J Neurosurg 95:256–262, 2001

Factors in the natural history of chronic subdural hematomas that influence their postoperative recurrence

HIROSHI NAKAGUCHI, M.D., PH.D., TAKEO TANISHIMA, M.D., PH.D., AND NORIO YOSHIMASU, M.D., PH.D.

Department of Neurosurgery, Teraoka Memorial Hospital, Hiroshima; and Department of Neurosurgery, Tokyo Kosei Nenkin Hospital, Tokyo, Japan

Object. Factors affecting the postoperative recurrence of chronic subdural hematomas (CSDHs) have not been sufficiently investigated. The authors have attempted to determine features of CSDHs that are associated with a high or low recurrence rate on the basis of the natural history of these lesions and their intracranial extension.

Methods. One hundred six patients (82 men and 24 women) harboring 126 CSDHs who were treated at Tokyo Kosei Nenkin Hospital between January 1989 and April 1998 were studied. Types of CSDHs were classified according to hematoma density and internal architecture, and the intracranial extension of the hematomas were investigated. The postoperative recurrence rate was calculated for each factor.

Based on the internal architecture and density of each hematoma, the CSDHs were classified into four types, including homogeneous, laminar, separated, and trabecular types. The recurrence rate associated with the separated type was high, whereas that associated with the trabecular type was low.

Chronic subdural hematomas are believed to develop initially as the homogeneous type, after which they sometimes progress to the laminar type. A mature CSDH is represented by the separated stage and the hematoma eventually passes through the trabecular stage during absorption.

Based on the intracranial extension of each hematoma, CSDHs were classified into three types, including convexity, cranial base, and interhemispheric types. The recurrence rate of cranial base CSDHs was high and that of convexity CSDHs was low.

Conclusions. Classification of CSDHs according to the internal architecture and intracranial extension may be useful for predicting the risk of postoperative recurrence.

KEY WORDS • chronic subdural hematoma • hematoma recurrence • natural history • middle meningeal artery • computerized tomography

CHRONIC subdural hematomas have been reported to have recurrence rates of 3 to 20% after burr-hole surgery;[1–3,7,21] however, the factors influencing their recurrence have not been sufficiently investigated.

In our prior report on the relationship between the position of a drainage catheter and postoperative recurrence of these lesions,[10] we found that frontal drainage is associated with the lowest recurrence rate and the least subdural air collection after closed system drainage for CSDH. In the present study, potential indicators of postoperative recurrence of CSDHs in 106 patients were investigated by examining the relationships between postoperative recurrence and both the natural progression of CSDHs and the extent of their feeding vessels,[19] which are considered to provide a significant influence on the risk of recurrence of these lesions.

Clinical Material and Methods

Patient Population

Among the 144 patients with CSDHs who were treated at Tokyo Kosei Nenkin Hospital between January 1989 and April 1998, 106 consecutive patients (82 men and 24 women) were studied. Bilateral hematomas were present in 20 patients and thus there were a total of 126 lesions.

We defined CSDH as an SDH surrounded by a thin capsule (hematoma membrane) and consisting of dark reddish liquefied blood at operation. If the date of head trauma was clear, a CSDH was defined as a hematoma that had persisted more than 3 weeks after the patient suffered head trauma.

Nine patients with one or more risk factors that already had been documented to influence recurrence[1] were excluded from the study: four with thrombocytopenia; four receiving anticoagulant or thrombolytic drugs; one receiving hemodialysis; and one with a ventriculoperitoneal shunt. Another 29 patients were also excluded: nine who did not undergo surgery and 20 who could not be observed for 3 months postoperatively.

In all cases, we examined the patient's age and sex, the time from head injury to initial CT scanning, the type of hematoma categorized according to our new classification of internal architecture, and the intracranial extension of the lesion on preoperative and postoperative CT scans.

At our hospital, CT scanning is generally performed five times in every patient with CSDH: preoperatively, on Days 1 to 3, on Day 7, and on approximately Day 30 and Day 90 postoperatively.

Abbreviations used in this paper: CSDH = chronic subdural hematoma; CT = computerized tomography; MMA = middle meningeal artery; PR = postoperative recurrence; RO = repeated operation; SDH = subdural hematoma.

TABLE 1
Relationship between sex of patient and PR rate of CSDHs

Patient's Sex	No. of Cases (%)	PR Rate (%)	p Value*
male	82 (77)	20	0.5541
female	24 (23)	13	
total	106 (100)	17	

* The Fisher exact test was used for analysis of differences.

