Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations

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OBJECT Chronic subdural hematomas (SDHs) are more common among veterans and elderly persons than among members of the general population; however, precise incidence rates are unknown. The purposes of this study were 1) to determine the current incidence of chronic SDH in a US Veterans Administration (VA) population and 2) to create a mathematical model for determining the current and future incidence of chronic SDH as a function of population age, sex, and comorbidity in the United States VA and civilian populations.

METHODS To determine the actual number of veterans who received a radiographic diagnosis and surgical treatment for SDH during 2000–2012, the authors used the VISN03 VA database. On the basis of this result and data from outside the United States, they then created a mathematical model accounting for age, sex, and alcohol consumption to predict the incidence of SDH in the VA and civilian populations during 2012–2040.

RESULTS Of 875,842 unique (different patient) visits to a VA hospital during the study period, 695 new SDHs were identified on CT images. Of these 695 SDHs, 203 (29%) required surgical drainage. The incidence rate was 79.4 SDHs per 100,000 persons, and the age-standardized rate was 39.1 ± 4.74 SDHs per 100,000 persons. The authors' model predicts that incidence rates of chronic SDH in aging United States VA and civilian populations will reach 121.4 and 17.4 cases per 100,000 persons, respectively, by 2030, at which time, approximately 60,000 cases of chronic SDH will occur each year in the United States.

CONCLUSIONS The incidence of chronic SDH is rising; SDH is projected to become the most common cranial neurosurgical condition among adults by the year 2030.

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KEY WORDS atrophy; incidence; mathematical model; subdural hemorrhage; subdural hematoma; trauma; vascular disorders

INCIDENCE of chronic subdural hematoma (SDH) is increasing, and the prognosis for patients with chronic SDH is deceptively poor. For up to 20% of patients, neurological outcomes are poor, resulting in significant disability.^{8,13,19,22} Perioperative mortality rates for patients with chronic SDH range from 1.2% to 11%. Among elderly patients who undergo treatment with a drainage intervention, the 1-year mortality rate is 32%.³¹ The outcome is even worse if chronic SDH converts to acute SDH.^{13,22,30,34} According to a recent study, the mean survival time for 209 patients after treatment for chronic SDH is 4.4 years, which is significantly shorter (hazard ratio 1.94, $p <$

0.0002) than the mean survival time of 6.0 years computed from actuarial life tables.³¹

The incidence rate for chronic SDH is progressively increasing as the population ages. Current trends in an aging population predict that after approximately 20%–25% of the population is older than 65 years, chronic SDH will surpass primary brain tumors (up to 14 cases/100,000 persons/year)¹⁰ and metastases (approximately 28 cases/100,000 persons/year)¹⁶ as the most common cranial surgical condition.²⁶ In the United States, this prediction is projected to occur by the year 2030.⁴¹

Among global populations, the incidence of chronic

ABBREVIATIONS IEN = internal entry number; LPC = liters per capita; SDH = subdural hematoma; VA = Veterans Administration.

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SDH has steadily risen since 1967. It was 1.7 cases per 100,000 persons in Helsinki, Finland, during 1967–1973,¹¹ and 2.0 cases per 100,000 persons in Sweden in 1969.³⁰ The incidence was 13.1 cases per 100,000 persons in Japan during 1986–1988²⁶ and increased to 20.6 cases per 100,000 persons by 2005.²⁵ Among persons older than 80 years, who comprise one-third of the total afflicted population,^{11,13,26,30} the incidence is 127.1 cases per 100,000 persons.²⁵ An annual incidence rate of 20 cases per 100,000 persons suggests that starting in 2030, approximately 60,000 Americans will become afflicted with chronic SDH each year.

Although chronic SDH can resolve spontaneously,^{18,24} an untreated hematoma can be fatal, either as a result of cerebral decompensation or a deteriorating neurological condition.²⁸ For patients with large chronic SDHs, recurrence is less (15%) and neurological recovery is more complete for those who undergo drainage than for those who do not (26%).³⁵ Thus, surgery may either be necessary or have utility for chronic SDH management.

