons, 85% of respondents preferred BHC to either craniotomy or TDC for the initial treatment of cSDH [10]. It is most frequently performed under general anesthesia but may also be done under local anesthesia. There are a number of variations of this technique. Some surgeons prefer a single burr-hole, whereas others use two, and there has not been conclusive evidence to definitively support either approach. For instance, Taussky et al. [88] demonstrated that patients undergoing a single burr hole have a significantly higher recurrence rate, longer average hospitalization length, and higher wound infection rate. Other comparative studies [30, 39], however, do not suggest significant differences in patients treated with two burr holes compared to one with regards to recurrence, complications, mortality, or outcome.

The use of closed-system drainage after BHC has also been suggested to decrease the recurrence rate [94]. Among the few the studies addressing this issue [91, 100, 102], Santarius et al. [78] published a randomized, controlled trial which demonstrated significant benefit not only in recurrence, but also mortality and discharge outcome for patients with subdural drain placement after evacuation of the cSDH using two burr holes. Although a recent survey suggested that many neurosurgeons preferred not to place a drain following burr-hole drainage of a cSDH [79], the publication of this randomized trial will likely change the practice across many of these centers [78].

Other surgeons have suggested subperiosteal (subgaleal) drainage catheters as an alternative to subdural catheters [19, 102]. Gazzeri et al. [19] first reported this technique, in which a Jackson–Pratt drain was inserted into the subgaleal space to allow for continuous post-operative drainage. This method was utilized over 4 years to treat 224 patients. The authors only documented post-operative morbidity in three patients (1.3%), whereas 17 patients (7.6%) required a second operation for recurrence. Another recent series published by Zumofen et al. [102] supports the use of subgaleal drainage. In this study, the authors report a series of 183 symptomatic cSDH that were drained in 147 patients. Peri-operative mortality rate was 3.4%, and recurrence occurred in 13.1% of cases, requiring re-intervention in 9.3% of cases. The authors document a post-operative seizure rate of 6.6%, as well as an infection rate of 1.6%. Both of these series support the safety and efficacy of subperiosteal drainage following cSDH drainage. Although continuous drainage following drainage of cSDH is supported by the recent literature, further randomized study is necessary to compare the rates of complication and recurrence between patients undergoing subperiosteal

teal and subdural drainage, prior to formulating definitive recommendations.

### *Twist-drill craniostomy*

By comparison, TDC can be performed either at the bedside under local anesthesia or in the operating room using either local or general anesthesia. As TDC can be performed at the bedside, it is an attractive option for those elderly patients with multiple medical co-morbidities who are poor surgical candidates. However, TDC drainage is most effective in cases of cSDH in which the blood is almost completely liquefied, and there is a theoretical increased risk of contamination when performed at the bedside. A closed system Jackson–Pratt bulb drainage system is often placed at the time of surgery to allow for continuous drainage, which is thought to promote post-operative brain expansion, particularly in elderly patients with prominent brain atrophy.

### *Adjunctive techniques*

A number of additional adjunct techniques of cSDH drainage have been reported in the literature, and a review of these techniques is informative for the practicing neurosurgeon.

Takeda et al. [86] reported an interesting method for evacuation of cSDH without the use of irrigation or drainage. In this study, 77 patients with cSDH underwent local anesthesia with a needle device-bolt over the parietal eminence through a TDC. The authors then injected 10 cc of oxygen into the hematoma cavity and aspirated an equal volume with a syringe. The patient's head was subsequently rotated from lateral to supine position according to the volume of hematoma. The authors were successful in evacuating hematoma in all of their patients, with satisfactory neurological recovery in 70 patients. They observed recurrence in seven patients (10%), each of whom was successfully treated with a repeat procedure. The authors believe that their oxygen instillation technique minimizes the post-operative headache associated with low pressure, and they did not observe a higher rate of recurrent hematoma than in prior studies. The concept of filling the subdural space with air following cSDH evacuation has also been promoted by Kubo et al. [44]. In this study, the authors treated 16 patients by conventional burr-hole irrigation and closed-system drainage and compared this group to 18 subsequent patients who instead underwent CO<sub>2</sub> insufflation. They found that patients treated with gas insufflation exhibited a smaller volume of the subdural space by the first post-operative day. Although they observed recurrences in zero of the patients treated with insufflation relative to two patients treated conventionally,



this difference did not reach significance. The authors believe that CO<sub>2</sub> insufflation promotes brain expansion and may reduce recurrences following cSDH.

A variety of alternative techniques have also been promulgated in the setting of recurrent hematoma formation. For example, a number of groups have reported on the efficacy of middle meningeal artery embolization in the treatment of refractory chronic subdural hematoma [38, 53, 58, 85]. These series have all demonstrated angiography revealing abnormal vascular stains thought to represent formation of neo-capillaries in the subdural membranes. In these several reports, no further enlargements or recurrences of the cSDH were observed following embolization of these vessels.

