Minimally Invasive Surgical Approaches for Chronic Subdural Hematomas

Ian A. Buchanan, MD, William J. Mack, MD*

KEYWORDS

• Chronic subdural hematoma • Burr hole • Twist drill • Craniostomy

KEY POINTS

- Chronic subdural hematomas have had an increasing incidence in modern neurosurgical practices because of the aging population.
- Twist drill craniostomy and burr hole craniostomy are the 2 minimally invasive approaches available for addressing chronic subdural hematomas. Of the two, burr hole craniostomy is the most widely used technique worldwide.
- Twist drill craniostomy is a relatively safe and effective first-line option for chronic subdural hematoma evacuation that obviates general anesthetic, making it an attractive option in high-risk cohorts.
- Reported estimates on cure rates, recurrence, morbidity, and mortality for the various treatment modalities are highly variable.
- Despite their prevalence, there is no consensus regarding first-line management for chronic subdural hematomas.

INTRODUCTION

Chronic subdural hematomas (cSDH) are one of the fastest growing neurologic conditions, fueled in part by an aging Western population and the bourgeoning use of anticoagulant and antiplatelet therapy.[1] In the United States alone, the total number of individuals 65 years and older is projected to double by the middle of this century, and, as a consequence, the disease burden from this clinical entity is expected to follow.[2] Current trends indicate that cSDHs are poised to become the most common intracranial diagnosis to require surgical intervention by year 2030, exceeding both primary brain tumors and metastases.[3,4] Given the substantial morbidity and mortality they carry, cSDHs therefore constitute a remarkable economic and social burden to societies worldwide.

CSDHs are the end product of liquefied blood degradation within the subdural space. Although trauma is often an inciting agent in their formation, roughly a third of patients have no such recollection, suggesting that even minor seemingly inconsequential events are precipitants of this pathologic condition.[5] The propensity for cSDH formation in the elderly can be explained by the shrinking brain volume within the confines of the cranial vault. As this occurs, tension on the bridging parasagittal veins that drain the cortical surface predisposes them to injury and hemorrhage at the dura-arachnoid interface. The theoretic subdural space, which is normally obliterated by tight adherence between the meningeal layers on account of dural border cells, is thus transformed by hematoma formation.[6]

Disclosure Statement: The authors have no commercial relationships or financial interests to disclose.
Department of Neurosurgery, Keck School of Medicine, University of Southern California, 1520 San Pablo Street, Suite 3800, Los Angeles, CA 90033, USA
* Corresponding author.
E-mail address: william.mack@med.usc.edu

Neurosurg Clin N Am 28 (2017) 219–227
http://dx.doi.org/10.1016/j.nec.2016.11.004

Discovery of cSDHs in the elderly can be viewed as a harbinger of systemic pathologic conditions in much the same way that hip fractures are seen as a proxy for mortality. Recent studies suggest that the 1-year mortality rate in patients 70 years and older with newly diagnosed cSDH approaches 32%.[7] Besides age, other factors that predispose to cSDH formation include, but are not limited to, a history of trauma, hereditary bleeding diatheses, epilepsy, ethanol abuse, and anticoagulation. Of these, age and anticoagulant use have garnered the most attention in recent years for their contributions to increasing hematoma incidence. Anticoagulation increases the risk of cSDH by more than 40-fold relative to the general population.[8] Although the mechanism in this scenario may be the result of uncontrolled bleeding from a severed vessel, more common is that there is microbleeding that progresses unchecked from an asymptomatic hemorrhage to one with neurologic sequelae.[9]

Transformation of acute bleeding to chronic bleeding follows a series of pathologic processes in which there is fibrin deposition followed by subsequent organization, enzymatic fibrinolysis, and clot liquefaction.[2] In many cases, this process is sufficient to achieve complete resorption of the hematoma. However, in other instances, a chronic inflammatory reaction is set in motion, entailing dural border cell proliferation, collagen synthesis, and neomembrane formation with neovascularization. The latter is a seminal event in the formation of cSDHs, and this ingrowth of fragile capillaries into the neomembranes underlies their propensity for microhemorrhage with cyclical recurrence. Some even propose that antithrombotic and fibrinolytic substances are secreted by the neomembrane into the hematoma cavity, enabling persistence and gradual enlargement over time.[10,11]

Three primary techniques for addressing cSDH entail sequentially larger cranial openings for hematoma drainage: twist drill craniostomy (TDC), burr hole craniostomy (BHC), and craniotomy. The process of creating windows in the skull for intracranial conditions is not new but dates as far back as the Neolithic era (8000–5000 BC).[12] Trephination, or trepanation, represents the earliest attempt by prehistoric man at neurosurgery. Although its beginnings were likely mystical in origin and based on the magico-ritual practice of freeing evil spirits from within the skull, over time it evolved into a therapeutic process for depressed skull fractures and intracranial hematomas.[12] The earliest description is documented in the Hippocratic treatise, On Injuries of the Head, in which for the first time a systematic account is provided complete with indications, timing, and technical notes. After Hippocrates' publication, trephination became widespread. It has undergone numerous iterations over the centuries, but it was not until the advent of antisepsis and improved anesthesia in the nineteenth century that the craniotomy became cemented into modernity.[13]