The epidemiology of chronic SDH, despite its growing incidence, has not been rigorously studied in the United States population. Neurosurgeons must be prepared to face the inevitable increased workload that will accompany the increased incidence of chronic SDH. The objective of this study was to determine the incidence of chronic SDH in a portion of the Veterans Administration (VA) population. The VA provides health care to approximately 40% of veterans. Veterans who use the VA system are significantly older (median age 64 years) and have lower household income than those who do not (median age 53 years).³³

Using information obtained from the VA database, as well as data from outside the United States, we created a mathematical model to predict the future incidence of chronic SDH in both VA and civilian populations.

Methods

Institutional Review Board approval was obtained before study initiation.

The VA Data Search Algorithm

Patient data were extracted from the New York Harbor Healthcare System VISTA (Veterans Health Information Systems and Technology Architecture) network, which was accessed by using FileMan software through the Attachmate Reflection graphic user interface. Information was retrieved from the PATIENT (File #2), RAD/NUC MED REPORTS (File #73), VISIT (File #9000010), and SURGERY (File #130) files by using FileMan filtering and parsing tools.

Each patient in the database was referenced by the internal entry number (IEN) of their unique PATIENT record. Patients with duplicate names and incorrect or altered Social Security numbers retain a unique IEN, negating the danger of losing or misattributing relationships between information stored in the different files.

The RAD/NUC MED REPORTS file IMPRESSION TEXT (#300) was searched for the case-insensitive terms “subdural,” “sub dural,” and/or “sub-dural.” The IEN of the PATIENT NAME (#2) was retrieved by using the IN-

TERNAL (PATIENT NAME) method. For each record, the EXAM DATE/TIME (#3) and IMPRESSION TEXT (#300) was printed.

The results of the file were visually scanned, and those with a radiologist’s impression (impression text) interpreted as not diagnostic of an SDH were excluded. These excluded impression texts were those specifically denying the appearance of subdural hematomas or hemorrhages as well as those diagnosing subdural hygromas. The resulting record set was assumed to be a continuous list of all radiographic diagnoses of SDHs accompanied by the date the scan was performed. Patients with radiographic diagnoses of SDH before 2000 were excluded from the study. Because many chronic SDHs had acute components, these were not excluded and acute SDHs were not differentiated in other ways.

The SURGERY file entitled PRINCIPAL PROCEDURE (#26) was searched for terms including “subdural,” “seps,” “bur,” “sdh,” “craniotomy,” “hematoma,” and/or “hemorrhage.” The IEN of PATIENT (#0.01), the PRINCIPLE PROCEDURE (#26), and the DATE OF OPERATION (#0.09) were retrieved. These results were manually scanned for procedures that did not involve SDH evacuation. Patients who had undergone SDH evacuation before the year 2000 were excluded from the study.

Database software was used to find the first of either the date of SDH evacuation or the first of the date of radiographic diagnosis of an SDH for the remaining patients who did not receive an SDH diagnosis until after the beginning of 2000. The results were stratified by year, and the number of patients with a new SDH diagnosis each year during 2000–2012 were counted. By using each patient’s birth date linked to the previous results via patient IEN, the age and sex of each of the counted patients at the year of diagnoses were assigned, and every patient encounter was searched via the VISIT file. Each search was restricted to 1 year of the VISIT/ADMIT DATE&TIME (#0.01) and retrieved only the IEN of PATIENT NAME (0.05). The number of unique IEN values per year was then calculated by using database software. To obtain a year-stratified record set of the number of unique patients with at least 1 encounter per year, accompanied by the age and sex of each patient at the time, the database was cross-referenced with the patient demographic information.

The results of this search were then stratified by age and sex over the 13 years of the study.

Mathematical Model for Projecting Future SDH Incidence Rates

To make a mathematical model of the incidence of chronic SDH in the general US population, we made the following calculations:

When formulas are given, “% of a population” means the fraction (i.e., a number between 0 and 1).