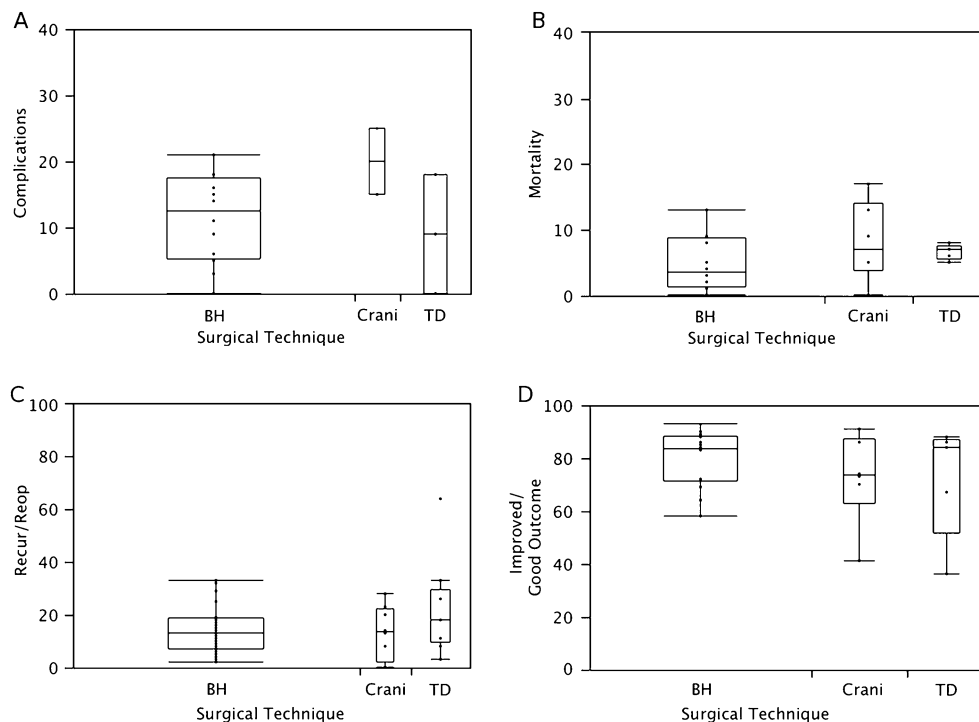
An additional adjunctive technique proposed in the treatment of recurrent cSDH is implantation of an ommaya reservoir, which permits repeated punctures and aspiration of subdural fluid. In an early prospective study by Laumer et al. [47], 144 patients with cSDH were randomly divided into three groups following burr-hole evacuation. The first group underwent burr-hole aspiration with external closed system drainage, the second underwent simple burr-hole aspiration, without any drainage, and the third underwent aspiration with placement of a subgaleal reservoir. This enabled repeated puncture and aspiration in patients who demonstrated either post-operative radiographic enlargement of the cSDH or clinical deterioration. The reoperation rate was fourfold greater in the groups treated with conventional therapy compared to the group treated with the implant system. A more recent study by Sato et al. [82]

also examined 16 patients with refractory cSDH who underwent implantation of a subgaleal ommaya CSF reservoir with the catheter placed into the subdural space. When the volume of the hematoma decreased very slowly or increased on follow-up imaging, the reservoir was punctured, and the hematoma was aspirated. All of the hematomas decreased to a thickness of <3 mm over a range of 30–143 days. A similar technique that has been employed for the treatment of recurrent cSDH is subdural-operitoneal shunting [59, 66, 80]. Although only reported in a small number of series, there have been no reported complications, and only a single recurrence reported following placement. However, placement of a subdural-operitoneal shunt requires general anesthesia, increased operative time, and carries the risk of increased infection. Therefore, further investigation of this technique for treatment of recurrent cSDH is necessary.

#### Relative safety and efficacy of the various cSDH evacuation techniques

The vast majority of studies evaluating the safety and efficacy of cSDH techniques have been single-center series (Table 1 and Fig. 2). Several of these studies support TDC as an ideal first-line option [67]. When examining a combined cohort encompassing all of the studies presented (Table 2), complications occurred in 0–18% of TDC patients, [36, 81, 83] with mortality rates ranging from 5–8% [8, 36, 67, 83]. By comparison, hematoma recurrence was detected in 3–33% of patients [21, 26, 61], with

**Fig. 2** Box-and-whisker plot of the percentages of the four different outcomes (a–d) presented in Table 1 studied as a function of surgical technique. The lines demarcate the median and inter-quartile range, with the whiskers indicating the upper and lower limits of the data. Points more than 1.5 times the inter-quartile range from either end of the box are considered outliers and plotted outside of the whisker. Boxes without whiskers have three or fewer data points (*Recur/Reop* recurrence or reoperation)



**Table 2** Outcomes for surgical treatment of cSDH, combined cohorts

|                            | TDC   | BHC   | Craniotomy         |
|----------------------------|-------|-------|--------------------|
| Complications (%)          | 0–18  | 0–25  | 15–25 <sup>a</sup> |
| Recurrence (%)             | 3–33  | 2–31  | 0–28               |
| Reoperation (%)            | 8–26  | 8–33  | 12–23              |
| Neurologic improvement (%) | 67–86 | 69–88 | 100                |
| Good outcome (%)           | 88    | 58–90 | 41–73              |
| Mortality (%)              | 5–8   | 0–13  | 0–17               |

TDC twist drill craniotomy, BHC burr hole craniotomy

<sup>a</sup> Represents a combined group of BHC and craniotomy patients

re-operations necessary in 8–26% [8, 36, 81, 83]. Neurological improvement was demonstrated in 67–86% of patients [8, 36, 69], with 88% of patients demonstrating good outcome as measured on the Glasgow Outcome Scale. While craniotomy has been deemed by some as a first-line in certain populations [6], it is more frequently recommended as a second-tier treatment for recurrent cases, when BHC or TDC prove ineffective [36, 95, 101]. Two studies report a 0% rate of recurrence with this technique (Table 2) [6, 87]; however, the rate of re-operation from other studies ranged between 12% and 23% [14, 48]. Mortality following craniotomy has been reported between 0% and 17% [6, 87, 97], while mortality of groups combining BHC and craniotomy were between 5% and 13% [36, 48]. No studies have specifically examined complication rates in a group of craniotomy-treated patients. Combined BHC and craniotomy group complication rates ranged between 15.2% and 25% [29, 36]. Neurological outcome improved for 100% of the cSDH patients treated with craniotomy in one study [87], while groups combining BHC and craniotomy showed neurological outcome improvement in 74–86% of the patients [29, 36].

Additional data beyond that provided by single-center series may be gleaned from systematic review of multiple studies. In 2003, Weigel et al. [95] published a comprehensive evidence-based review comparing the various neurosurgical techniques for treatment of cSDH. In this paper, the authors reported an average recurrence rate of 11% following craniotomy, 12% after BHC, and 18% following TDC [95]. The difference between the recurrence rates following TDC relative to the other two techniques reached statistical significance. The average morbidity rates were 12%, 3.8%, and 3.0% for craniotomy, BHC, and TDC, respectively [95]. Although the differences in morbidity from craniotomy versus BHC and TDC were statistically significant, differences in cure rates did not reach statistical significance. The authors of this study concluded that BHC provides the best balance between recurrence and morbidity for the treatment of cSDH. Nevertheless, the range in recurrence, morbidity, and mortality rates of the reviewed series overlapped for all three surgical techniques.