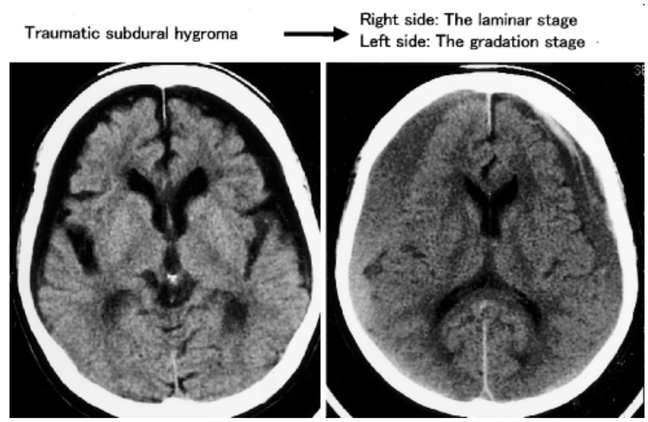

FIG. 1. Computerized tomography scans obtained in representative cases of CSDH for which temporal profiles were available.

All patients, except one who displayed neurological deficits caused by CSDH, underwent surgical intervention including creation of one burr hole, irrigation of the hematoma with sterile saline, and postoperative closed system drainage with the aid of a silicone tube (Type B ventricular drainage catheter; Hanako Medical Co., Urawa, Japan).

Craniotomy was performed in one patient with a recurrent hematoma of the interhemispheric type.

Regardless of the presence of residual hematoma and subdural air on CT scans 1 day postsurgery, all drainage catheters were removed within 48 hours after surgery and the daily activities of the patients were not restricted afterward. Until the catheter was removed, patients were required always to keep the head as high as the heart.

Recurrence of CSDH was defined as an increase in the volume of the SDH on the operated side and compression of the brain surface observed on CT scans obtained within 3 months postoperatively, when compared with findings 1 day after surgery. Repeated operations were performed when neurological symptoms reappeared or the cerebral sulci were diffusely effaced by recurrent hematoma. Patients without neurological deficits or with a small amount of residual hematoma were observed. The PR rate and the RO rate were calculated and compared among the various factors that we investigated.

Natural History of CSDHs

There were 18 patients with CSDHs who initially displayed no marked neurological deficits and received no surgical intervention for several weeks or more, while being monitored by CT scanning, which was performed at least twice. We examined changes in the density and internal architecture of the CSDHs on these CT scans to investigate the natural history of CSDHs.

Interval From Head Injury to Initial CT Scan

Although there were many patients in whom a history of head injury was unknown or unclear, the date of head trauma was able to be determined in 63 patients (59%), and the interval from head injury to the initial CT scan was calculated for these patients. We analyzed the correlation between this interval and both the recurrence rate and the hematoma type.

Internal Architecture of the Hematoma

All hematomas were classified into four types according to their internal architecture, which corresponded to possible stages in the natural history of CSDH: homogeneous, laminar, separated, and trabecular types.

The homogeneous type was defined as a hematoma that exhibited a homogeneous density (low–high). The laminar type was defined as a subtype of the homogeneous type that had a thin high-density layer along the inner membrane. The separated type was defined as a hematoma containing two components of different densities with a clear boundary lying between them; that is, a lower density component located above a higher density component. If the boundary was indistinct, with the low density and high density being mingled at the border, this was called the "gradation" type and was considered to be a subtype of the separated type. The trabecular type was defined as a hematoma with inhomogeneous contents and a high-density septum running between the inner and outer membrane on a low-density to isodense background.

Three series of CT scans (that is, scans obtained preoperatively, scans obtained 1 day after surgery, and scans obtained 7 days after surgery) were assessed according to this classification.

Intracranial Extension of CSDHs

The CSDHs were classified into three types according to their intracranial extension: hematomas localized at the convexity without involvement of the cranial base were named the convexity type; hematomas that extended to the cranial base were named cranial base CSDHs; and hematomas that extended to the interhemispheric fissure were named interhemispheric CSDHs.

The cranial base type was divided into two subtypes: hematomas limited to frontal base were called frontal base CSDHs; whereas those extending from the temporal base to the frontal base were named frontotemporal base CSDHs.

The convexity type was also divided into two subtypes: hematomas limited to the frontal region were called frontal convexity CSDHs and those spanning the frontal and parietal regions were called frontoparietal convexity CSDHs.

There were no patients in whom the CSDH was localized to the parietal convexity, occipital convexity, temporal base, or posterior fossa.