Despite the increasing number of cSDH cases diagnosed annually, there is no consensus for their general management. Definitive evidence regarding the superiority of one surgical modality over the other is lacking, and the decision to treat is therefore determined on an individual basis after consideration of the patient's clinical status, comorbidities, and radiologic appearance of the hematoma. Here, we discuss minimally invasive approaches for cSDH management (TDC and BHC) along with adjunctive therapies with the potential for influencing cure rates, recurrence, and other complications.

CLINICAL PRESENTATION AND INDICATIONS FOR SURGERY

A symptomatic subdural hematoma that is chronic in nature can present in a multitude of ways, earning it the nickname *great imitator*.[14] This hematoma can occur over a protracted period with isolated cognitive decline mimicking dementia or can present acutely in the context of focal neurologic deficits, as seen in stroke, or dramatically with coma and even death. Symptoms result from increased intracranial pressure or mass effect on crucial structures. However, because cSDHs tend to develop slowly and in the context of marked brain atrophy, they may not become clinically apparent until they are of a large enough size for which compensation by the cortex is no longer possible. Symptoms most commonly include headache, nausea, emesis, drowsiness, vertigo, seizures, mental deterioration, gait instability, and limb paresis. When symptoms are exceptionally vague, diagnosis can sometimes prove difficult, because there is often no history of a traumatic event to trigger routine cranial imaging. However, continued advances in radiology and the widespread availability of computed tomography scanners have contributed to an increase in cSDH diagnosis.

CSDH appears as a crescent-shaped lesion along the cerebral convexity on computed tomography (CT) imaging. It readily crosses cranial suture lines and is hypodense in appearance but can contain hyperdense regions coinciding with areas of calcification or membrane formation. Identification of dense membranes or significant loculation at the time of diagnosis is important because it may influence the pursuit of a larger craniotomy in favor of more minimally invasive approaches. The decision to evacuate a cSDH is determined by clinical presentation and the radiographic size and features of a hematoma. Surgical drainage is recommended for hematomas greater than 1 cm in diameter regardless of symptoms,[15] but this threshold is rather arbitrary and absolute volumetric size cutoffs do not exist. Asymptomatic lesions or those producing only mild symptoms can be observed in a monitored setting with medical management. The justification here is that these lesions can sometimes resolve spontaneously without further sequelae.[16] However, once focal deficits or marked changes in neurologic status become evident, the consensus is that surgical drainage should be pursued as long as there are no medical contraindications.

SURGICAL TECHNIQUE

Preoperative Considerations

After radiographic confirmation of a cSDH, patients are typically admitted for serial neurologic examinations in a carefully monitored setting. Those who may require drainage are given nothing by mouth, and a routine preoperative workup is initiated to ensure clearance for the operating room. During this period, vigilance is maintained for any signs of clinical deterioration that would warrant acceleration of a surgical timeline. As in all arenas of critical care, the "ABCs" take precedence, and any patient with a Glasgow Coma Score of 8 or lower should be intubated. Unlike in trauma, insertion of an external ventricular drain for poor GCS is not indicated and, in fact, should be avoided because drainage of cerebrospinal fluid can exacerbate the underlying cSDH by increasing retraction on draining veins, in much the same way that aggressive lowering of settings in implanted ventriculoperitoneal shunts can give rise to subdural collections. In the event of impending herniation, diuresis can be attempted as a desperate bridging therapy to surgery. However, in stable patients its use should be avoided because intravascular volume depletion severely limits brain expansion postoperatively and increases the chances of hematoma recurrence.

With the burgeoning use of anticoagulant therapy in our aging society, these medications are increasingly being implicated in cSDH hospital admissions. A thorough medical history and routine hematology and coagulation studies should therefore be performed to help identify any underlying coagulopathy as the etiology of hematoma formation. When coagulation abnormalities are discovered, it is imperative that they be preemptively addressed during the initial evaluation. If the coagulopathy is iatrogenic in origin, offending agents should be promptly discontinued to minimize the risks of enlargement from acute hemorrhage into a chronic hematoma. Additionally, the effects of antiplatelet and anticoagulant therapy should be promptly reversed. If life-threatening hematoma progression is imminent, it is universally accepted that rapid reversal be initiated regardless of the indication for anticoagulation use, as cessation of intracranial bleeding takes precedence over the preexisting medical indication that necessitated its use to begin with.