In order to more precisely evaluate the efficacy and complications associated with the surgical methods of cSDH evacuation, we undertook a meta-analysis of fourteen studies (listed as bold in Table 1) that directly compared two treatment groups. Unfortunately, these studies were distinctly heterogeneous, frequently flawed in study design, and reached mixed conclusions. Four of these studies were prospective, the remaining ten were retrospective. Six compared TDC to BHC [21, 26, 61, 74, 83, 98], one compared TDC to a combined group of BHC and craniotomy [36], and the other seven compared BHC to craniotomy [14, 29, 48, 52, 60, 87, 97]. The studies evaluated suggest that TDC may be superior to BHC in terms of infection rate [83], but worse in terms of the proportion of patients that developed signs of clinical deterioration [98]. Most studies also reported non-significant differences in recurrence rate [21, 26, 52, 61], and no study demonstrated a significant difference in length of hospitalization [61, 74], mortality [61, 74, 83], rate of re-operation [36], clinical course [21, 60], or neurological improvement between treatment cohorts [61].

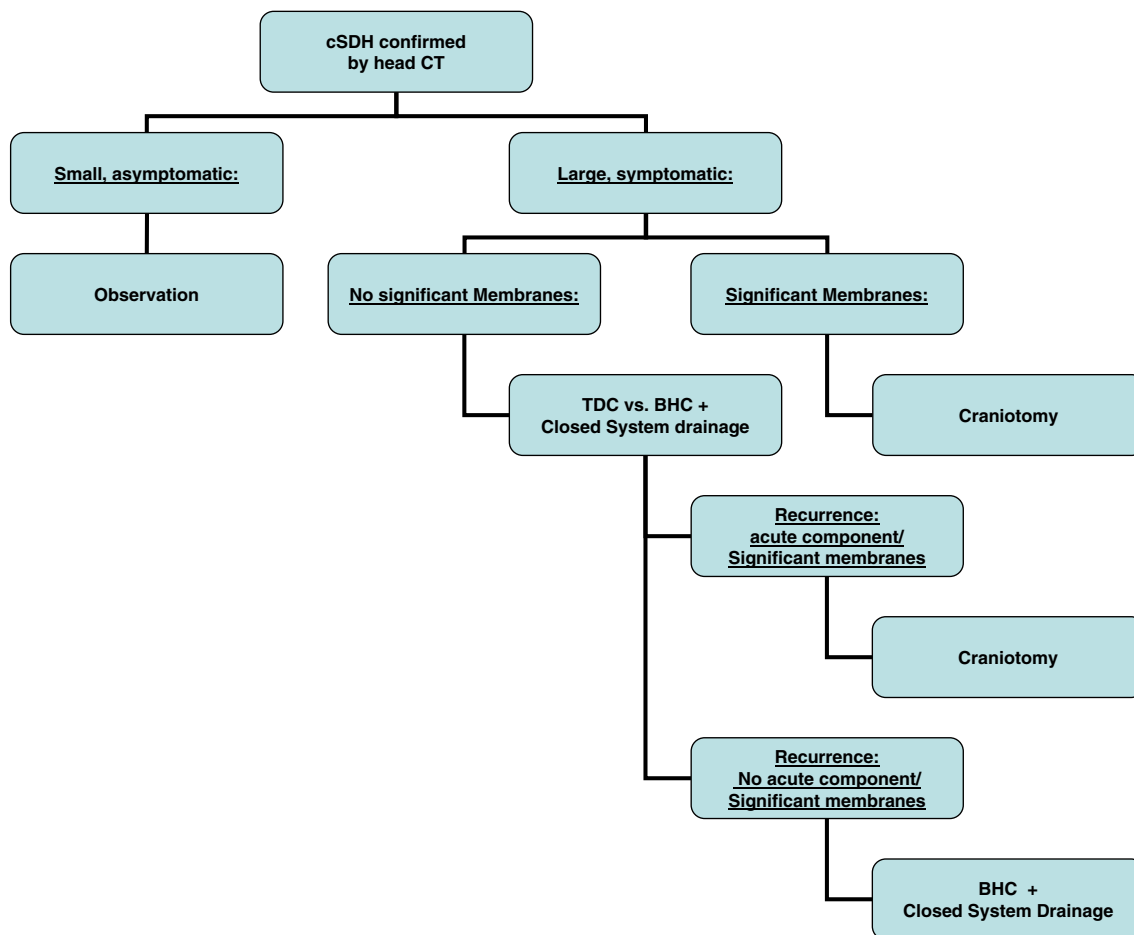
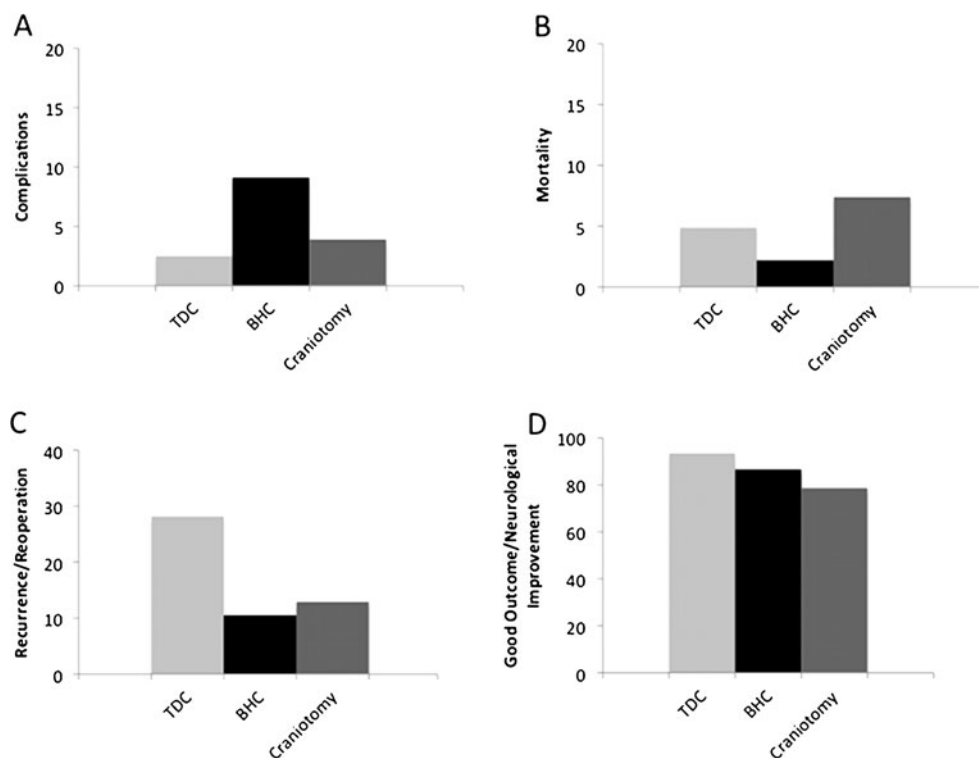
The results of our meta-analysis comparing rates of complications, mortality, recurrence/reoperation, and good outcome are presented in Table 3 and Fig. 3. This analysis revealed that rates of complications of TDC (2.5%) and craniotomy (3.9%) were lower than for BHC (9.3%,  $p<0.0001$  and  $p=0.0046$ , respectively). However, mortality was higher for craniotomy (12.2%) than either TDC (5.1%,  $p=0.0028$ ) or BHC (3.8%,  $p<0.0001$ ). The rates of recurrence or reoperation in TDC (28.1%) were significantly higher than in BHC (11.7%,  $p<0.0001$ ) and craniotomy (19.4%,  $p<0.0001$ ). The rate for BHC was also significantly lower than for craniotomy ( $p=0.0017$ ). Frequency of good outcomes, on the other hand, was the highest in the TDC patients (93.5%)—significantly better than for either BHC (86.4%,  $p<0.0001$ ) or craniotomy (74.4%,  $p<0.0001$ ). BHC