We obtained US population data for 1980–2050, classified by sex and broad age groups (0–14, 15–64, ≥ 65 years) and by sex and 5-year age groups (0–4, 5–9, 10–14 years, etc.) (<http://www.census.gov/population/international/data/idb/informationGateway.php>). The Japan-based formula was derived from an incidence study²⁶ enabling calculation of cases of chronic SDH per 100,000 persons, classified by age. For determination of an overall rate (per

100,000 persons) for a particular year, the following calculation was performed:

Overall rate = $3.4 \times (\% \text{ of population } < 65 \text{ years}) + 58.1 \times (\% \text{ of population } \geq 65 \text{ years})$

The same principle applies to finding the total number of chronic SDH cases for a particular year:

Total no. = $[3.4 \times (\text{no. of persons } < 65 \text{ years}) + 58.1 \times (\text{no. of persons } \geq 65 \text{ years})] / 100,000 \text{ persons}$

The Finland-based formula was similarly derived from another incidence study on chronic SDH that detailed cases per 100,000 persons.¹¹ To determine an overall rate (per 100,000 persons) for a particular year, the following calculations were performed:

Overall rate = $0.25 \times (\% \text{ of male population } 20\text{--}29 \text{ years}) + 0 \times (\% \text{ of female population } 20\text{--}29 \text{ years}) + 1.56 \times (\% \text{ of male population } 30\text{--}39 \text{ years}) + 0.76 \times (\% \text{ of female population } 30\text{--}39 \text{ years}) + 4 \times (\% \text{ of male population } 40\text{--}49 \text{ years}), \text{ etc.}$

The same principle applies to finding the total number of chronic SDH cases for a particular year:

Total no. = $[0.25 \times (\text{no. of males } 20\text{--}29 \text{ years}) + 0 \times (\text{no. of females } 20\text{--}29 \text{ years}) + 1.56 \times (\text{no. of males } 30\text{--}39 \text{ years}) + 0.76 \times (\text{no. of females } 30\text{--}39 \text{ years}) + 4 \times (\text{no. of males } 40\text{--}49 \text{ years}), \text{ etc.}] / 100,000$

Alcoholism/alcohol dependence is a major risk factor for chronic SDH. However historical data on these parameters are not readily available. As a substitute, we used average alcohol consumption measured in liters per capita (LPC).

At the approximate time of the Kudo et al. study in Japan (1990),²⁶ alcohol consumption by persons 15 years of age or older in Japan was 8 LPC; at the approximate time of the Foelholm and Waltimo study in Finland (1970),¹¹ alcohol consumption by persons 15 years of age or older in Finland was 5.8 LPC (<http://www.nationmaster.com/country-info/stats/Lifestyle/Food-and-drink/Alcohol/Consumption/1970>). Because the formulas and data from these studies were based on these levels of alcohol consumption, they were kept constant over the years being analyzed. In the United States, alcohol consumption by persons 15 years of age or older was 9.5 LPC in 1970, 10.5 LPC in 1980, 9.3 LPC in 1990, 8.3 LPC in 2000, and 9.4 LPC in 2005 (<http://www.nationmaster.com/country-info/stats/Lifestyle/Food-and-drink/Alcohol/Consumption/1970>). For the years between those for which values are given, alcohol consumption was linearly interpolated on the basis of the end points. The alcohol consumption for every year after 2005 was assumed to be 9.4 LPC. For Japan-based numbers, for each year, the nonadjusted value for number of chronic SDH cases each year was multiplied by the ratio between alcohol consumption in the United States and that in Japan in 1990 (8 LPC). Last, the age-stratified incidence rates were again age-standardized to the VA population from 2010 through 2040 (estimated), and the results were plotted as the predicted age-standardized rate of SDHs in the future over time until 2040.

Results

Over the course of the study period, 2000–2012, a total of 875,842 unique patient visits to a VA facility were re-

ported, counting each patient once per year but including patients counted more than once if the visits occurred in different years. The average number of patient visits per year was $67,372 \pm 15,773$. During that period, 695 new chronic SDHs were diagnosed, accounting for an average of 53 ± 11 visits per year; of those, 203 SDHs were surgically treated. The incidence rate across all years of the study was 79.4 chronic SDHs per 100,000 persons (Fig. 1, Table 1). This incidence was stratified by age and sex, demonstrating that the incidence of chronic SDH increases with age (Table 1). Chronic SDH recurred for 11% of patients, including recurrence as long as 3 years after initial diagnosis.