Statistical Analysis

For statistical analysis, the Fisher exact test or the chi-

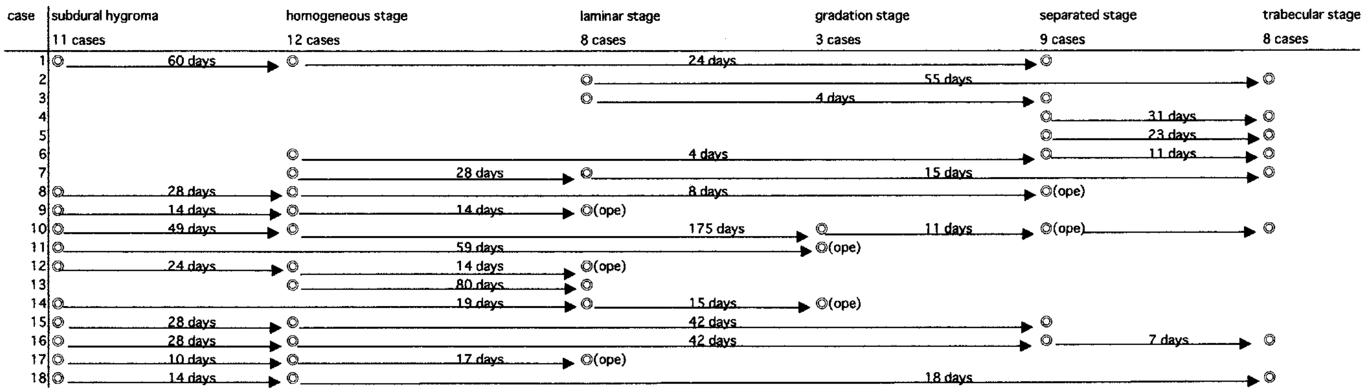

FIG. 2. Summary of temporal profiles of 18 CSDHs in which CT scans could be used to document temporal changes in the densities and internal architectures of the lesions. ope = operation.

square test for independence was performed using commercially available statistical computer software (Stat View R J Version 4.11 software; SAS Institute, Cary, NC).

Results

For the 126 CSDHs in 106 patients, 142 surgical procedures were performed. There were 82 men (77%) and 24 women (23%) in the study, ranging in age from 20 to 96 years with an average age of 67 years.

The PR rate was 17% (21 lesions) and the RO rate was 13% (16 lesions). The PR and RO rates were 20% (16 lesions) and 16% (13 lesions), respectively, in male patients, whereas the rates were 13% (three lesions) and 4% (one lesion), respectively, in female patients (Table 1). Although men had a higher RO rate, no significant difference was seen between the sexes.

The PR rate of patients aged less than 60 years was 16% and that of patients aged 70 years or older was 15%. There was no significant difference between younger and older patient groups.

Natural History of CSDHs

Changes in 18 CSDHs could be examined over time by referring to 51 CT scans obtained from 4 to 235 days posttrauma, with the average being 49 days (Figs. 1 and 2).

Eleven of the CSDHs were initially subdural hygromas that displayed a homogeneous water density in the subdural space and no compression of the brain. High-resolution CT scans revealed high-density membranous structures floating in the subdural space, which were thought to represent the torn arachnoid membrane.

After an average of 28 days (range 10–60 days), the subdural hygroma changed into an isodense to high-density homogeneous hematoma. This stage of CSDH was seen in 12 patients and was named the homogeneous stage. During the homogeneous stage, the inner membrane was sometimes observed as a high-density line on CT scans. In eight CSDHs, this high-density line developed to form an indistinct high-density structure along the inner membrane on a homogeneous isodense background. We named this the laminar type of CSDH.

Following the homogeneous stage, a line of demarcation separating an upper thin component from a lower thick component was seen inside the hematoma cavity in eight patients, developing from the homogeneous stage in six and from the laminar stage in two. The interval from the homogeneous stage to the separated stage was 51 days on average (range 4–186 days). During this stage, the hematoma was divided into a low-density region lying above a high-density region and a demarcation line was observed in the hematoma cavity at the boundary.

A subtype of this stage, the gradation stage, was observed on CT scans obtained in three patients. In these patients, the lower density area displayed a gradual transition in the higher density area. At the next stage, the hematoma developed trabeculae with a moderately high density which were thought to consist of fibrous tissue[6] lying in a low-density to isodense matrix. We named this the trabecular stage. The average interval between the homogeneous stage and the trabecular stage was 31 days (range 15–49 days). This type of CSDH was observed on CT scans in eight patients. In four patients, it developed from the separated stage after an average of 18 days, and in two patients, it developed from the laminar stage after an average of 35 days.

To summarize, a CSDH is considered to originate as the homogeneous type and sometimes develops into the laminar type. It then matures as the separated type and is finally absorbed as the trabecular type.

Interval From Head Trauma to Initial CT Scanning

The date of head trauma was able to be determined in 63 patients (59%) and the number of days from head injury to the time of initial CT scanning was calculated for this group (Table 2). Thirty-one CSDHs were detected within 60 days of head trauma and the PR rate was 32% (10 of 31 patients). In 32 patients, 60 days or more had passed since head injury, and the PR rate was 3% (1 of 32 patients). There was a significant difference between the two groups ($p = 0.0027$; Fisher exact test).