**Table 3** Meta-analysis of procedure and outcomes

|                                       | Procedure              | n (%)        | P value                |
|---------------------------------------|------------------------|--------------|------------------------|
| Complications                         | TDC ( $n=320$ )        | 8 (2.5)      | <0.0001 vs. BHC        |
|                                       | BHC ( $n=2,274$ )      | 211 (9.3)    |                        |
|                                       | Craniotomy ( $n=230$ ) | 9 (3.9)      | 0.0046 vs. BHC         |
| Mortality                             | TDC ( $n=338$ )        | 18 (5.1)     | 0.0028 vs. craniotomy  |
|                                       | BHC ( $n=2,726$ )      | 101 (3.7)    |                        |
|                                       | Craniotomy ( $n=238$ ) | 29 (12.2)    | <0.0001 vs. craniotomy |
| Recurrence/reoperation                | TDC ( $n=555$ )        | 156 (28.1)   | <0.0001 vs. TDC        |
|                                       | BHC ( $n=4,414$ )      | 514 (11.7)   |                        |
|                                       | Craniotomy ( $n=438$ ) | 85 (19.4)    | <0.0001 vs. TDC        |
| Good Outcome/neurological improvement | TDC ( $n=553$ )        | 517 (93.5)   | <0.0001 vs. TDC        |
|                                       | BHC ( $n=1,481$ )      | 1,258 (84.9) |                        |
|                                       | Craniotomy ( $n=297$ ) | 221 (74.4)   | <0.0001 vs. TDC        |

TDC twist drill craniotomy, BHC burr hole craniotomy

**Fig. 3** Bar graph of the four different outcome measures studied as a function of surgical technique (a–d) derived from the meta-analysis (Table 3)



**Fig. 4** Flowchart depicting the decision-making process in the surgical management of cSDH

patients also experienced significantly better outcomes than did patients undergoing craniotomy ( $p < 0.0001$ ).

Given the varying results arising from analysis of these heterogeneous studies, it is difficult to distill definitive management recommendations. We feel that the primary advantage of the twist drill technique is that it can be performed at the bedside, which may be desirable in specific clinical situations and patient populations, such as those whose clinical condition precludes transportation to the operating room. Additionally, bedside TDC evacuation is far less costly than a traditional operative evacuation. We therefore recommend that the evacuation technique should be determined on an individual basis, based on the imaging characteristics of the hematoma, as well as the clinical status and relevant medical co-morbidities of the patient. Generally, we favor: (1) primary TDC for patients who are high-risk surgical candidates with unseptated cSDH, (2) primary craniotomy for those cSDH patients with significant membranes observed on pre-operative imaging, and (3) a management protocol preferentially utilizing less invasive techniques (TDC or BHC), followed by craniotomy if the cSDH fails to resolve (Fig. 4). Furthermore, based primarily on the randomized, controlled trial by Santarius et al. [78], we favor post-operative closed external drainage in an effort to reduce cSDH recurrence and subsequent reoperation.

## Conclusions

We undertook a comprehensive literature review in an attempt to explore several aspects of the surgical management of cSDH. There exists a general consensus that symptomatic cSDH patients who present to a hospital on anticoagulant or antiplatelet medication must have their coagulopathy reversed, which can be successfully accomplished either rapidly or gradually depending on urgency as well as individual patient co-morbidities. Likewise, studies addressing the safety and effectiveness of AED administration for seizure prophylaxis in the setting of cSDH have been inconclusive. We generally recommend prophylaxis in patients who are likely to be at a higher risk for seizures, such as alcoholics and those with a history of significant TBI. However, caution should be exercised in the elderly population, in whom AEDs may cause significant morbidity. Regarding the surgical technique for cSDH evacuation, we recommend primary TDC drainage at the bedside for cSDH patients who are high-risk surgical candidates with unseptated hematomas and craniotomy in the setting of cSDH with significant membranes. If burr-hole evacuation of cSDH is performed, available literature supports the placement of a closed drainage system. Nevertheless, given the heterogeneity of the existing literature, future carefully

designed, prospective, randomized trials are necessary to definitively address these management issues.