The age standardized rate of SDH occurrence among VA patients (Table 1) weighted against the rate of chronic SDH in the world standard population according to the 2000–2025 SEER (Surveillance, Epidemiology, and End Results) database was 39.1 ± 4.74 . To gauge the standard error of the mean, we used a Poisson comparison of the age distribution of the person-years in the study population to the standard weighted age distribution of the person-years in the world population. For comparison, the age-standardized rate relative to the world standard, from a previous study of the incidence of subdural hematomas in Finland,¹¹ was found to be 2.4 per 100,000 persons. The incidence of chronic SDH among the general US population was then projected to estimate the effect of an aging population on future incidence of chronic SDH. In Fig. 1, the upward trend of the blue area depicts the aging American population. The graph illustrates the present and projected increase in the incidence rate of SDH until 2040 as the population continues to age (Fig. 1). In the same way, incidence rates for chronic SDH among the VA population were projected and the results were depicted in a histogram (Fig. 2). The histogram shows incidence rate of chronic SDH in the VA population as a function of time in years during 2000–2012. The histogram illustrates an increasing trend in both operative and nonoperative cases of chronic SDH during the study period.

Discussion

Despite the fact that chronic SDH is a common surgical condition associated with a high rate of complications, few studies have assessed its incidence in the United States. Our results show that during the study period (2000–2012), incidence among the VA population was much higher than the mathematically calculated incidence for the general US population. The incidence rate for chronic SDH across the 13 years of study in the VA population was 79.6 cases per 100,000 persons, and the average incidence rate for the general US population, according to the Japan-based formula in the same years,²⁶ was 10.35 cases per 100,000 persons. When the Finland-based formula was used,¹¹ the average incidence rate for the general US population was 7.24 cases per 100,000 persons. The nearly 10-fold difference in incidence rate between VA and nonveteran populations within the same geographic area probably reflects differences in age, sex, and other risk factors between these widely disparate populations.

Given current aging trends, the incidence of chronic

FIG. 1. Graph showing the incidence rate for chronic SDH (cSDH) in the general US population as a function of time. The y-axis on the left shows the scale for the percentage of the US population older than 65 years. The y-axis on the right shows the scale for incidence rate, shown as number of cases of chronic SDH per 100,000 persons. The upward trend of the blue area in the graph depicts the aging American population. The continuous maroon line depicts the incidence rate for chronic SDH, according to the Japan-based formula, and the interrupted maroon line shows the alcohol-adjusted incidence rate. The continuous orange line depicts the incidence rate according to the Finland-based formula, and the interrupted orange line shows the alcohol-adjusted incidence rate. The graph illustrates the present and projected increase in incidence rates for chronic SDH until 2040 as the population continues to age. Figure is available in color online only.

SDH for the VA population is projected to reach up to 121.4 cases per 100,000 persons by 2030, and the incidence for the general US population at the same time, according to the Japan-based formula, will be 17.1 cases per 100,000 persons after adjustment for alcohol consumption. The Finland-based formula shows that by 2030, the incidence rate will be 8.75 cases per 100,000 persons.

For patients who received treatment at the New York Harbor VA facility during 2008–2010, the length of hospital stay for those with chronic SDH requiring evacuation of the hemorrhage (9.3 ± 6.8 days, $n = 74$) was significantly greater than that for a brain tumor patients undergoing craniotomy (7.0 ± 0.5 days, $n = 94$).⁵ The noted SDH recurrence rate of 11% was consistent with recurrence rates ranging from 5% to 14%.^{11,13,26,30}