Internal Architecture of the Hematoma

Preoperative CT scans were available for 108 CSDHs, but not for the other 18 lesions because films had been lost. The homogeneous type of CSDH was seen in 31% of cases (33 lesions), the laminar type in 19% (21 lesions), the separated type in 20% (22 lesions), and the trabecular

TABLE 2

Relationship between interval from head trauma to initial CT scan and PR rate of CSDHs

Days From Trauma to Initial CT Scan	No. of Cases (%)	PR Rate (%)	p Value*
<60	31 (49)	32	0.0027
≥60	32 (51)	3	
total	63 (100)	17	

* The Fisher exact test was used for analysis of differences.

TABLE 3

Relationship between type of CSDH based on internal structure and PR rates

Classification of Internal Structure	No. of Lesions (%)	PR Rate (%)	p Value*	p Value†
homogeneous	33 (31)	15	>0.9999	
laminar	21 (19)	19	0.7388	
separated	22 (20)	36	0.0064	0.0042
trabecular	32 (30)	0	0.0026	
total	108 (100)	16		

* Compared with other types; the Fisher exact test was used for analysis of differences.
† Chi-square test for independence.

type in 30% of cases (32 lesions) (Table 3 and Fig. 3). The PR rate was 15% (five of 33 lesions) in the homogeneous type, 19% (four of 21 lesions) in the laminar type, 36% (eight of 22 lesions) in the separated type, and 0% (none of 32 lesions) in the trabecular type. There was a significant difference between separated and trabecular CSDHs (p = 0.0003, Fisher exact test), between laminar and trabecular CSDHs (p = 0.0204), between trabecular and other types (p = 0.0026), and between separated and other types of CSDHs (p = 0.0064).

Intracranial Extension of the Hematoma

By examining preoperative or postoperative Day 1 CT scans, 116 CSDHs were classified according to their extension (Fig. 4); data were not available for the other 10 lesions. The frontal convexity type accounted for 5% (six lesions), the frontoparietal convexity type for 22% (25 lesions), the frontal base type for 57% (66 lesions), the frontotemporal base type for 14% (16 lesions), and the interhemispheric type for 3% (three lesions). The PR rate was 0% (0 of six lesions) in the frontal convexity type, 4% (one of 25 lesions) in the frontoparietal convexity type, 20% (13 of 66 lesions) in the frontal base type, 31% (five of 16 lesions) in the frontotemporal base type, and 67% (two of three lesions) in the interhemisphere type. There were significant differences among the aforespecified five types (p = 0.0221; chi-square test for independence) and between the convexity hematomas (frontal convexity and frontoparietal convexity CSDHs, one of 31 lesions or a 3% PR rate) and the cranial base hematomas (frontal base and frontotemporal base CSDHs, 18 of 82 lesions or a 22% PR rate) (p = 0.0218, Fisher exact test).

Discussion

Determination According to the Natural History of CSDHs

The internal architecture at each stage during the natural history of CSDHs is the following.

Water Density Stage or Subdural Hygroma. Before a CSDH emerges, there is a water density stage or a subdural hygroma. According to previous reports,[4,8,9,12,13,18,22] traumatic subdural hygroma is considered to be caused by cerebrospinal fluid leaking from tears in the arachnoid membrane. If a cerebrospinal fluid collection remains in the subdural space for more than a few weeks, it is thought to induce migration or proliferation of inflammatory cells derived from the dural border cells and this produces a layer of fibroblasts along the dura, which develops into the outer membrane of the hematoma.[4,6,13,15,17]

To predict which subdural hygromas will progress to become CSDHs is difficult and the rate of transformation is still unknown.

Stage 1 of CSDH: the Homogeneous Stage. During the homogeneous stage, the outer and inner membranes develop around the subdural space.[6,14,17] As the hematoma matures, blood vessels derived from the MMA are believed to extend into the hematoma membrane, causing vascular congestion.[6,19] Arterial pressure stresses the walls of the sinusoidal microcapillaries, resulting in minor, but persistent, subdural bleeding episodes. However, as Nomura, et al.,[11] have reported, rebleeding is moderate and the balance between coagulative and fibrinolytic activities is maintained in isodense CSDHs, as well as in high-density CSDHs, which are equivalent to our homogeneous type.

Subtype of Stage 1: the Laminar Stage. The laminar stage is considered to be a subtype of the homogeneous stage. We are probably the first to mention the laminar type of CSDH. The high-density laminar structure running along the inner membrane is considered to consist of fresh blood from the hematoma membrane. At this stage, the recurrence rate has increased to 19%. The higher recurrence rate of this type may be related to a greater vascularity than that of the purely homogeneous type.

Stage 2 of CSDH: the Separated Stage (Including the Gradation Stage). As the hematoma matures, fibrinolysis occurs. During the separated stage, the hematoma separates into two components, such that a thin component becomes mixed with a thick component of the liquefied hematoma and normal head motion cannot homogenize these components. The hematoma increases in volume and the surrounding brain tissue becomes compressed and congested. Nomura, et al.,[11] demonstrated by an analysis of the concentrations of fibrinogen, fibrin monomer, and d-dimer that the layering type of CSDH, which is equivalent to our separated type, is active, with a high tendency to rebleed and for hyperfibrinolytic activity.