**Acknowledgements** None.

## References

1. Abouzari MRA, Rezaii J, Esfandiari K, Asadollahi M, Aleali H, Abdollahzadeh M (2007) The role of postoperative patient posture in the recurrence of traumatic chronic subdural hematoma after burr-hole surgery. *Neurosurgery* 61(4):794–797
2. Ansell J, Hirsh J, Poller L, Bussey H, Jacobson A, Hylek E (2004) The pharmacology and management of the vitamin K antagonists. *Chest* 126(3):204S–233S
3. Apuzzo ML (ed) (1993) Brain surgery: complication avoidance and management. Churchill Livingstone Inc., New York
4. Asghar M, Adhiyaman V, Greenway MW, Bhowmick BK, Bates A (2002) Chronic subdural haematoma in the elderly—a North Wales experience. *J R Soc Med* 95(6):290–292. doi:10.1258/jrsm.95.6.290
5. Baechli H, Nordmann A, Bucher HC, Gratzl O (2004) Demographics and prevalent risk factors of chronic subdural haematoma: results of a large single-center cohort study. *Neurosurg Rev* 27(4):263–266. doi:10.1007/s10143-004-0337-6
6. Beatty RA (1999) Subdural haematomas in the elderly: experience with treatment by trephine craniotomy and not closing the dura or replacing the bone plate. *Br J Neurosurg* 13(1):60–64
7. Bourgeois PSM, Louis E, Haddad E, Touzet G, Fichten A, Lejeune JP (1999) Chronic subdural hematoma in patients over 80 years of age. *Neurochirurgie* 45(2):124–128
8. Camel M, Grubb RL Jr (1986) Treatment of chronic subdural hematoma by twist-drill craniotomy with continuous catheter drainage. *J Neurosurg* 65(2):183–187
9. Cartmill M, Dolan G, Byrne JL, Byrne PO (2000) Prothrombin complex concentrate for oral anticoagulant reversal in neurosurgical emergencies. *Br J Neurosurg* 14(5):458–461
10. Cenic A, Bhandari M, Reddy K (2005) Management of chronic subdural hematoma: a national survey and literature review. *Can J Neurol Sci* 32(4):501–506
11. Choudhury AR (1994) Avoidable factors that contribute to complications in the surgical treatment of chronic subdural haematoma. *Acta Neurochir (Wien)* 129(1–2):15–19
12. Cousseau DHEMG, Gaspari M, Gonorazky SE (2001) Chronic and subacute subdural haematoma. An epidemiological study in a captive population. *Rev Neurol* 32(9):821–824
13. Drapkin AJ (1991) Chronic subdural hematoma: pathophysiological basis for treatment. *Br J Neurosurg* 5(5):467–473
14. Ernestus R-I, Beldzinski P, Lanfermann H, Klug N (1997) Chronic subdural hematoma: surgical treatment and outcome in 104 patients. *Surg Neurol* 48(3):220–225
15. Estol CJKC (2003) Need for continued use of anticoagulants after intracerebral hemorrhage. *Curr Treat Options in Cardiovasc Med* 5(3):201–209
16. Ferreri S, Roth MT, Casteel C, Demby KB, Blalock SJ (2008) Methodology of an ongoing, randomized controlled trial to prevent falls through enhanced pharmaceutical care. *Am J Geriatr Pharmacother* 6(2):61–81
17. Forster M, Mathe A, Senft C, Scharrer I, Seifert V, Gerlach R (2010) The influence of preoperative anticoagulation on outcome and quality of life after surgical treatment of chronic subdural hematoma. *J Clin Neurosci* 17(8):975–979
18. Friede RL (1971) Incidence and distribution of neo-membranes of dura mater. *J Neurol Neurosurg Psychiatry* 34(4):439–446