The increasing incidence of chronic SDH in an aging population underscores the fact that chronic SDH is as much, if not more, a sequela of degenerative disease than of trauma. Historically, the pathogenesis of chronic SDH

has never been solely attributed to trauma.^{14,39} Virchow first hypothesized in 1857, after postmortem studies, that organized exudates accumulate in the subdural space as a result of a generalized inflammatory process, and he coined the term “pachymeningitis haemorrhagica interna.”¹⁴ In 1914, Trotter reported 4 cases of surgically treated chronic SDH and proposed trauma, as opposed to an inflammatory process, as their cause.⁴⁰ Subsequent studies show that blood in the subdural space, secondary to meningeal trauma, provokes a nonspecific inflammatory reaction by dural cells, eventually leading to formation of a vascular neomembrane,^{23,32} which is responsible for repeat microhemorrhages and resulting growth of the hematoma.^{14,23,32}

In a recent study of 778 patients with chronic SDH in Brazil, as many as 40% of patients denied a history of trauma.³⁷ Furthermore, chronic SDH has been reported as late as 9 months after an initiating trauma.³⁶ Trauma results in extravasation of blood into the subdural space;^{7,28,42} thus, a larger subdural space predisposes to blood collec-

TABLE 1. Incidence rates for chronic SDH among persons in the VA, 2000–2012, stratified by age and sex*

Patient Age Range (yrs)	No. of SDHs	Incidence Rates		
		Female Patients	Male Patients	Total
0–4	0	0.0	0.0	0.0
5–9	0	0.0	0.0	0.0
10–14	0	0.0	0.0	0.0
15–19	0	0.0	0.0	0.0
20–24	2	0.0	13.9	9.8
25–29	4	0.0	17.4	12.3
30–34	8	22.3	28.5	27.0
35–39	1	13.1	0.0	3.4
40–44	14	53.4	36.2	40.6
45–49	19	0.0	48.3	39.0
50–54	30	10.7	43.0	39.0
55–59	55	0.0	65.3	59.3
60–64	55	33.8	56.7	55.2
65–69	60	0.0	83.6	80.8
70–74	75	91.6	114.0	112.5
75–79	105	54.6	115.0	114.0
80–84	127	204.8	161.5	161.1
85–89	93	50.6	213.2	209.2
90–94	34	0.0	244.9	237.2
95–99	12	0.0	756.2	699.7
≥100	1	0.0	178.9	150.8

* Incidence rate = no. of cases/100,000 persons. The average incidence rate for this period was 79.4.

tion. Microscopy of postmortem material demonstrates that the subdural portion of the bridging veins has thinner vessel walls with less collagen, resulting in greater fragility than the subarachnoid portion.⁴³

Risk Factors

In our study, 72.9% of chronic SDHs were reported for persons older than 65 years. This finding is consistent with results from previous studies in other geographic regions, where about two-thirds (57%–78.7%) of chronic SDHs were in persons older than 65 years.^{26,29,37} This susceptibility of the elderly has been postulated to result from increased cerebral atrophy, which is exemplified by a positive correlation between the incidence of chronic SDH and the amount of atrophy of the brain, as demonstrated by volumetric analysis of CSF.⁴⁴ This increased atrophy in turn enables minor stress or trauma to provoke separation of the dura-arachnoid interface, as also occurs with subdural hygroma.²⁷ Cat and dog models suggest that after the dura and arachnoid separate, fibrin (from serum or exudates) can induce proliferation of granulation tissue on the inner dural surface.¹ Also, in an atrophied brain, the lack of tamponade effect from the brain surface on the dura

promotes the development and expansion of a hematoma beneath the dura.^{2,28} Thus, brain atrophy, along with the increased use of anticoagulant drugs in the elderly, is postulated to account for the increased incidence of chronic SDH in this subpopulation.

In our study, 680 (97.85%) chronic SDHs were in men and 15 (2.15%) in women. The incidence rate among women was 18.6 cases per 100,000 persons, and the incidence rate among men was 85.5 cases per 100,000 persons. These rates give a male/female ratio of 4.6:1. This finding is consistent with results from other studies that show a predominance among men.^{3,4,6,23,32,37,38}