Antiepileptic pharmacotherapy is an important perioperative adjunct to consider in cSDH management. Seizure rates in patients who undergo drainage are highly variable and range from 2% to 19%.[17,18] This rate increases markedly if cortical injury is present secondary to concomitant trauma at the time of hematoma formation or if neomembrane violation occurs during cSDH evacuation.[19] Several published studies in cSDH patients posit that antiepileptic drug (AED) prophylaxis incurs unnecessary morbidity without reducing seizure frequency, except in certain high-risk cohorts.[17,20] Such high-risk patients include those with a documented history of seizures, underlying traumatic brain injury, or alcoholism. On the other hand, others strongly advocate for general AED use around the operative period, citing significant reductions in seizure rates, albeit with no impact on discharge outcomes.[21–23] These seemingly contradictory reports contribute to inconsistent AED use in cSDH treatment.

Although it is certainly reasonable to administer AEDs during the perioperative period, their use can increase the risk of falling in the elderly.[24] Special consideration should therefore be given when administering AEDs in those 65 years and older, as the increased risk of recurrent hemorrhage from falling must be weighed against the morbidity of seizures in this population.

Twist Drill Craniostomy

Surgical evacuation remains the gold standard for treating cSDHs symptomatic from mass effect. There are 3 primary surgical interventions for addressing cSDH: minimally invasive approaches, twist drill and burr hole craniostomy, and formal craniotomy. Craniotomy makes use of a bone flap (usually >30 mm), which is replaced after surgery usually with some form of plating system. It is the most invasive of the listed options and will be addressed elsewhere. TDC, as the name implies, is a percutaneous technique that entails the use of a handheld twist drill for creating a single cranial opening less than 10 mm. It is typically performed under local anesthesia at the patient's bedside and may be used in conjunction with or without subdural drain insertion or closed suction drainage attached to an implanted transosseous bolt.

TDC was first described by Tabaddor and Shulmon in 1977 when they reported on its efficacy as an alternative to larger craniotomies.[25] In the original description, a 1-cm incision is made over the hematoma. A craniostomy is then performed by placing a twist drill angled about 45° to the surface of the skull to avoid inadvertent injury to the underlying parenchyma. The dura and outer cSDH membrane can be violated with the drill but preferably with the use of a sharp needle in a more controlled fashion. A cannula is then directed into the longitudinal aspect of the hematoma and connected to a gravity drainage bag for ongoing evacuation of the subdural space. Since its development, TDC has been repeatedly validated as a first-line therapy in high-risk cohorts.[26,27] Its use is considered favorable in scenarios in which multiple comorbidities confer an unacceptably high risk for undergoing general anesthesia. One of its main disadvantages, however, is the increased risk for infection when performed bedside.

Several modifications have been made over the years to enhance the safety and sterility of TDC. In one such technique, a hollow screw or bolt is threaded down the craniostomy opening to set up a hermetically sealed drainage system, sometimes referred to as the subdural evacuating port system.[28,29] This modification not only minimizes infection, but obviates blind passage of a drain into the subdural space by relying on slight negative pressure for subdural evacuation and, hence, limits cortical injury. In another TDC variation, a special bolt is placed over the parietal eminence to facilitate instillation of oxygen or some other insufflation agent as the driving force for hematoma removal.[30] Often cited advantages of this technique include decreased likelihood for postoperative pneumocephalus and headache. Preliminary evidence suggests that these novel TDC modifications are at least equivalent, if not superior, in efficacy to their predecessor.[31]

Burr Hole Craniostomy

BHC is the most commonly used technique for evacuating cSDHs in many countries because of its low recurrence rate and low morbidity and mortality indices.[32–34] This technique was popularized by Markwalder and colleagues[35] in the 1980s after their report on its use as a viable first-line alternative to formal craniotomy. BHC can be performed under local anesthesia, but patients are typically taken to the operating room where they are placed under general or at least systemic anesthesia. BHC consists of making 2 small cranial openings 10 to 30 mm in size along the cerebral convexity. These are made with the use of a high-speed drill and spaced some distance apart to facilitate saline irrigation between them. The dura is then opened and the leaflets bipolared to the edge of the craniostomy to ensure that the subdural space remains in communication with the cranial opening. Any subdural collection is then irrigated in a reciprocating fashion until the effluent becomes clear.

Although 2 burr holes are commonly used to allow for back-and-forth irrigation, a single burr hole may be used as well. There is no conclusive evidence specifying an optimal number of cranial openings, but there are data to suggest that use of a single craniostomy results in longer hospital stays and higher rates of recurrence and wound infection.[36] Alternatively, there is also evidence in the other direction suggesting that the number of holes has no influence whatsoever on outcomes.[37–39] Although it seems obvious that visualization of the effluent during reciprocating irrigation via a 2-hole technique provides visual feedback on the adequacy of cSDH evacuation and would therefore dictate its use, the number of craniostomies made remains entirely based on surgeon preference.