19. Gazzeri R, Galarza M, Neroni M, Canova A, Refice GM, Esposito S (2007) Continuous subgaleal suction drainage for the treatment of chronic subdural haematoma. *Acta Neurochir (Wien)* 149(5):487–493. doi:[10.1007/s00701-007-1139-8](https://doi.org/10.1007/s00701-007-1139-8), discussion 493
20. Gennarelli TATL (1982) Biomechanics of acute subdural hematoma. *J Trauma* 22(8):680–686
21. Gökmen M, Sucu HK, Ergin A, Gökmen A, Bezircioglu H (2008) Randomized comparative study of burr-hole craniostomy versus twist drill craniostomy; surgical management of unilateral hemispheric chronic subdural hematomas. *Bohrlochtrepanation versus twist drill Kraniostomie, ein randomisierter Vergleich: Die chirurgische Therapie von unilateralen chronischen subduralen Hämatomen* (3):129–133
22. Goksu E, Akyuz M, Ucar T, Kazan S (2009) Spontaneous resolution of a large chronic subdural hematoma: a case report and review of the literature. *Ulus Travma Acil Cerrahi Derg* 15(1):95–98
23. Gonugunta V, Buxton N (2001) Warfarin and chronic subdural haematomas. *Br J Neurosurg* 15(6):514–517
24. Grobelny BT, Ducruet AF, Zacharia BE, Hickman ZL, Andersen KN, Sussman E, Carpenter A, Connolly ES (2009) Preoperative antiepileptic drug administration and the incidence of postoperative seizures following burr hole-treated chronic subdural hematoma. *J Neurosurg* 111(6):1257–1262. doi:[10.3171/2009.6.JNS0928](https://doi.org/10.3171/2009.6.JNS0928)
25. Grossman RIYD (ed) (2003) *Neuroradiology: the requisites*, vol 243, 2nd edn. Mosby, Philadelphia
26. Gurelik MAA, Gurelik B, Ozum U, Karadag O, Kars HZ (2007) A safe and effective method for treatment of chronic subdural haematoma. *Can J Neurol Sci* 34(1):84–87
27. Haines DE (1991) On the question of a subdural space. *Anat Rec* 230(1):3–21. doi:[10.1002/ar.1092300103](https://doi.org/10.1002/ar.1092300103)
28. Haines DE, Harkey HL, al-Mefty O (1993) The “subdural” space: a new look at an outdated concept. *Neurosurgery* 32(1):111–120
29. Hamilton MG, Frizzell JB, Tranmer BI (1993) Chronic subdural hematoma: the role for craniotomy reevaluated. *Neurosurgery* 33(1):67–72
30. Han H, Park C, Kim E, Yoo C, Kim Y, Kim W (2009) One vs. two burr hole craniostomy in surgical treatment of chronic subdural hematoma. *J Korean Neurosurg Soc* 46:87–92
31. Handin RI, Valeri CR (1971) Hemostatic effectiveness of platelets stored at 22 degrees C. *N Engl J Med* 285(10):538–543
32. Hanley JP (2004) Warfarin reversal. *J Clin Pathol* 57(11):1132–1139
33. Haselsberger KPR, Auer LM (1988) Prognosis after acute subdural or epidural haemorrhage. *Acta Neurochir (Wien)* 90(3–4):111–116
34. He W, Sengupta, Manisha, Velkoff, Victoria A, DeBarros, Kimberly A (2005) 65+ in the United States: 2005. Current Population Reports. U.S. Census Bureau
35. Hirsh J, Guyatt G, Albers GW, Harrington R, Schunemann HJ (2008) Executive summary: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 133(6 Suppl):71S–109S. doi:[10.1378/chest.08-0693](https://doi.org/10.1378/chest.08-0693)
36. Horn EM, Feiz-Erfan I, Bristol RE, Spetzler RF, Harrington TR (2006) Bedside twist drill craniostomy for chronic subdural hematoma: a comparative study. *Surg Neurol* 65(2):150–153. doi:[10.1016/j.surneu.2005.05.030](https://doi.org/10.1016/j.surneu.2005.05.030), discussion 153–154
37. Imaizumi S, Onuma T, Kameyama M, Naganuma H (2001) Organized chronic subdural hematoma requiring craniotomy—five case reports. *Neurol Med Chir (Tokyo)* 41(1):19–24
38. Ishihara H, Ishihara S, Kohyama S, Yamane F, Ogawa M, Sato A, Matsutani M (2007) Experience in endovascular treatment of recurrent chronic subdural hematoma. *Interv Neuroradiol* 13(Suppl 1):141–144
39. Kansal R, Nadkarni T, Goel A (2010) Single versus double burr hole drainage of chronic subdural hematomas. A study of 267 cases. *J Clin Neurosci* 17(4):428–429
40. Kaufman HH, Herschberger J, Kopitnik T, McAllister P, Hogg J, Conner T (1992) Chronic extradural haematomas: indications for surgery. *Br J Neurosurg* 6(4):359–364
41. Kaufman HH, Singer JM, Sadhu VK, Handel SF, Cohen G (1980) Isodense acute subdural hematoma. *J Comput Assist Tomogr* 4(4):557–559
42. Kawamata T, Takeshita M, Kubo O, Izawa M, Kagawa M, Takakura K (1995) Management of intracranial hemorrhage associated with anticoagulant therapy. *Surg Neurol* 44(5):438–442, discussion 443
43. Kohler M (1999) Thrombogenicity of prothrombin complex concentrates. *Thromb Res* 95(4 Suppl 1):S13–S17
44. Kubo S, Takimoto H, Nakata H, Yoshimine T (2003) Carbon dioxide insufflation for chronic subdural haematoma: a simple addition to burr-hole irrigation and closed-system drainage. *Br J Neurosurg* 17(6):547–550
45. Kurabe S, Ozawa T, Watanabe T, Aiba T (2010) Efficacy and safety of postoperative early mobilization for chronic subdural hematoma in elderly patients. *Acta Neurochir (Wien)* 152(7):1171–1174. doi:[10.1007/s00701-010-0627-4](https://doi.org/10.1007/s00701-010-0627-4)
46. Lankiewicz MW, Hays J, Friedman KD, Tinkoff G, Blatt PM (2006) Urgent reversal of warfarin with prothrombin complex concentrate. *J Thromb Haemost* 4(5):967–970. doi:[10.1111/j.1538-7836.2006.01815.x](https://doi.org/10.1111/j.1538-7836.2006.01815.x)
47. Laumer R, Schramm J, Leykauf K (1989) Implantation of a reservoir for recurrent subdural hematoma drainage. *Neurosurgery* 25(6):991–996
48. Lee J-Y, Ebel H, Ernestus R-I, Klug N (2004) Various surgical treatments of chronic subdural hematoma and outcome in 172 patients: is membranectomy necessary? *Surg Neurol* 61(6):523–527
49. Lee KS, Bae WK, Park YT, Yun IG (1994) The pathogenesis and fate of traumatic subdural hygroma. *Br J Neurosurg* 8(5):551–558
50. Lee KS, Doh JW, Bae HG, Yun IG (1996) Relations among traumatic subdural lesions. *J Korean Med Sci* 11(1):55–63
51. Lin J, Hanigan WC, Tarantino M, Wang J (2003) The use of recombinant activated factor VII to reverse warfarin-induced anticoagulation in patients with hemorrhages in the central nervous system: preliminary findings. *J Neurosurg* 98(4):737–740
52. Lindvall P, Koskinen L-OD (2009) Anticoagulants and antiplatelet agents and the risk of development and recurrence of chronic subdural haematomas. *J Clin Neurosci* 16(10):1287–1290
53. Mandai S, Sakurai M, Matsumoto Y (2000) Middle meningeal artery embolization for refractory chronic subdural hematoma. Case report. *J Neurosurg* 93(4):686–688. doi:[10.3171/jns.2000.93.4.0686](https://doi.org/10.3171/jns.2000.93.4.0686)
54. Maxeiner HWM (2002) Pure subdural hematomas: a postmortem analysis of their form and bleeding points. *Neurosurgery* 50(3):503–509
55. Mayer SRL (ed) (2000) Head injury in: *Merritt's neurology*. Williams & Wilkins, Lippincott
56. Mayer SA, Brun NC, Begtrup K, Broderick J, Davis S, Diringer MN, Skolnick BE, Steiner T (2008) Efficacy and safety of recombinant activated factor VII for acute intracerebral hemorrhage. *N Engl J Med* 352(8):777–785. doi:[10.1056/NEJMoa042991](https://doi.org/10.1056/NEJMoa042991)
57. Mayer SA, Brun NC, Begtrup K, Broderick J, Davis S, Diringer MN, Skolnick BE, Steiner T (2008) Efficacy and safety of recombinant activated factor VII for acute intracerebral hemorrhage. *N Engl J Med* 358(20):2127–2137. doi:[10.1056/NEJMoa0707534](https://doi.org/10.1056/NEJMoa0707534)
58. Mino R, Nishimura S, Hori E, Kohama M, Yonezawa S, Midorikawa H, Kaimori M, Tanaka T, Nishijima M Efficacy of