The separated type of CSDHs had the highest recurrence rate of all CSDH types, as some previous reports have demonstrated.[5,11] Therefore, meticulous perioperative management and frequent postoperative follow-up examinations are necessary for managing this type of CSDH.

During the gradation stage, mild head movement causes homogenization of the hematoma. Therefore, the hematoma is not completely separated and the gradation stage

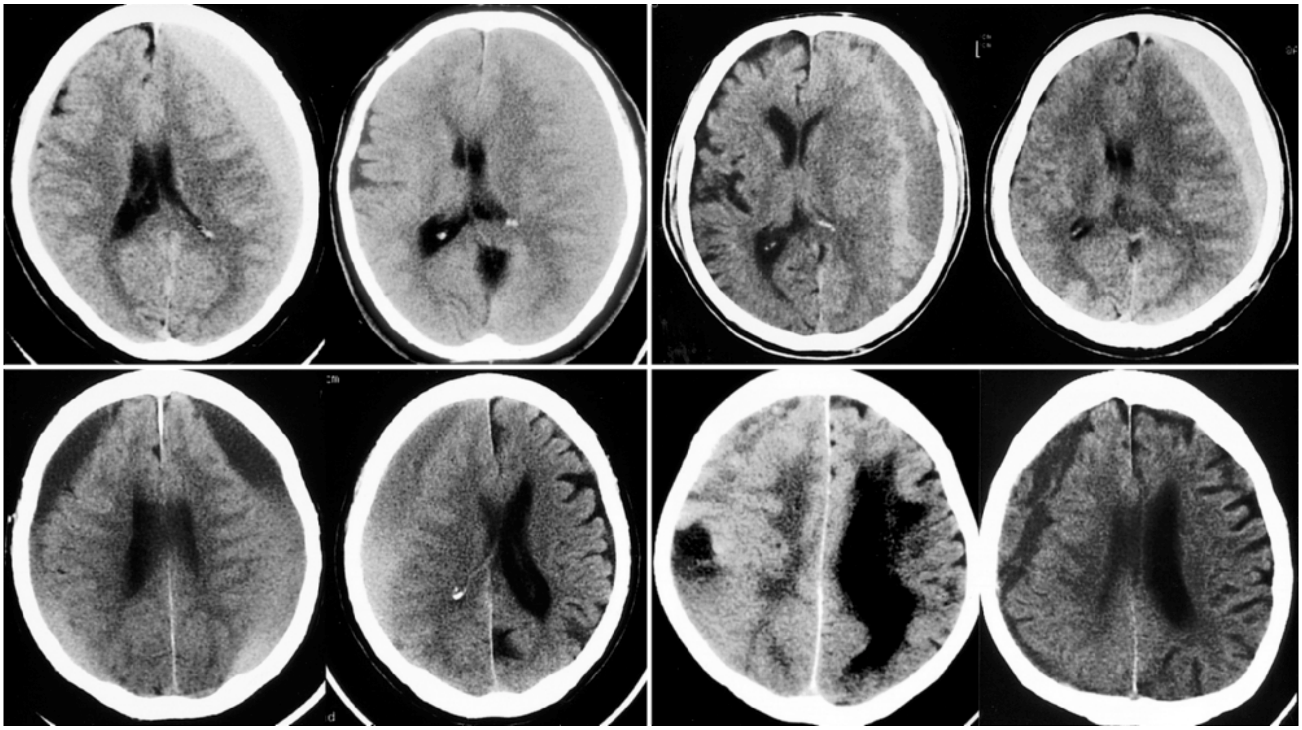

FIG. 3. Computerized tomography scans demonstrating classification of CSDHs according to their internal architectures. *Upper Left:* Homogeneous type (PR rate 15%, RO rate 12%). *Upper Right:* Laminar type (PR rate 19%, RO rate 14%). *Lower Left:* Separated type (PR rate 36%, RO rate 23%). *Lower Right:* Trabecular type (PR rate 0%, RO rate 0%).

is thought to come between the homogeneous and separated stages. In our series, the gradation type of hematoma was seen in five patients, one of whom displayed recurrence postoperatively.

Stage 3 of CSDH: the Trabecular Stage. In the trabecular stage, which features high-density septa created by fibrosis,[6] the interstitial hematoma matrix changes from an isodense to a low-density signal on CT scans, and on surgical inspection, it changes from a dark reddish to xanthochromic translucent liquefied hematoma, which diminishes in volume over time. This is considered to be the resolution stage of CSDH. The PR rate of this type was 0% in our series, and thus frequent irrigation during surgery may not be needed. During this stage, the risk of bleeding from the hematoma capsule seems to abate and symptoms may not be related to bleeding from macrocapillaries in the hematoma membranes, but rather to cerebral congestion caused by the bulky CSDHs.