A study conducted in Sweden during 1969–1993 showed that alcoholics comprised 14.7% of all patients with chronic SDH.³⁰ Alcohol causes brain atrophy, which in turn is an independent risk factor for chronic SDH.^{2,28,44} Moreover, it is hypothesized that the increased estrogen that accompanies alcoholic hepatopathy³⁸ may cause the blood vessels in the inner dural layer to become more ectatic because of estrogen priming of these vessels and thus more likely to bleed.²⁸ Another effect of alcoholic hepatopathy, and one possibly more contributory to chronic SDH, is the coagulopathy that accompanies liver damage.^{9,21,38} The deficiency of platelets and coagulation factors is believed to induce a state of hypercoagulability in persons with chronic alcoholism and alcoholic hepatopathy and thus predispose them to chronic SDH development after minor trauma.^{6,12,38} Among chronic SDH patients in the same study, 18% were taking anticoagulation medication and an additional 17% consumed aspirin. Among 1000 surgically treated chronic SDH patients in Spain, 13% abused alcohol and 12% were taking anticoagulation medication.¹⁷ Anticoagulation increases the risk for all intracranial hemorrhage by 7–10 fold (OR 1.64); 30% of hemorrhages occur in the subdural space.²⁰ This finding is consistent with the hypothesis that antiplatelet/anticoagulant use increases the risk for chronic SDH.

Limitations

Limitations of this study include inaccuracies inherent to the electronic medical record software that reduce the validity of the findings. VISTA does not keep a record of problem lists, including the date of first diagnosis, which was determined by searching for visits that first mentioned the International Classification of Diseases code for the diagnosis. Patients who received a chronic SDH diagnosis at an outside facility would thus not have been included. Patients with recurrent chronic SDH and a previous diagnosis of chronic SDH on paper, predating electronic medical records, might receive a misdiagnosis of new chronic SDH. Because VA facilities are not trauma centers, relatively few acute SDHs were noted. Patients with acute SDH may have been missed by the study if they received treatment at outside hospitals with trauma centers and did not subsequently seek follow-up care for the SDH within the VA system.

Another limitation of the study is that we are comparing the cases per year to the entire VA population each year. If patients visited the hospital for any reason during the year, they were considered to be part of the VA population that year. As a result, the population of the VISN03 system varies from year to year. Patients who were within

FIG. 2. Histogram showing the incidence rate for chronic SDH (cSDH) in the VA population as a function of time in years during the study period, 2000–2012. On the y-axis is the scale for incidence rate, shown as number of cases of chronic SDH per 100,000 persons. The *blue bars* depict the incidence rate for operative chronic SDH cases, and the *purple bars* depict the incidence rate for nonoperative cases of chronic SDH for that year. The *continuous maroon line* depicts the estimated incidence rate for chronic SDH in the VA, according to the Japan-based formula when the rate is age weighted to the age distribution of the VA population, and the *interrupted maroon line* shows the alcohol-adjusted incidence rate. The *continuous orange line* depicts the incidence rate according to the Finland-based formula when age weighted for the age distribution of the VA population, and the *interrupted orange line* shows the alcohol-adjusted incidence rate. Figure is available in color online only.

the catchment area of the hospital but did not take advantage of its services were excluded that year, even if they were technically part of the VA population. Patients who visited once were included in the VA population for that year, even if they immediately left to go to another region.

Conclusions

We calculated the incidence rate for chronic SDH in the VA population and used these data to extrapolate future VA and civilian incidence rates by using a mathematical model based on incidence rates for Finland and Japan. We predict that by 2030, the incidence of chronic SDH will reach about 121.4 cases per 100,000 persons in the VA population and 17.6 cases per 100,000 persons in the general US population. Thus by 2030, chronic SDH drainage may be the most commonly performed neurosurgical procedure. The length of stay after treatment of chronic SDH might be longer than that after tumor resection,

which could result in an increasing number of neurosurgical inpatients. Thus, it is imperative that hospitals and neurosurgeons are well equipped to treat this condition in an efficient and cost-effective manner.