- middle meningeal artery embolization in the treatment of refractory chronic subdural hematoma. *Surg Neurol Int* 1:78. doi:10.4103/2152-7806.73801
59. Misra M, Salazar JL, Bloom DM (1996) Subdural-peritoneal shunt: treatment for bilateral chronic subdural hematoma. *Surg Neurol* 46(4):378–383
  60. Mondorf Y, Abu-Owaimer M, Gaab MR, Oertel JMK (2009) Chronic subdural hematoma—craniotomy versus burr hole trepanation. *Br J Neurosurg* 23(6):612–616. doi:10.3109/02688690903370297
  61. Muzii VFBS, Zalaffi A, Carangelo B, Mariottini A, Palma L (2005) Chronic subdural hematoma: comparison of two surgical techniques. Preliminary results of a prospective randomized study. *J Neurol Sci* 49(2):41–46
  62. Nakajima H, Yasui T, Nishikawa M, Kishi H, Kan M (2002) The role of postoperative patient posture in the recurrence of chronic subdural hematoma: a prospective randomized trial. *Surg Neurol* 58(6):385–387, discussion 387
  63. Ohaegbulam SC (1981) Surgically treated traumatic subacute and chronic subdural haematomas: a review of 132 cases. *Injury* 13(1):23–26
  64. Ohno K, Maehara T, Ichimura K, Suzuki R, Hirakawa K, Monma S (1993) Low incidence of seizures in patients with chronic subdural haematoma. *J Neurol Neurosurg Psychiatry* 56(11):1231–1233
  65. Parlato C, Guarracino A, Moraci A (2000) Spontaneous resolution of chronic subdural hematoma. *Surg Neurol* 53(4):312–315, discussion 315–317
  66. Probst C (1988) Peritoneal drainage of chronic subdural hematomas in older patients. *J Neurosurg* 68(6):908–911. doi:10.3171/jns.1988.68.6.0908
  67. Ramnarayan R, Arulmurugan B, Wilson PM, Nayar R (2008) Twist drill craniostomy with closed drainage for chronic subdural haematoma in the elderly: an effective method. *Clin Neurol Neurosurg*. doi:10.1016/j.clineuro.2008.04.013
  68. Ranucci M, Nano G, Pazzaglia A, Bianchi P, Casana R, Tealdi DG (2007) Platelet mapping and desmopressin reversal of platelet inhibition during emergency carotid endarterectomy. *J Cardiothorac Vasc Anesth* 21(6):851–854. doi:10.1053/j.jvca.2007.05.009
  69. Reinges MH, Hasselberg I, Rohde V, Kuker W, Gilsbach JM (2000) Prospective analysis of bedside percutaneous subdural tapping for the treatment of chronic subdural haematoma in adults. *J Neurol Neurosurg Psychiatry* 69(1):40–47
  70. Rocchi G, Caroli E, Salvati M, Delfini R (2007) Membranectomy in organized chronic subdural hematomas: indications and technical notes. *Surg Neurol* 67(4):374–380. doi:10.1016/j.surneu.2006.08.066, discussion 380
  71. Rohde V, Graf G, Hassler W (2002) Complications of burr-hole craniostomy and closed-system drainage for chronic subdural hematomas: a retrospective analysis of 376 patients. *Neurosurg Rev* 25(1–2):89–94
  72. Roob G, Schmidt R, Kapeller P, Lechner A, Hartung HP, Fazekas F (1999) MRI evidence of past cerebral microbleeds in a healthy elderly population. *Neurology* 52(5):991–994
  73. Rubin G, Rappaport ZH (1993) Epilepsy in chronic subdural haematoma. *Acta Neurochir (Wien)* 123(1–2):39–42
  74. Rughani AI, Lin C, Dumont TM, Penar PL, Horgan MA, Tranmer BI (2010) A case-comparison study of the subdural evacuating port system in treating chronic subdural hematomas. *J Neurosurg* 113(3):609–614. doi:10.3171/2009.11.JNS091244
  75. Rust T, Kierner N, Erasmus A (2006) Chronic subdural haematomas and anticoagulation or anti-thrombotic therapy. *J Clin Neurosci* 13(8):823–827. doi:10.1016/j.jocn.2004.12.013
  76. Sabo RA, Hanigan WC, Aldag JC (1995) Chronic subdural hematomas and seizures: the role of prophylactic anticonvulsive medication. *Surg Neurol* 43(6):579–582
  77. Sajanti J, Majamaa K (2003) High concentrations of procollagen propeptides in chronic subdural haematoma and effusion. *J Neurol Neurosurg Psychiatry* 74(4):522–524. doi:10.1136/jnnp.74.4.522
  78. Santarius T, Kirkpatrick PJ, Ganesan D, Chia HL, Jalloh I, Smielewski P, Richards HK, Marcus H, Parker RA, Price SJ, Kirollos RW, Pickard JD, Hutchinson PJ (2009) Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. *Lancet* 374(9695):1067–1073
  79. Santarius T, Lawton R, Kirkpatrick PJ, Hutchinson PJ (2008) The management of primary chronic subdural haematoma: a questionnaire survey of practice in the United Kingdom and the Republic of Ireland. *Br J Neurosurg* 22(4):529–534. doi:10.1080/02688690802195381
  80. Santarius T, Qureshi HU, Sivakumaran R, Kirkpatrick PJ, Kirollos RW, Hutchinson PJ (2010) The role of external drains and peritoneal conduits in the treatment of recurrent chronic subdural hematoma. *World Neurosurg* 73(6):747–750. doi:10.1016/j.wneu.2010.03.031
  81. Santos-Ditto RA, Santos-Franco JA, Pinos-Gavilanes MW, Mora-Benitez H, Saavedra T, Martinez-Gonzales V (2007) Management of chronic subdural hematoma with twist-drill craniostomy. Report of 213 patients. *Gac Med Mex* 143(3):203–208
  82. Sato M, Iwatsuki K, Akiyama C, Kumura E, Yoshimine T (2001) Implantation of a reservoir for refractory chronic subdural hematoma. *Neurosurgery* 48(6):1297–1301
  83. Smely CMA, Scheremet R (1997) Chronic subdural haematoma—a comparison of two different treatment modalities. *Acta Neurochir (Wien)* 139(9):818–825
  84. Stanisic M, Lund-Johansen M, Mahesparan R (2005) Treatment of chronic subdural hematoma by burr-hole craniostomy in adults: influence of some factors on postoperative recurrence. *Acta Neurochir (Wien)* 147(12):1249–1256. doi:10.1007/s00701-005-0616-1, discussion 1256–1247
  85. Takahashi K, Muraoka K, Sugiura T, Maeda Y, Mandai S, Gohda Y, Kawachi M, Matsumoto Y (2002) Middle meningeal artery embolization for refractory chronic subdural hematoma: 3 case reports. *No Shinkei Geka* 30(5):535–539
  86. Takeda N, Sasaki K, Oikawa A, Aoki N, Hori T (2006) A new simple therapeutic method for chronic subdural hematoma without irrigation and drainage. *Acta Neurochir (Wien)* 148(5):541–546. doi:10.1007/s00701-005-0689-x
  87. Tanikawa MMM, Yamada K, Yamashita N, Matsumoto T, Banno T, Miyati T (2001) Surgical treatment of chronic subdural hematoma based on intrahematoma membrane structure on MRI. *Acta Neurochir (Wien)* 143(6):613–618
  88. Taussky P, Fandino J, Landolt H (2008) Number of burr holes as independent predictor of postoperative recurrence in chronic subdural haematoma. *Br J Neurosurg* 22(2):279–282. doi:10.1080/02688690701818885
  89. Temkin NR (2003) Risk factors for posttraumatic seizures in adults. *Epilepsia* 44(Suppl 10):18–20
  90. Torihashi K, Sadamasa N, Yoshida K, Narumi O, Chin M, Yamagata S (2008) Independent predictors for recurrence of chronic subdural hematoma: a review of 343 consecutive surgical cases. *Neurosurgery* 63(6):1125–1129. doi:10.1227/01.NEU.0000335782.60059.17, discussion 1129
  91. Tsai T-H, Lieu A-S, Hwang S-L, Huang T-Y, Hwang Y-F (2010) A comparative study of the patients with bilateral or unilateral chronic subdural hematoma: precipitating factors and postoperative outcomes. *J Trauma-Inj Infect Crit Care* 68(3):571–575
  92. Victor MRA (ed) (2001) Adams and Victor's principles of neurology, 7th edn. McGraw-Hill, New York
  93. Vigue B, Ract C, Tremey B, Engrand N, Leblanc PE, Decaux A, Martin L, Benhamou D (2007) Ultra-rapid management of oral