Current level I evidence is in strong support of closed-system drainage of the subdural space after burr holes because of significant reductions in recurrence and length of hospitalization and concomitant improvements in mortality.[40] A temporary subdural or subgaleal drain should therefore be left in place whenever feasible to evacuate any fluid that reaccumulates in the immediate postoperative period. Because subdural drain insertion carries a risk of cortical injury, epilepsy, and infection, subgaleal or subperiosteal drainage has been proposed as a less-invasive alternative. Preliminary studies show statistically equivalent recurrence and complication rates between the 2 approaches.[41–44] Further, prospective randomized trials are needed to sufficiently determine whether subgaleal and subdural drainage are indeed comparable in efficacy and outcome.

POSTOPERATIVE CONSIDERATIONS

After treatment, strict blood pressure control within the normotensive range is paramount to minimizing acute hemorrhage in the postoperative period. An immediate postoperative CT scan should be obtained to assess the adequacy of drainage and establish a baseline for comparison going forward. If the residual clot burden is minimal and the patient found to be at their neurologic baseline, diet can be advanced by postoperative day 1. Use of perioperative antibiotics for gram-positive skin flora is generally not required beyond the first 24 hours. As the rates of recurrence are not negligible, patients should be carefully monitored for signs of reaccumulation resulting in new neurologic deficit. In otherwise asymptomatic patients, CT imaging should be repeated 1 to 2 weeks after surgery to screen for hematoma recurrence.

Early postoperative mobilization whenever feasible is one of the basic tenets of surgical management. This is especially important in the frail and elderly who are predisposed to pneumonia, deep venous thrombosis, and pulmonary emboli, well-known complications of prolonged immobilization. Notwithstanding the benefits of early mobilization, there is some evidence to suggest that bed rest promotes brain expansion and thus reduces cSDH recurrence.[45,46] This practice is however highly controversial, and only about 50% of surgeons subscribe to its use.[32,34] Randomized, controlled trials have been conducted to assess the relative contributions of postoperative position on hematoma recurrence and have reached differing conclusions. Work by Nakajima and colleagues[47] found that patient posture had no significant impact on the likelihood of cSDH recurrence. This finding has been confirmed by other studies but with an increased tendency toward medical complications—pneumonia and urinary tract infections—in the immobilized group.[48] Conversely, Abouzari and colleagues[45] discovered that patients who had the head of their bed elevated by 30° to 40° immediately after evacuation had significantly higher rates of hematoma re-formation relative to those maintained in a recumbent position. Because both studies were severely limited by the small size of their cohorts, additional work is needed before any conclusive guidelines can be established on this aspect of cSDH management.

All cSDH patients should have sequential compression devices applied at the time of admission, provided there are no contraindications. Postoperatively, anyone with impaired mobility should be started on thromboembolism prophylaxis with low-dose unfractionated heparin or its low-molecular-weight variant as soon as it is deemed safe by the operating provider. In the case of adequate intraoperative hemostasis, this could be in as little as 12 to 24 hours after surgery but preferably after removal of any drainage catheters. One obvious caveat of prophylaxis initiation is the potential for increases in hematoma recurrence. In one retrospective study, these rates were reported to be as high as 2-fold for enoxaparin.[49] Evidence-based guidelines of the optimal timing for introducing deep vein thrombosis chemoprophylaxis are lacking, and the decision is therefore at the discretion of the treating physician and may be guided by neuroimaging.

For those at high risk for thromboembolic events (eg, mechanical cardiac valves), conversion to full-dose anticoagulation is a different matter altogether and relies on a delicate balance of the likelihood of hematoma recurrence against the risk of thrombotic complications while off pharmacotherapy. To that effect, rather than a fixed interval to resumption, the neurosurgeon must assess every case individually to determine the optimal cessation period based on intraoperative findings and whether postoperative imaging demonstrates acute hemorrhage or signs of hematoma recurrence. Other important factors to consider include functional status, comorbidities, indication for anticoagulation, and likelihood for further falls or head trauma. Empirical evidence is sparse, but some data indicate that warfarin can be reintroduced safely within 72 hours of operation in the most high risk of cohorts.[50,51] There is also a dearth of information on the timing for reinitiating antiplatelet agents; however, some data suggest that this can be done safely within 7 days of any interventions.[52] Regardless of the length of time that full-dose anticoagulation or antiplatelet therapy was withheld, it would be prudent to obtain follow-up imaging after a therapeutic dose has been reinstituted and the patient proven to be within the desired therapeutic range.