References

1. Apfelbaum RI, Guthkelch AN, Shulman K: Experimental production of subdural hematomas. *J Neurosurg* 40:336–346, 1974
2. Aronson SM, Okazaki H: A Study of some factors modifying response of cerebral tissue to subdural hematomata. *J Neurosurg* 20:89–93, 1963
3. Asghar M, Adhiyaman V, Greenway MW, Bhowmick BK, Bates A: Chronic subdural haematoma in the elderly—a North Wales experience. *J R Soc Med* 95:290–292, 2002
4. Baechli H, Nordmann A, Bucher HC, Gratzl O: Demographics and prevalent risk factors of chronic subdural haematoma: results of a large single-center cohort study. *Neurosurg Rev* 27:263–266, 2004
5. Balser D, Rodgers SD, Johnson B, Shi C, Tabak E, Samadani

- U: Evolving management of symptomatic chronic subdural hematoma: experience of a single institution and review of the literature. **Neurol Res** 35:233–242, 2013
6. Chen JC, Levy ML: Causes, epidemiology, and risk factors of chronic subdural hematoma. **Neurosurg Clin N Am** 11:399–406, 2000
 7. De Cauwer H, Van Giel R, Mortelmans L, van den Hauwe L: An uncommon cause of headache after headbanging at a party. **Eur J Emerg Med** 16:212–213, 2009
 8. De Jesús O, Pacheco H, Negron B: Chronic and subacute subdural hematoma in the adult population. The Puerto Rico experience. **P R Health Sci J** 17:227–233, 1998
 9. Ferguson JW, Helmy A, Ludlam C, Webb DJ, Hayes PC, Newby DC: Hyperfibrinolysis in alcoholic cirrhosis: relative plasminogen activator inhibitor type 1 deficiency. **Thromb Res** 121:675–680, 2008
 10. Filippini G: Epidemiology of primary central nervous system tumors. **Handb Clin Neurol** 104:3–22, 2012
 11. Foelholm R, Waltimo O: Epidemiology of chronic subdural haematoma. **Acta Neurochir (Wien)** 32:247–250, 1975
 12. Forster MT, Mathé AK, Senft C, Scharer I, Seifert V, Gerlach R: The influence of preoperative anticoagulation on outcome and quality of life after surgical treatment of chronic subdural hematoma. **J Clin Neurosci** 17:975–979, 2010
 13. Frontera JA, de los Reyes K, Gordon E, Gowda A, Grilo C, Egorova N, et al: Trend in outcome and financial impact of subdural hemorrhage. **Neurocrit Care** 14:260–266, 2011
 14. Fujisawa H, Ito H, Saito K, Ikeda K, Nitta H, Yamashita J: Immunohistochemical localization of tissue-type plasminogen activator in the lining wall of chronic subdural hematoma. **Surg Neurol** 35:441–445, 1991
 15. Furlow LT: Chronic subdural hematoma. **Arch Surg** 32:688–708, 1936
 16. Gavrilovic IT, Posner JB: Brain metastases: epidemiology and pathophysiology. **J Neurooncol** 75:5–14, 2005
 17. Gelabert-González M, Iglesias-Pais M, García-Allut A, Martínez-Rumbo R: Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. **Clin Neurol Neurosurg** 107:223–229, 2005
 18. Göksu E, Akyüz M, Uçar T, Kazan S: Spontaneous resolution of a large chronic subdural hematoma: a case report and review of the literature. **Ulus Travma Acil Cerrahi Derg** 15:95–98, 2009
 19. Hamilton MG, Frizzell JB, Tranmer BI: Chronic subdural hematoma: the role for craniotomy reevaluated. **Neurosurgery** 33:67–72, 1993
 20. Hart RG, Boop BS, Anderson DC: Oral anticoagulants and intracranial hemorrhage. Facts and hypotheses. **Stroke** 26:1471–1477, 1995
 21. Horie Y: Granulocytapheresis and plasma exchange for severe alcoholic hepatitis. **J Gastroenterol Hepatol** 27 (Suppl 2):99–103, 2012
 22. Iantosca MR, Simon RH: Chronic subdural hematoma in adult and elderly patients. **Neurosurg Clin N Am** 11:447–454, 2000