Although a thickened or calcified hematoma capsule sometimes remains as a crescent-shaped subdural mass for years, this is thought to be residue of the CSDH and none ever displayed enlargement afterward in our series.

Takahashi and associates[16] classified CSDHs by using CT values, but did not assess their internal architecture. Therefore, those authors' classification could not differentiate between homogeneous and heterogeneous hematomas, which we consider to be a significant point.

Nomura, et al.,[11] classified CSDHs into five types according to CT findings: low-density, isodense, high-density, mixed-density, and layered types. Tsutsumi, et al.,[20] reported that there were no statistical differences in postoperative recurrence among stages. They found that the high-density and isodense types were similar in rebleeding, as well as in coagulation and fibrinolysis activity. In our investigation, low-density and isodense hematomas developed continuously into high-density hematomas, and it was difficult to classify lesions into two types. Considering their homogeneous appearance on CT scans and their similar recurrence rates, there seems to be no significant problem in grouping these lesions together in a single stage.

The mixed-density type described by Nomura, et al.,[11] includes both the trabecular and laminar types described in our classification. Despite their similar mixed-density appearance on CT scans, the laminar stage should be distinguished from the trabecular stage, because the laminar type of hematoma has a far higher recurrence rate and is considered to be an earlier stage in the natural history of these lesions than the trabecular stage.

Hematoma Extension

After an analysis of superselective MMA arteriograms obtained in six patients, Tanaka, et al.,[19] reported that CSDH membranes are mainly supplied from the MMA. If a CSDH is mostly fed by the MMA, hematomas with cranial base extensions are expected to have a higher rebleeding rate or a higher postoperative recurrence rate than those without cranial base extension, because the feeding vessels are distributed more widely.