SURGICAL COMPLICATIONS AND MANAGEMENT

Barring catastrophic circumstances, the most worrisome complication in the management of cSDH is a recurrence that warrants repeat surgery. Reported recurrence rates in the literature are highly variable and range anywhere from 0% to 33% for BHCs and TDCs.[2,5,53,54] However, those requiring reoperation only account for about 10% to 20%. Although the etiology for re-accumulation can be multifactorial, the most likely culprit is failure of brain expansion to eliminate intracranial dead space. This failure is more common in scenarios in which the original hematoma contained complex features not amenable to drainage by minimally invasive techniques (eg, solid clot or multiple membranes with loculation).[55] Known risk factors besides poor brain re-expansion and loculated membranes include age, large original hematoma size, bilateral lesions,[56,57] cerebral atrophy, anticoagulant or antiplatelet use, alcoholism,[58] presence of cerebrospinal fluid shunt,[59] pneumocephalus,[60] and persistent postoperative of midline shift.[61] Irrespective of the causal pathologic conditions, repeat surgical drainage via the original methodology is efficacious in more than 70% of cases, although formal craniotomy for maximal exposure would certainly be reasonable.[62]

Several strategies have been developed over the years to lessen cSDH recurrence by encouraging brain expansion and obliterating dead space. Avoidance of diuresis around the time of surgery, prompt intravenous return of perioperative fluid losses, and gentle hyperhydration are all practices devised with such reasoning in mind.[63] In a like manner, high-flow oxygen supplementation via nonrebreather mask and prolonged bed rest with recumbency were instituted to minimize pneumocephalus as a nidus for hematoma reformation. Despite these efforts, the only proven method for decreasing the possibility of relapse is intraoperative placement of a closed-system catheter for draining the subdural space. Initial trials validating the practice were all conducted with subdural drains, but early results on subgaleal catheters seem just as promising.[2,43]

The elderly are particularly susceptible to even minor complications from general anesthesia. These can blossom into adverse events that lead to prolonged ventilator dependence, untimely tracheostomy with gastrostomy, and even death. It is therefore crucial that vigilance be maintained over the course of their hospitalization so that ostensibly trivial matters can be addressed in a timely manner. These and other common complications encountered in cSDH management are summarized in **Table 1**.

SURGICAL OUTCOMES

Surgical evacuation remains the gold standard for treating patients symptomatic from a radiologically confirmed cSDH. Drainage facilitates prompt reversal of neurologic deficits and results in a favorable outcome in most patients.[2,64] With the advent of better health care, patients are living well beyond their anticipated life expectancies, and studies show surgical treatment to be safe and efficacious even in the eldest of elderly, including nonagenarians and centenarians.[64] Three principal surgical modalities exist and they use progressively larger cranial openings to address the underlying hematoma. Although the treatment algorithm is guided by the radiographic features of the cSDH, most lesions without calcification or extensive membranes can be addressed via any of the 3 modalities. There is no class I evidence available directly comparing techniques and, as a consequence, the optimal modality remains controversial with the final decision often being determined by surgeon preference.

Much of the comparison across techniques can be found in various comprehensive systematic reviews and meta-analyses.[2,62,63,65] In a 2003 review, Weigel and colleagues[62] determined that TDC, BHC, and craniotomy had similar cure and mortality rates. However, formal craniotomy was found to have a substantially higher morbidity profile (12.3%) when compared with minimally invasive approaches, such as TDC (3%; $P<.001$) and BHC (3.8%; $P<.001$). Interestingly, when recurrence rates were tallied, TDC (33%) proved far less effective at preventing relapses relative to BHC (12%; $P<.001$) and craniotomy (10.8%). This finding led to their support for BHC as the preferred first-line approach in light of its favorable cure-to-complication ratio.

A subsequent meta-analysis by Ducruet and colleagues[2] similarly found that TDC had the highest rates of recurrence (28.1%) versus BHC (11.7%, $P<.0001$) and craniotomy (19.4%, $P<.0001$). However, unlike the aforementioned study, it was BHC that carried significantly higher complication rates (9.3%) compared with TDC (2.5%; $P<.001$) and craniotomy (3.9%; $P = .0046$). Because TDC had a disproportionately larger number of patients who showed neurologic improvement, the authors advocated for its use over its counterparts, particularly when the cost implications of avoiding a trip to the operating room were considered. The challenge with distilling recommendations from these comparative data is that much of the analyses are based on studies with unclear or high risks of bias. It is not surprising then that more recent meta-analyses failed to highlight the superiority of one technique over the other, citing no significant differences regarding cure rates, recurrence, morbidity, or mortality.[63,65] Carefully designed,

Table 1
Procedure-specific and medical complications associated with chronic subdural hematoma management

Surgical Complications	Medical Complications
• Cortical injury • Intracerebral hemorrhage • Seizure • Status epilepticus • Surgical site infection • Subdural empyema • Tension pneumocephalus	• Ischemic stroke • Myocardial infarction • Hospital-acquired respiratory infection • Ventilator dependence requiring tracheostomy/gastrostomy • Venous thrombosis • Pulmonary embolism • Urinary tract infection