 23. Ito U, Fujimoto T, Inaba Y: [Formation of the chronic subdural hematoma: 1. Clinico-pathological study of the chronic subdural hematoma (author's transl).] **No Shinkei Geka** 2:47–61, 1974 (Jpn)
 24. Juković M, Kojadinović Ž, Till V: Complete spontaneous resolution of compressive chronic subdural hematoma in a patient with liver failure. **Med Glas (Zenica)** 9:417–420, 2012
 25. Karibe H, Kameyama M, Kawase M, Hirano T, Kawaguchi T, Tominaga T: [Epidemiology of chronic subdural hematomas.] **No Shinkei Geka** 39:1149–1153, 2011 (Jpn)
 26. Kudo H, Kuwamura K, Izawa I, Sawa H, Tamaki N: Chronic subdural hematoma in elderly people: present status on Awaji Island and epidemiological prospect. **Neurol Med Chir (Tokyo)** 32:207–209, 1992
 27. Lee KS: The pathogenesis and clinical significance of traumatic subdural hygroma. **Brain Inj** 12:595–603, 1998
 28. Markwalder TM: Chronic subdural hematomas: a review. **J Neurosurg** 54:637–645, 1981
 29. Maurice-Williams RS, Kitchen N: The scope of neurosurgery for elderly people. **Age Ageing** 22:337–342, 1993
 30. Møllergård P, Wisten O: Operations and re-operations for chronic subdural haematomas during a 25-year period in a well defined population. **Acta Neurochir (Wien)** 138:708–713, 1996
 31. Miranda LB, Braxton E, Hobbs J, Quigley MR: Chronic subdural hematoma in the elderly: not a benign disease. **J Neurosurg** 114:72–76, 2011
 32. Nakamura N: [Relationship between head injuries and chronic subdural hematoma.] **No To Shinkei** 18:702–709, 1966 (Jpn)
 33. National Center for Veterans Analysis and Statistics: Unique Veteran Users Report: FY 2012. **US Department of Veterans Affairs**. (http://www.va.gov/vetdata/docs/Special-Reports/Profile_of_Unique_Veteran_Users.pdf) [Accessed January 27, 2015]
 34. Ramachandran R, Hegde T: Chronic subdural hematomas—causes of morbidity and mortality. **Surg Neurol** 67:367–373, 2007
 35. Sarnvivat P, Chiewchanvechakul W, Chumnanvej S: Chronic subdural hematoma: drainage vs. no drainage. **J Med Assoc Thai** 94:1352–1356, 2011
 36. Sato M, Nakano M, Sasanuma J, Asari J, Watanabe K: Very late onset of chronic subdural hematoma. Case report. **Neurol Med Chir (Tokyo)** 45:41–43, 2005
 37. Sousa EB, Brandão LF, Tavares CB, Borges IB, Neto NGF, Kessler IM: Epidemiological characteristics of 778 patients who underwent surgical drainage of chronic subdural hematomas in Brasília, Brazil. **BMC Surg** 13:5, 2013
 38. Suzuki J, Komatsu S: Estrogen in patients with chronic subdural hematoma. **Surg Neurol** 8:243–247, 1977
 39. Tanaka Y, Ohno K: Chronic subdural hematoma – an up-to-date concept. **J Med Dent Sci** 60:55–61, 2013
 40. Trotter W: Chronic subdural haemorrhage of traumatic origin, and its relationship to pachymeningitis haemorrhagica interna. **Br J Surg** 2:271–291, 1914
 41. Vincent GK, Velkoff VA: **The Next Four Decades. The Older Population in the United States: 2010 to 2050**. Washington, DC: US Census Bureau, 2010 (<http://www.census.gov/prod/2010pubs/p25-1138.pdf>) [Accessed February 3, 2015]
 42. Yamakami I, Mine S, Yamaura A, Fukutake T: Chronic subdural haematoma after riding a roller coaster. **J Clin Neurosci** 12:81–83, 2005
 43. Yamashita T, Friede RL: Why do bridging veins rupture into the virtual subdural space? **J Neurol Neurosurg Psychiatry** 47:121–127, 1984
 44. Yang AI, Balser DS, Mikheev A, Offen S, Huang JH, Babb J, et al: Cerebral atrophy is associated with development of chronic subdural haematoma. **Brain Inj** 26:1731–1736, 2012

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