- anticoagulant therapy-related surgical intracranial hemorrhage. *Intensive Care Med* 33(4):721–725. doi:[10.1007/s00134-007-0528-z](https://doi.org/10.1007/s00134-007-0528-z)
94. Wakai S, Hashimoto K, Watanabe N, Inoh S, Ochiai C, Nagai M (1990) Efficacy of closed-system drainage in treating chronic subdural hematoma: a prospective comparative study. *Neurosurgery* 26(5):771–773
  95. Weigel R, Schmiedek P, Krauss JK (2003) Outcome of contemporary surgery for chronic subdural haematoma: evidence based review. *J Neurol Neurosurg Psychiatry* 74(7):937–943
  96. Weir B (1971) The osmolality of subdural hematoma fluid. *J Neurosurg* 34:528–533
  97. White M, Mathieson CS, Campbell E, Lindsay KW, Murray L (2010) Treatment of chronic subdural haematomas. A retrospective comparison of minicraniectomy versus burr-hole drainage. *Br J Neurosurg* 24(3):272–275. doi:[10.3109/02688691003675218](https://doi.org/10.3109/02688691003675218)
  98. Williams RG, Baskaya MK, Menendez J, Polin R, Willis B, Nanda A (2001) Burr-hole versus twist-drill drainage for the evacuation of chronic subdural haematoma: a comparison of clinical results. *J Clin Neurosci* 8(6):551–554
  99. Wilms G, Marchal G, Geusens E, Raaijmakers C, Van Calenberghe F, Goffin J, Plets C (1992) Isodense subdural haematomas on CT: MRI findings. *Neuroradiology* 34(6):497–499
  100. Yu GJ, Han CZ, Zhang M, Zhuang HT, Jiang YG (2009) Prolonged drainage reduces the recurrence of chronic subdural hematoma. *Br J Neurosurg* 23(6):606–611. doi:[10.3109/02688690903386983](https://doi.org/10.3109/02688690903386983)
  101. Zidan AH (2007) Commentary. *Surg Neurol* 67(4):373
  102. Zumofen DMD, Regli LMD, Levivier MMD, Krayenbuhl NMD (2009) Chronic subdural hematomas treated by burr hole trepanation and a subperiosteal drainage system. *Neurosurgery* 64(6):1116–1122

## Comments

Christoph Woernle, Zurich, Switzerland

The authors reviewed and carefully analyzed the literature on treatment of chronic subdural hematoma. Looking back on history, Ambroise Pare, a French surgeon first described a subdural hemorrhage in 1559. Rudolf Virchow defined this disease in 1857 as pachymeningitis hemorrhagica interna. As the authors have nicely illustrated in this review, it is still considered one of the major issues in neurosurgery. Demographic changes will emphasize this in the future, demanding further evidence-based data on treatment modalities, but also on patients' outcome. Therefore, initiatives for multicenter, prospective, randomized trials to further evaluate the appropriate handling of patients with chronic subdural hematoma, need to be established.