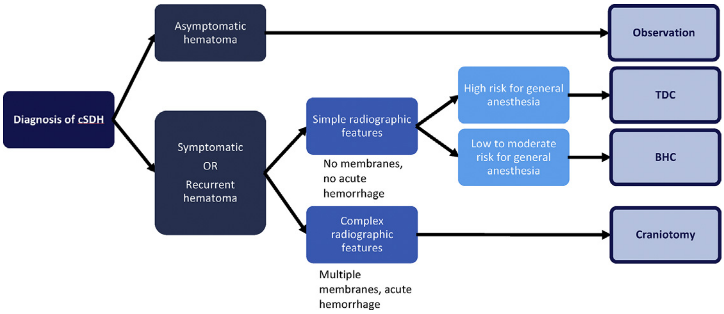

Fig. 1. Potential decision matrix for determining the appropriate surgical intervention for cSDHs.

prospective, randomized trials are needed to further elucidate the optimal method of chronic SDH evacuation.

Because evidence-based guidelines are lacking, the question of which technique to use must therefore be answered by the clinical status of the patient and the radiographic appearance of the hematoma (**Fig. 1**). Acute hemorrhage or the presence of multiloculated, thickened or calcified membranes calls for a larger craniotomy than is offered by minimally invasive techniques. In the absence of complex features, TDC and BHC are the first line of surgical therapy, with BHC being the preferred method based on its favorable cure-to-complication profile. However, in high-risk surgical cohorts TDC is favored for its bedside application and circumvention of general anesthesia. Regardless of the minimally invasive strategy used, postoperative drain placement is now the standard of care based on statistically significant reductions in recurrence and hospital length of stay.[40]

REFERENCES

1. Kolias AG, Chari A, Santarius T, et al. Chronic subdural haematoma: modern management and emerging therapies. Nat Rev Neurol 2014;10(10):570–8.
2. Ducruet AF, Grobelny BT, Zacharia BE, et al. The surgical management of chronic subdural hematoma. Neurosurg Rev 2012;35(2):155–69 [discussion: 169].
3. Filippini G. Epidemiology of primary central nervous system tumors. Handb Clin Neurol 2012;104:3–22.
4. Gavrilovic IT, Posner JB. Brain metastases: epidemiology and pathophysiology. J Neurooncol 2005;75(1):5–14.
5. Gelabert-Gonzalez M, Iglesias-Pais M, García-Allut A, et al. Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. Clin Neurol Neurosurg 2005;107(3):223–9.
6. Haines DE, Harkey HL, al-Mefty O. The "subdural" space: a new look at an outdated concept. Neurosurgery 1993;32(1):111–20.
7. Miranda LB, Braxton E, Hobbs J, et al. Chronic subdural hematoma in the elderly: not a benign disease. J Neurosurg 2011;114(1):72–6.
8. Rust T, Kiemer N, Erasmus A. Chronic subdural haematomas and anticoagulation or anti-thrombotic therapy. J Clin Neurosci 2006;13(8):823–7.
9. Roob G, Schmidt R, Kapeller P, et al. MRI evidence of past cerebral microbleeds in a healthy elderly population. Neurology 1999;52(5):991–4.
10. Ito H, Yamamoto S, Komai T, et al. Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. J Neurosurg 1976;45(1):26–31.
11. Ito H, Komai T, Yamamoto S. Fibrinolytic enzyme in the lining walls of chronic subdural hematoma. J Neurosurg 1978;48(2):197–200.
12. Newman WC, Chivukula S, Grandhi R. From mystics to modern times: a history of craniotomy & religion. World Neurosurg 2016;92:148–50.
13. Finger S, Clower WT. Victor Horsley on "trephining in pre-historic times". Neurosurgery 2001;48(4):911–7 [discussion: 917–8].
14. Potter JF, Fruin AH. Chronic subdural hematoma–the "great imitator". Geriatrics 1977;32(6):61–6.
15. Greenberg M. Chronic subdural hematoma. Handbook of Neurosurgery. 7th edition. New York: Thieme; 2010.
16. Parlato C, Guarracino A, Moraci A. Spontaneous resolution of chronic subdural hematoma. Surg Neurol 2000;53(4):312–5 [discussion: 315–7].
17. Ohno K, Maehara T, Ichimura K, et al. Low incidence of seizures in patients with chronic subdural haematoma. J Neurol Neurosurg Psychiatry 1993;56(11):1231–3.
18. Ratilal BO, Pappamikail L, Costa J, et al. Anticonvulsants for preventing seizures in patients with chronic subdural haematoma. Cochrane Database Syst Rev 2013;(6):CD004893.
19. Temkin NR. Risk factors for posttraumatic seizures in adults. Epilepsia 2003;44(Suppl 10):18–20.
20. Rubin G, Rappaport ZH. Epilepsy in chronic subdural haematoma. Acta Neurochir (Wien) 1993;123(1–2):39–42.
21. Sabo RA, Hanigan WC, Aldag JC. Chronic subdural hematomas and seizures: the role of prophylactic anticonvulsive medication. Surg Neurol 1995;43(6):579–82.