We therefore classified CSDHs according to the extents

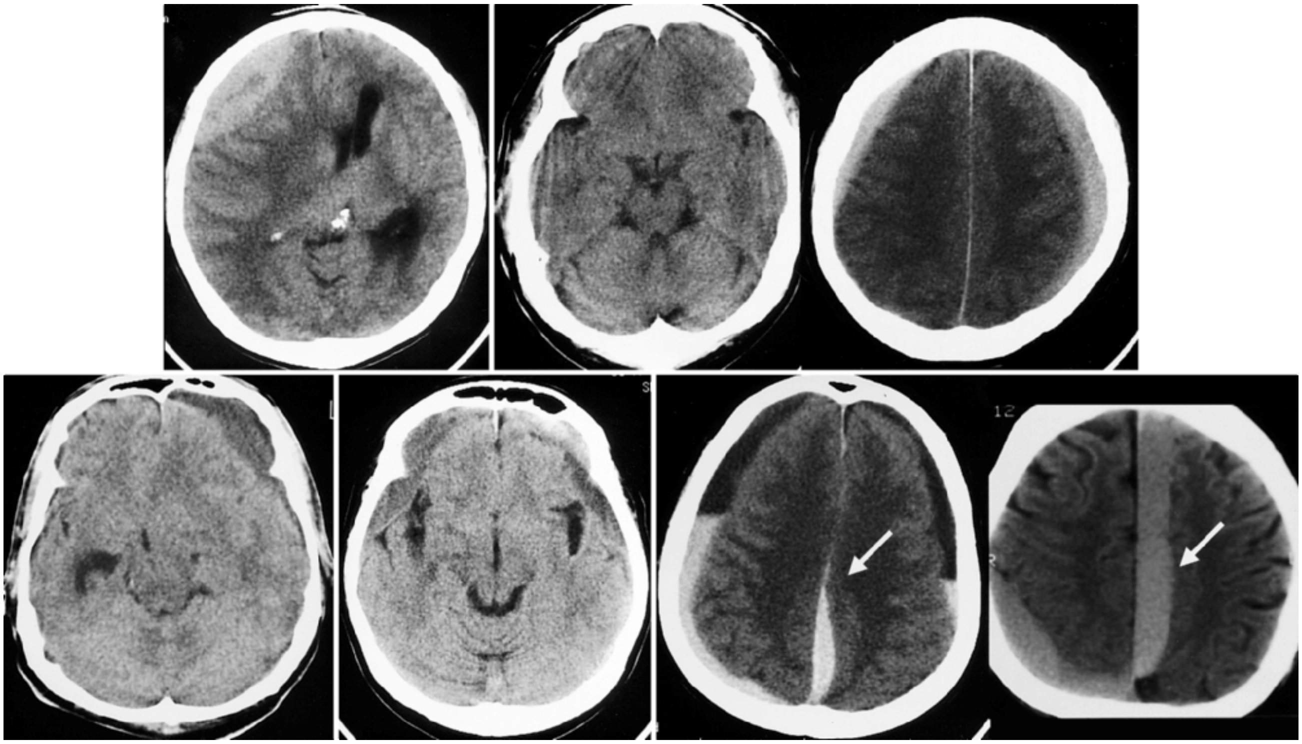

FIG. 4. Computerized tomography scans demonstrating classification of CSDHs according to their intracranial extensions. *Upper Left:* Convexity Type 1 or frontal convexity type (PR rate 0%, RO rate 0%). *Upper Right:* Convexity Type 2 or frontoparietal convexity type (PR rate 4%, RO rate 0%). *Lower Left:* Cranial base Type 1 or frontal base type (PR rate 20%, RO rate 18%). *Lower Center:* Cranial base Type 2 or frontotemporal base type (PR rate 31%, RO rate 25%). *Lower Right:* Interhemispheric type *(arrows)* (PR rate 67%, RO rate 33%).

to which these lesions involved the MMA: the frontoparietal convexity type was related to the peripheral parietal and frontal branches of the MMA; the frontal convexity type was related only to the peripheral frontal branch of the MMA; the frontal base type was related to the frontal trunk of the MMA; and the frontotemporal base type was related to the two main trunks of the MMA. The interhemispheric type of CSDH was also differentiated from the others because the MMA does not play an important role in feeding this type of hematoma. Using this classification, the cranial base type was found to have the highest recurrence rate, as we had predicted.

In addition to the extent of feeding vessels from the MMA, a more enlarged subdural space and greater difficulty in hematoma removal encountered with the cranial base CSDHs, compared with convexity CSDHs, are considered to be factors influencing the higher recurrence rate.

The extremely low recurrence rate of the convexity type of CSDH indicates that the bridging veins at the convexity do not have an important role in the recurrence of CSDHs, although arachnoid tears in the vicinity of the convexity bridging veins are considered to induce the earlier stage of CSDH.

Timing of Surgery and CT Scanning After Head Trauma

When surgical intervention was performed less than 60 days after head trauma occurred, there was an extremely high postoperative recurrence rate and this was especially noted for the homogeneous type of CSDH. When the interval from head trauma to initial CT scanning was less than 60 days, the recurrence rate of the homogeneous type was far higher than when the interval was 60 days or more (48% compared with 5%). We consider this, however, only to reflect the fact that fibrosis of the capsule or trabeculae tends to be immature and organization of the hematoma is limited in younger CSDHs, because there was no predominant difference in internal architecture of CSDHs between lesions less than 60 days old and lesions 60 days or more after head trauma.

To sum up, it is important to classify CSDH into lesions

TABLE 4

Summary of results of classifications of internal structure and intracranial extension of CSDHs with respect to PR rate

Type of CSDH	PR Rate (%)
conditions for a lower PR rate*	
trabecular type	0
convexity type	3
conditions for a higher PR rate†	
separated type	36
separated type on cranial base	38
cranial base type	22

* All factors were proved statistically to have a low recurrence rate ($p < 0.05$).

† All factors were proved statistically to have a high recurrence rate ($p < 0.05$).

with a high recurrence rate and lesions with a low recurrence rate according to our system (Table 4), and to select appropriate surgical procedures and postoperative management to treat this condition efficiently. If a CSDH is classified as the convexity or trabecular type, it is expected to exhibit quite a low recurrence rate (one [2%] of 50 CSDHs) and frequent irrigation during surgery may not be needed. By contrast, if a CSDH is determined to be neither the convexity nor trabecular type, most of which are properly classified as the cranial base type, and at the same time as the homogeneous or separated type, it is expected to have quite a high recurrence rate (14 [29%] of 49 CSDHs). If they are found to be in such a higher recurrence group, patients should be informed of the high postoperative recurrence rate on admission and meticulous perioperative management should be undertaken to reduce postoperative recurrence, including placing the tip of the drainage catheter in the frontal convexity and removing subdural air sufficiently, as we proved in our prior report;[10] frequent postoperative follow-up examinations are also necessary.

Conclusions

Three major points should be reiterated. 1) We believe that CSDHs originate in the homogeneous stage and sometimes develop in the laminar stage. The hematoma becomes mature during the separated stage, and is finally absorbed during the trabecular stage. 2) According to the internal architecture and density of hematomas, CSDHs were classified into four types: homogeneous, laminar, separated, and trabecular types. The recurrence rate of the separated type was high and that of the trabecular type was low. 3) According to their intracranial extension, CSDHs were classified into three types: convexity, cranial base, and interhemispheric types. The recurrence rate of the cranial base type of CSDHs was high and that of the convexity type was low.