22. Grobelny BT, Ducruet AF, Zacharia BE, et al. Preoperative antiepileptic drug administration and the incidence of postoperative seizures following bur hole-treated chronic subdural hematoma. J Neurosurg 2009;111(6):1257–62.
23. Chen CW, Kuo JR, Lin HJ, et al. Early post-operative seizures after burr-hole drainage for chronic subdural hematoma: correlation with brain CT findings. J Clin Neurosci 2004;11(7):706–9.
24. Ferreri S, Roth MT, Casteel C, et al. Methodology of an ongoing, randomized controlled trial to prevent falls through enhanced pharmaceutical care. Am J Geriatr Pharmacother 2008;6(2):61–81.
25. Tabaddor K, Shulmon K. Definitive treatment of chronic subdural hematoma by twist-drill craniostomy and closed-system drainage. J Neurosurg 1977;46(2):220–6.
26. Hubschmann OR. Twist drill craniostomy in the treatment of chronic and subacute subdural hematomas in severely ill and elderly patients. Neurosurgery 1980;6(3):233–6.
27. Ramnarayan R, Arulmurugan B, Wilson PM, et al. Twist drill craniostomy with closed drainage for chronic subdural haematoma in the elderly: an effective method. Clin Neurol Neurosurg 2008;110(8):774–8.
28. Emonds N, Hassler WE. New device to treat chronic subdural hematoma–hollow screw. Neurol Res 1999;21(1):77–8.
29. Rughani AI, Lin C, Dumont TM, et al. A case-comparison study of the subdural evacuating port system in treating chronic subdural hematomas. J Neurosurg 2010;113(3):609–14.
30. Takeda N, Sasaki K, Oikawa A, et al. A new simple therapeutic method for chronic subdural hematoma without irrigation and drainage. Acta Neurochir (Wien) 2006;148(5):541–6.
31. Kubo S, Takimoto H, Nakata H, et al. Carbon dioxide insufflation for chronic subdural haematoma: a simple addition to burr-hole irrigation and closed-system drainage. Br J Neurosurg 2003;17(6):547–50.
32. Cenic A, Bhandari M, Reddy K. Management of chronic subdural hematoma: a national survey and literature review. Can J Neurol Sci 2005;32(4):501–6.
33. Nayil K, Ramzan A, Sajad A, et al. Subdural hematomas: an analysis of 1181 Kashmiri patients. World Neurosurg 2012;77(1):103–10.
34. Santarius T, Lawton R, Kirkpatrick PJ, et al. The management of primary chronic subdural haematoma: a questionnaire survey of practice in the United Kingdom and the Republic of Ireland. Br J Neurosurg 2008;22(4):529–34.
35. Markwalder TM, Steinsiepe KF, Rohner M, et al. The course of chronic subdural hematomas after burr-hole craniostomy and closed-system drainage. J Neurosurg 1981;55(3):390–6.
36. Taussky P, Fandino J, Landolt H. Number of burr holes as independent predictor of postoperative recurrence in chronic subdural haematoma. Br J Neurosurg 2008;22(2):279–82.
37. Kansal R, Nadkarni T, Goel A. Single versus double burr hole drainage of chronic subdural hematomas. A study of 267 cases. J Clin Neurosci 2010;17(4):428–9.
38. Han HJ, Park CW, Kim EY, et al. One vs. Two Burr Hole Craniostomy in Surgical Treatment of Chronic Subdural Hematoma. J Korean Neurosurg Soc 2009;46(2):87–92.
39. Belkhair S, Pickett G. One versus double burr holes for treating chronic subdural hematoma meta-analysis. Can J Neurol Sci 2013;40(1):56–60.
40. Santarius T, Kirkpatrick PJ, Ganesan D, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet 2009;374(9695):1067–73.
41. Gazzeri R, Galarza M, Neroni M, et al. Continuous subgaleal suction drainage for the treatment of chronic subdural haematoma. Acta Neurochir (Wien) 2007;149(5):487–93 [discussion: 493].
42. Zumofen D, Regli L, Levivier M, et al. Chronic subdural hematomas treated by burr hole trepanation and a subperiostal drainage system. Neurosurgery 2009;64(6):1116–21 [discussion: 1121–2].
43. Bellut D, Woernle CM, Burkhardt JK, et al. Subdural drainage versus subperiosteal drainage in burr-hole trepanation for symptomatic chronic subdural hematomas. World Neurosurg 2012;77(1):111–8.
44. Kaliaperumal C, Khalil A, Fenton E, et al. A prospective randomised study to compare the utility and outcomes of subdural and subperiosteal drains for the treatment of chronic subdural haematoma. Acta Neurochir (Wien) 2012;154(11):2083–8 [discussion : 2088–9].
45. Abouzari M, Rashidi A, Rezaii J, et al. The role of postoperative patient posture in the recurrence of traumatic chronic subdural hematoma after burr-hole surgery. Neurosurgery 2007;61(4):794–7 [discussion: 797].
46. Choudhury AR. Avoidable factors that contribute to complications in the surgical treatment of chronic subdural haematoma. Acta Neurochir (Wien) 1994;129(1–2):15–9.
47. Nakajima H, Yasui T, Nishikawa M, et al. The role of postoperative patient posture in the recurrence of chronic subdural hematoma: a prospective randomized trial. Surg Neurol 2002;58(6):385–7 [discussion: 387].
48. Kurabe S, Ozawa T, Watanabe T, et al. Efficacy and safety of postoperative early mobilization for chronic subdural hematoma in elderly patients. Acta Neurochir (Wien) 2010;152(7):1171–4.
49. Tahsim-Oglou Y, Beseoglu K, Hänggi D, et al. Factors predicting recurrence of chronic subdural