References

1. Arbit E, Patterson RH Jr, Fraser RAR: An implantable subdural drain for treatment of chronic subdural hematoma. **Surg Neurol 15:**175–177, 1980
2. Asano Y, Hasuo M, Takahashi I, et al: [Recurrent cases of chronic subdural hematoma—its clinical review and serial CT findings.] **No to Shinkei 44:**827–831, 1992 (Jpn)
3. Ernestus RI, Beldzinski P, Lanfermann H, et al: Chronic subdural hematoma: surgical treatment and outcome in 104 patients. **Surg Neurol 48:**220–225, 1997
4. Friede RL, Schachenmayr W: The origin of subdural neomembranes. II. Fine structure of neomembranes. **Am J Pathol 92:** 69–84, 1978
5. Fujioka S, Matsukado Y, Kaku M, et al: [CT analysis of 100 cases with chronic subdural hematoma with respect to clinical manifestation and the enlarging process of the hematoma.] **Neurol Med Chir 21:**1153–1160, 1981 (Jpn)
6. Kawano N, Endo M, Saito M, et al: [Origin of the capsule of chronic subdural hematoma-an electron microscopic study.] **No Shinkei Geka 16:**747–752, 1988 (Jpn)
7. Kotwica Z, Brzezinski J: Chronic subdural haematoma treated by burr holes and closed system drainage: personal experience in 131 patients. **Br J Neurosurg 5:**461–465, 1991
8. Markwalder TM: Chronic subdural hematomas: a review. **J Neurosurg 54:**637–645, 1981
9. Nagata K, Asano T, Basugi N, et al: [Studies on the factors affecting the reduction of chronic subdural hematoma: effect of preoperative factors with special reference to cerebral atrophy.] **No Shinkei Geka 16:**1347–1353, 1988 (Jpn)
10. Nakaguchi H, Tanishima T, Yoshimasu N: Relationship between drainage catheter location and postoperative recurrence of chronic subdural hematoma after burr hole irrigation and closed system drainage. **J Neurosurg 93:**791–795, 2000
11. Nomura S, Kashiwagi S, Fujisawa H, et al: Characterization of local hyperfibrinolysis in chronic subdural hematomas by SDS-PAGE and immunoblot. **J Neurosurg 81:**910–913, 1994
12. Sato S, Suzuki J: Ultrastructural observations of the capsule of chronic subdural hematoma in various clinical stages. **J Neurosurg 43:**569–578, 1975
13. Schachenmayr W, Friede RL: The origin of subdural neomembranes. I. Fine structure of the dura-arachnoid interface in man. **Am J Pathol 92:**53–68, 1978
14. Shimoji T, Sato K, Ishii S: [A pathogenesis of chronic subdural hematoma; its relationship to subdural membrane.] **No Shinkei Geka 20:**131–137, 1992 (Jpn)
15. Takahashi Y: [Pathogenetic consideration on chronic subdural hematoma after intracranial surgery for nontraumatic lesions.] **Neurol Med Chir 27:**1080–1086, 1987 (Jpn)
16. Takahashi Y, Mikami J, Ueda M, et al: [Analysis of chronic subdural hematoma based on CT (part 3)-clinical stage classification based on CT findings.] **Neurol Med Chir 24:**607–614, 1984 (Jpn)
17. Takahashi Y, Mikami J, Ueda M, et al: [The origin of chronic subdural hematoma considered on the basis of hematoma membrane findings and contained fluid findings.] **Neurol Med Chir 25:**998–1009, 1985 (Jpn)
18. Tanaka T, Fujimoto S, Saito K, et al: [Histological study of operated cases of chronic subdural hematoma in adults: relationship between dura mater and outer membrane.] **No Shinkei Geka 25:**701–705, 1997 (Jpn)
19. Tanaka T, Fujimoto S, Saitoh K, et al: [Superselective angiographic findings of ipsilateral middle meningeal artery of chronic subdural hematoma in adults.] **No Shinkei Geka 26:** 339–347, 1998 (Jpn)
20. Tsutsumi K, Maeda K, Iijima A, et al: The relationship of preoperative magnetic resonance imaging findings and closed system drainage in the recurrence of chronic subdural hematoma. **J Neurosurg 87:**870–875, 1997
21. Wakai S, Hashimoto K, Watanabe N, et al: Efficacy of closed-system drainage in treating chronic subdural hematoma: a prospective comparative study. **Neurosurgery 26:**771–773, 1990
22. Yamashima T, Friede RL: [Light and electron microscopic studies on the subdural space, the subarachnoid space and the arachnoid membrane.] **Neurol Med Chir 24:**737–746, 1984 (Jpn)

Manuscript received July 31, 2000.
Accepted in final form April 24, 2001.
Address reprint requests to: Hiroshi Nakaguchi, Department of Neurosurgery, Teraoka Memorial Hospital, 37, Ooaza Shinichi, Shinichi town, Ashina gun, Hiroshima prefecture, 729–3103, Japan. email: hnakaguchi@hi-ho.ne.jp.