haematoma: the influence of intraoperative irrigation and low-molecular-weight heparin thromboprophylaxis. Acta Neurochir (Wien) 2012;154(6):1063–7 [discussion: 1068].
50. Yeon JY, Kong DS, Hong SC. Safety of early warfarin resumption following burr hole drainage for warfarin-associated subacute or chronic subdural hemorrhage. J Neurotrauma 2012;29(7):1334–41.
51. Kawamata T, Takeshita M, Kubo O, et al. Management of intracranial hemorrhage associated with anticoagulant therapy. Surg Neurol 1995;44(5):438–42 [discussion: 443].
52. Torihashi K, Sadamasa N, Yoshida K, et al. Independent predictors for recurrence of chronic subdural hematoma: a review of 343 consecutive surgical cases. Neurosurgery 2008;63(6):1125–9 [discussion: 1129].
53. Ohba S, Kinoshita Y, Nakagawa T, et al. The risk factors for recurrence of chronic subdural hematoma. Neurosurg Rev 2013;36(1):145–9 [discussion: 149–50].
54. Baechli H, Nordmann A, Bucher HC, et al. Demographics and prevalent risk factors of chronic subdural haematoma: results of a large single-center cohort study. Neurosurg Rev 2004;27(4):263–6.
55. Ramachandran R, Hegde T. Chronic subdural hematomas–causes of morbidity and mortality. Surg Neurol 2007;67(4):367–72 [discussion: 372–3].
56. Kung WM, Hung KS, Chiu WT, et al. Quantitative assessment of impaired postevacuation brain re-expansion in bilateral chronic subdural haematoma: possible mechanism of the higher recurrence rate. Injury 2012;43(5):598–602.
57. Tugcu B, Tanriverdi O, Baydin S, et al. Can recurrence of chronic subdural hematoma be predicted? A retrospective analysis of 292 cases. J Neurol Surg A Cent Eur Neurosurg 2014;75(1):37–41.
58. Sonne NM, Tonnesen H. The influence of alcoholism on outcome after evacuation of subdural haematoma. Br J Neurosurg 1992;6(2):125–30.
59. Delgado PD, Cogolludo FJ, Mateo O, et al. Early prognosis in chronic subdural hematomas. Multivariate analysis of 137 cases. Rev Neurol 2000;30(9):811–7 [in Spanish].
60. Mori K, Maeda M. Surgical treatment of chronic subdural hematoma in 500 consecutive cases: clinical characteristics, surgical outcome, complications, and recurrence rate. Neurol Med Chir (Tokyo) 2001;41(8):371–81.
61. Chon KH, Lee JM, Koh EJ, et al. Independent predictors for recurrence of chronic subdural hematoma. Acta Neurochir (Wien) 2012;154(9):1541–8.
62. Weigel R, Schmiedek P, Krauss JK. Outcome of contemporary surgery for chronic subdural haematoma: evidence based review. J Neurol Neurosurg Psychiatry 2003;74(7):937–43.
63. Ivamoto HS, Lemos HP Jr, Atallah AN. Surgical treatments for chronic subdural hematomas: a comprehensive systematic review. World Neurosurg 2016;86:399–418.
64. Lee L, Ker J, Ng HY, et al. Outcomes of chronic subdural hematoma drainage in nonagenarians and centenarians: a multicenter study. J Neurosurg 2016;124(2):546–51.
65. Almenawer SA, Farrokhyar F, Hong C, et al. Chronic subdural hematoma management: a systematic review and meta-analysis of 34,829 patients. Ann Surg 2014;259(3):449–57.